A STUDY ON ELECTROCARDIOGRAPHIC CHANGES IN HEAD INJURY AND THEIR CORRELATION WITH THE OUTCOME IN PATIENTS WITH TRAUMATIC BRAIN INJURY

By

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EMERGENCY MEDICINE

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INTRODUCTION

India is now facing triple burden of communicable diseases, non-communicable diseases and injuries. Of the worldwide average of 7,00,000 road accidents, 10% occur in India. India has reported the highest number of Head injuries in the world. With more than 100000 lives lost every year and 1 million suffering from severe head injuries.

Among various injuries, traumatic brain injury causes major worldwide death and disability. Traumatic brain injury accounts for 5.5 million cases each year worldwide. Patients who suffered traumatic brain injury frequently show changes in electrocardiograph. But their significance remains uncertain (2)

ECG changes are observed many neurological and neurosurgical cases, many of these changes are attributed to increased sympathetic tone and elevated catecholamine release, these ecg changes suggest ischemia and primary myocardial dysfunction. (16)

There have been documentation of dysfunction of cardia in traumatic brain injury and implied as an indicator of poor outcome in paradigms of brain injury where there seems to be a interaction between the heart -brain- lung leading to dysfunction of the myocardium.(1)

Bramwell was the first to describe the occurrence of electrocardiographic changes in brain injury patients [7] in the 1934. Ever since a major numbers of studies have shifted their focus on studying the possible connection between acute brain injury and the cardiovascular system. These investigations have showed upto 12% to 99% incidence of changes in ECG in patients with traumatic brain injury.

Singla et al. [8] in his analysis of cranio-cerebral injuries and changes in electrocardiograph observed, QTc prolongation , depression of T wave, diffuse changes of T waves to be associated with increased mortality.

Coghlan and coworkers' [9] in their study of patients with subarachnoid haemorrhage of aneurysmal origin , noticed ECG changes were specific , which comprised of heart rate variability, QTc interval, abnormalities of ST segment and T wave. Further these changes were linked with adverse outcome neurologically.

In traumatic brain injuries [1], the changes in electrocardiograph are quite frequent, comprising mainly of morphological changes, disturbances in rhythm . upto 49% to 100% of patients with subarachnoid haemorrhage had electrocardiographic changes[17.]

In a study, electrocardiograms from 164 Bantu patients who had suffered head injuries were recorded and compared to two control groups. Patients with head injuries had longer Q-Tc intervals, higher P wave voltages, more frequently elevated QRS voltages, inverted T waves in precordial leads V4 to V6, U waves taller than 1 mm, sinus arrhythmia with a fixed pacemaker, and sinus arrhythmia with a pacemaker wandering in the sinoatrial node. As the level of consciousness declined, there were more electrocardiographic anomalies (5)

Abnormalities of the repolarisation including the QT interval and ST segment changes , changes in morphology of T wave are the commonest abnormalities in ECG among patients with intracerebral haemorrhage and subarachnoid haemorrhage.

ECG changes suggestive of cardiac pathology can be associated with intracranial pathology, most notably subarachnoid haemorrhage. Large amounts of norepinephrine are released into the systemic circulation, resulting in hypertension, tachycardia, dysrhythmias and ECG changes[11].

Lacy et al[1] in his report observed that any interruption of connections of cardiovascular system between the brainstem and forebrain parts of the central nervous system, could lead to appearance of arrythmias of different type and thus the role played by these regions in the causation of the same[14].

Patients with subarachnoid haemorrhage (SAH), frequently demonstrate dysfunction of the cardia transiently, inversion of T wave, prolongation of QT interval along with parallel cardiac troponin release. Patients with stress cardiomyopathy that is tako tsubo, after sstressful events of ,on echocardiography have been observed to have transient apical ballooning syndrome, without any documentation of stenosis of coronary artery significantly[10].

Frontera et al, reported that Clinically important arrhythmias, most often atrial fibrillation or flutter, occurred in 4% of SAH patients.Arrhythmias increase the incidence of cardiovascular comorbidity, lengthen hospital stays, and have a negative impact on recovery or even result in mortality after SAH. Patients were followed up after three months and 16 of the twenty five patients with arrythmia Sixteen of 25 (64%) patients who experienced an arrhythmia were dead with one person being disabled [12]

Rudehill et al, reported variable presentation of ECG changes in patients with subarachnoid haemorrhage, u wave were prominent in more than forty seven percent, abnormal T wave in thirty two percent, prolongation of QTc interval in twety three percent [13].

Though studies have reported ECG changes brain injury cases, the mechanism still remains to be understood. Raised intracranial haemorrhage causing over activity of sympathetic system appears to be the main pathophysiology [10]

Studies that were conducted earlier have described the presence of associations between outcomes after brain injuries and electrocardiographic changes, but many of these are retrospective studies and had many limitations. ECG is an inexpensive bedside tool that can be used as a prognostic factor in traumatic brain injury.

This study aims to determine the relation of ECG changes and severity of the head injury and to assess the usefulness of ECG as a prognostic indicator in the outcome of patients with traumatic brain injury.

REVIEW OF LITERATURE:

Vijay Krishnamoorthy et al. [1] conducted a retrospective study in 2010 results showed among the fifty nine patients with TBI, 22% had tachycardia, prolongation of QTc was present in 42.4%. upto 10% showed repolarisation abnormalities. The research And last, it was determined that a 12-lead ECG might be used instead of invasive or expensive tests to evaluate individuals who have suffered a traumatic brain injury for cardiac abnormalities. [1]

FAN- Xin, Feng - he DU, Jun-ping Tan [2] conducted a retrospective study of ECG in acute brain injury at Beijing Tiantan Hospital. Of 335 ABI patients, two hundred and six had abnormal ECGs. The commonest was ST-t wave abnormality , succeeding that was followed by sinus tachycardia . Significant association was established between ECG changes and the severity of head injury and outcome. ST-T changes and QT prolongation significantly associated with short outcomes, showed by logistic regression analysis. . [2]

Huber S et al. [3] conducted a study on broken heart syndrome cardiovascular manifestation in traumatic brain injury. Heart mind, 2018. They concluded that clinicians need to keep in mind the probability of cardiovascular changes in patients with traumatic injury and there is need to conduct further transitional studies that are required to supplement these observations with changes in cardiac enzyme or associated abnormality. [3]

Mariam Astarbadi et al. [4] conducted a study on the impact of non-neurological organ dysfunction on outcomes in severe isolated traumatic brain injury, division of trauma, critical care University of Arizona, Tucson. J trauma acute care Surgery 2020, results showed one in every three isolated severe traumatic brain injury patients develop non-neurological organ dysfunction among which cardiovascular system is involved 12%. Non-neurological organ dysfunction is independently associated with worse outcomes [4]

Bryan R Collier et al. [5] in their study on traumatic SAH conducted at Pennsylvania trauma centre, found that QTc prolongation is associated with severe subarachnoid haemorrhage. Measurements and Main Results: QTcprolongation occurred in 67% of those with traumatic SAH As the severity of the tSAHincreased, the average QTc became more prolonged (Pearson's r = 0.855, P = 0.003). Conclusions: traumatic subarachnoid haemorrhage is a commonest cause of an acquired prolonged QTc . As the traumatic subarachnoid haemorrhage becomes more severe, the QTcbecomes more prolonged.[5]

Veda murthy samrudral et al. [6] did a retrospective study on ECG changes in traumatic brain injury in 2016, at the department of neurosurgery and cardiology in NarayanaHospital, Nellore. There were 109 patients in total, and there were also 109 admission ECGs that could be interpreted. The majority (65.1%) of head injuries were mild, followed by severe (18.3%) and moderate (15.6%) head injuries. A statistical analysis revealed a substantial association between the outcome, the ECG data, and the severity of the head injury. Therefore, it is essential to acknowledge the value of the ECG as a straightforward technique to detect circulatory alterations in patients with traumatic brain damage. However, additional prospective studies are required to add changes in cardiac enzyme levels, concomitant echocardiography abnormalities, and their relationships to ECG results and overall outcome to these findings. [6]

TRAUMATIC BRAIN INJURY – OVERVIEW

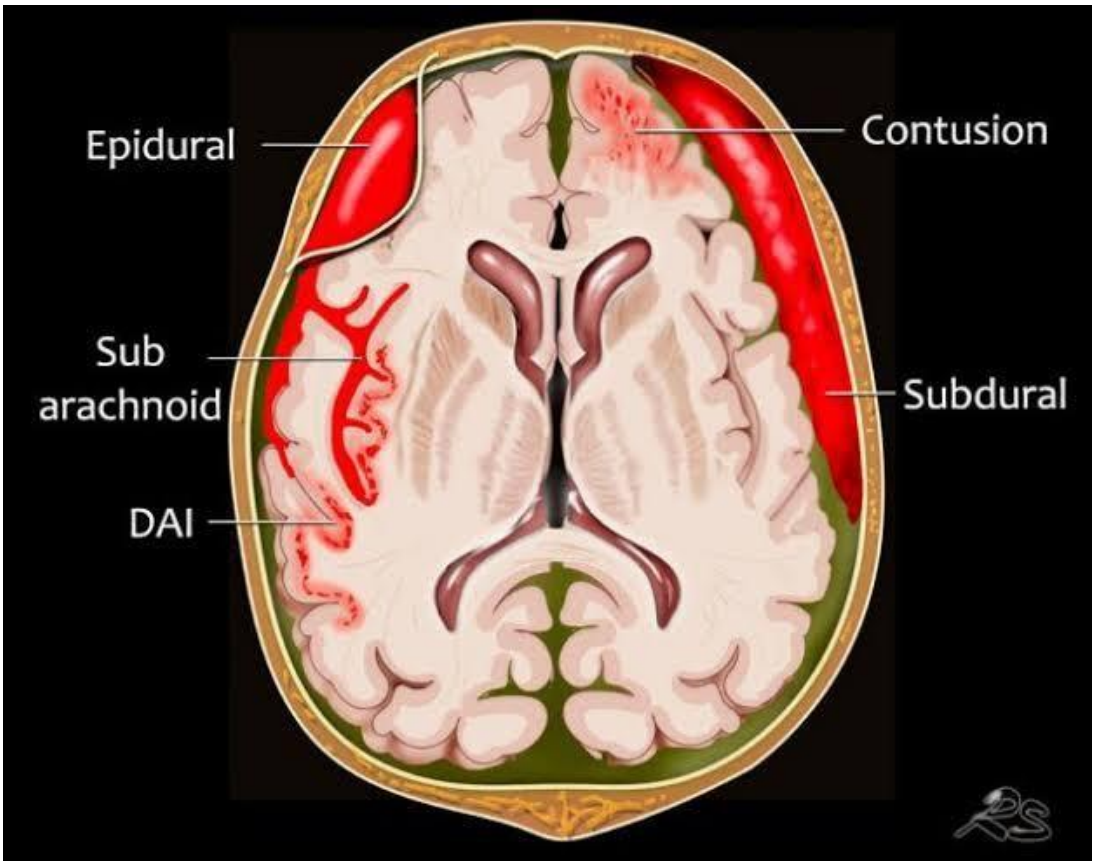


Fig1: Image depicting the most common types of traumatic brain injury.

Subarachnoid haemorrhage

One of the many different forms of injuries that make up TBI, traumatic subarachnoid haemorrhage (tSAH), is a significant contributor to morbidity and functional disability. Traumatic subarachnoid haemorrhage (tSAH), one of the many types of injuries that make up TBI, is a major factor in morbidity and

functional disability. The incidence of tSAH varies from 26% to 53% in people with TBI. In individuals with TBI, the incidence of tSAH ranges from 26% to 53%.(23).

While there is no confirmed cause for tSAH, the following are hypothesised mechanisms: Brief oscillatory movements of the brain are caused by: 1. rotational acceleration; 2. hyperextension stretching the vertebrobasilar artery; 3. tearing of the bridging veins or pial vessels; 5. blood diffusion from contusion into the subarachnoid space; and 5. a sudden rise in intra-arterial pressure from a blow to the cervical carotid artery.No cause is always apparent.

The largest quantity of subarachnoid haemorrhage (tSAH) was seen in a mixed pattern involving the cortical sulci (33.33%), the interhemispheric space (11.96%), and the cerebral hemispheres and the basal cisterns (39.31%). It is possible to separate or combine a subarachnoid haemorrhage with a Rotherham study (25). The highest tSAH concentrations were found in the cerebral hemispheres and basal cisterns (11.96%), followed by the cortical sulci (33.33%) and pace (39.31%).

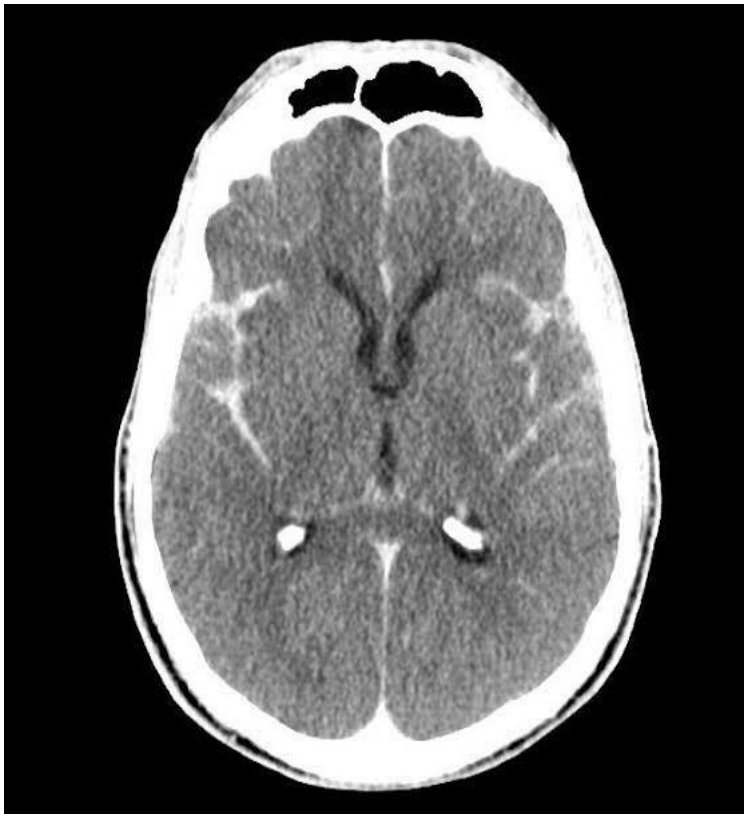


Fig 2: CT image of brain showing bilateral parietal subarachnoid hemorrhage.

The Glasgow coma scale post tSAH majorly determines the severity of head injury, mortality and morbidity. The electrocardiographic changes observed in patients post tSAH is varied. It could be tachycardia, QT prolongation to arrhythmia. There are two mechanisms that might mediate ECG changes in patientwith SAH, i.e. autonomic neural stimulation from the hypothalamus or elevated levels of circulating catecholamine(26)

Subdural haemorrhage

It is the blood clot that forms in the space between the dura and arachnoid layers of the brain, due the rupture of the dural bridging veins.The incidence of tSDH is more with advancing age due to the cerebral atrophy and wider subdural space (27).Even a minor trauma can result in tSDH in chronic alcoholics. SDH appears as a crescent shape hyper dense lesion on non contrast CT scan.

Subdural haemorrhage occupies the space intracranially and thus increases the pressure in the cranium significantly, leading to life threatening complications.

Subdural haemorrhage can be acute or chronic. Acute subdural haemorrhage usually occurs pot trauma to the brain, however it can also occur in patients with bleeding disorders, thrombophilia. Chronic subdural haemorrhage is common in the old age and alcoholics due to repeated minor trauma that goes unnoticed.

Cardiac abnormality in patients primary brain insult has been studied in many studies, however the electrocardiographic changes in patients post subdural haemorrhage has not been studies in particular.

A study was conducted by Busl et al in 2013 to specifically investigate the occurrence of cardiac abnormalities in patients with subdural haemorrhage, the results showed that no particular cardiac abnormalities were present in patients with subdural haemorrhage. The study did not show the occurrence any particular neurogenic changes in the electrocardiogram of these patients.

Although neurocardiogenic reasons are frequently attributed to the cardiac abnormalities in acute intracerebral damage, these are unlikely to be important process in subdural haemorrhage.

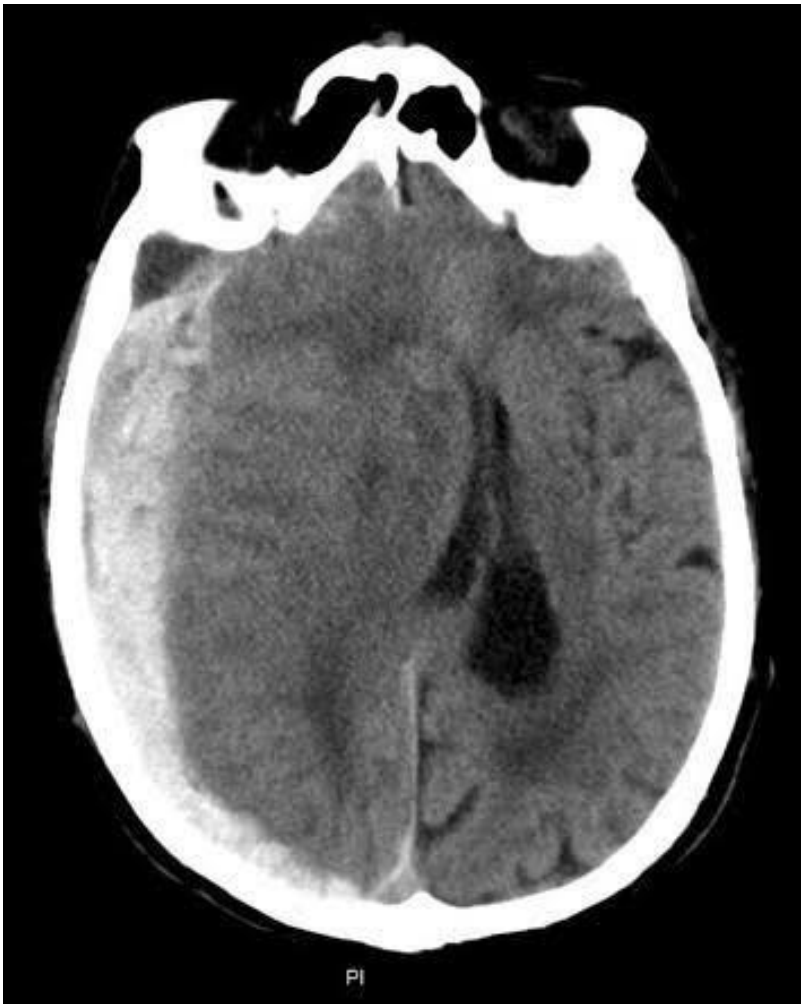


Fig 3: CT image of the brain showing right subacute subdural hemorrhage.

Epidural hematoma

It is the extra axial collection of blood between the inner table of skull and the dura mater. It is confined by the lateral sutures where the dura inserts, hence it does not cross suture lines. It occurs due to injury to middle meningeal artery. In 10 percent of cases extradural hematoma occurs due to venous bleed that is due to laceration of dural venous sinuses.

It occurs in ten percent of patients with TBI. Incidence is more common in males than in females, furthermore the usual age group which it is observed is in 20 to 30 years and rare after 50 to 60 years. With advancing age the dura matter adheres firmly to the overlying bone which may be the main cause of decreased incidence of extradural hematoma with advancing age.

Although trauma is most common cause of extradural hematoma, there are certain non traumatic causes of extradural hematoma , which includescoagulopathy, hemorrhagic tumours, vascular malformations.

In adults almost 75 percent of extradural hematoma occurs in temporal region.Clinical presentation includes initial loss of consciousness, followed by period of lucid interval then obtundation with focal neurological signs. It accounts to 1% - 5% of TBI (28).

Extradural haemorrhage on noncontrast CT scan appears as a biconvex hyper dense lesion. Based on radiographic features, extradural hematoma is divided into three types

Type 1 : Acute; begins on day 1 and is accompanied by a "swirl" of blood that isn't clotted.

Type 2: Subacute, lasting two to four days, and typically solid .

Type 3: Chronic, 7–20 day occurrence; mixed or lucent look with contrast enhancement. Type III.

The usual presentation of a case of extra dural is initial loss of consciousness followed by period of consciousness called that lucid interval and then control neurological detioration. The lucid interval can occur in patients who have various growing mass lesions and is not pathognomonic for an EDH. Pure EDHs that are quite big and exhibit active bleeding on a CT scan show the classic lucid interval.

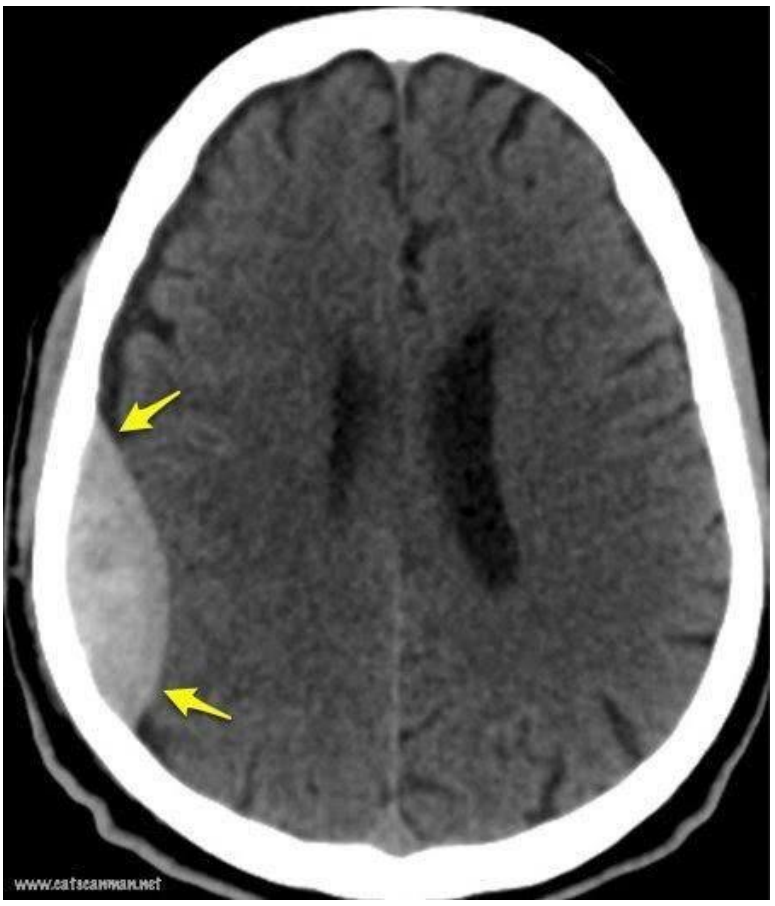


Fig 4: showing CT image of right parieto temporal extradural hemorrhage.

Intracranial pressure

Intracranial pressure is the pressure within the craniospinal compartment. The normal value of intracranial pressure varies with age. ICP levels in healthy persons should be between 5 and 15 mmHg, with increases to 30 mmHg being deemed pathological and 40 mmHg being life-threatening.(39)

The ICP changes during normal physiological conditions like coughing, sneezing, and can go up to 30 -50 mm hg , but it then rapidly returns back to normal.

There have been multiple cases of elevated intracranial pressure (ICP) following a neurological injury, including hydrocephalus, traumatic brain injury idiopathic intracranial hypertension (IIH), cerebral oedema and stroke.

Intracranial pressure can be affected by changes in CSF. The balance between cerebral blood inflow and outflow, which is a closed system made up of the brain, blood, and CSF inside the stiff, non-expandable skull, is crucial for maintaining a normal ICP.

Based on Monro - Kellie theory, change in the volume of brain, blood, or CSF will cause a corresponding change in one or both of the other two. In the event that this is not feasible, a further rise in volume will result in a rise in ICP.(38)

Cerebral perfusion pressure can be lowered by acute ICP rise (CPP)

$$CPP = MAP - ICP.$$

When the autoregulation of the cerebral blood arteries is compromised (as it is during a stroke), a major rise in ICP can result in alterations in brain perfusion that can affect CPP. Chronically increased ICP circumstances can also

cause papillary oedema, loss of eyesight, and death. Numerous CNS disorders, including hydrocephalus, traumatic brain injury, intracerebral haemorrhage, subarachnoid haemorrhage, ischemic stroke have been linked to increased intracranial pressure as a symptom or complication.(39)

Traumatic brain injury and Cardiac dysfunction

Traumatic brain injury has been linked to cardiac dysfunction, and experimental data suggests that interactions between the brain, heart, and lungs may contribute to myocardial dysfunction in TBI. (32).

In addition to electrocardiographic changes that resemble an ischemic episode and aberrant repolarization, patients with brain injuries may also experience reduced heart rate variability, which is indicative of autonomic nervous system dysfunction. (33)

After a variety of neurologic traumas, such as seizures, ischemia, SAH, and even mental stress, cardiac malfunction has been reported.

A catecholamine excess condition, which may be a contributing factor in the inflammatory cascade and lead to cardiac dysfunction, neurogenic pulmonary oedema, and probably other organ damage, is essential to the pathophysiology of brain-heart interactions (1). Findings on myocardial biopsy of patients with neurologic injury-induced brain death that are compatible with contraction band necrosis, the typical cardiac lesion of a catecholamine-excess condition, provide additional support for this. (34)

ELECTROCARDIOGRAPHIC CHANGES

TBI-related electrocardiographic (ECG) changes include ST-segment deviations, T wave inversions, and Q waves, as well as disturbances in rate, rhythm, P wave, QRS complex, PR interval, T wave, and ST segment. (5)

An analysis of the subgroups of TBI patients revealed that subarachnoid haemorrhage (49-100%), intracranial haemorrhage, ischemic stroke, cerebral venous thrombosis, head trauma, neurosurgical procedures, cryohypophysectomy, acute meningitis, intracranial space-occupying tumours, and epilepsy are the conditions where ECG changes occur most frequently (18)

QT prolongation :

The most common ecg abnormality observed in traumatic brain injury is QT prolongation. QTc- prolongation is more prevalent in men (25).

QTc-prolongation (20) and lesions in the right-sided insular cortex were more frequently associated with ECG abnormalities.(20)

Research has shown that stimulating the left insular cortex causes parasympathetic cardiac responses with the right insular cortex showing sympathetic responses (21).

In a cohort study on association of ecg changes with TBI, conduction disorders occurred in 45%, including mostly QTc-prolongation and arrhythmias, mostly of supraventricular origin, were present in 38% of patients.(22)

Repolarization abnormalities

The ECG changes observed in a patient with acute myocardial infarction and in cerebral vascular accident are sometimes very similar, notably the STsegment changes.

ST-segment abnormalities were the most commonly reported ventricular repolarization disorders in TBI. St changes and t waves changes are also seen in patients with stroke. Studies have reported independent association of repolarization abnormalities with outcome in patients with acute TBI.(2)

Up to 55% of SAH patients have also been observed to have inverted or flat T waves.(35) St changes and changes in T wave morphology has also been observed in patients with intracranial bleed.

Autopsy examination of 5 patients died of SAH and had ECG alterations provides more concrete data. There were no indications of epicardial coronary disease in any of the autopsied subjects. (36)

Twelve patients with acute sub arachnoid haemorrhage and ST elevation in the ECG underwent thorough cardiac evaluations by Kono and colleagues. Although the echocardiography revealed anomalies in the apical wall motion of the individuals, an angiography showed no signs of stenosis or vasospasm of coronary artery.(37)

Raised ICP and ECG

Electrocardiographic changes that are seen in traumatic brain injuries , many of the E.C.G. abnormalities are related to changing intracranial pressure.

A study conducted by S.J.Jachuck et al, on electrocardiographic abnormalities in raised intracranial pressure, confirmed positive association between raised intracranial pressure and electrocardiographic changes.

Serial electrocardiography were performed on patients with intracranial conditions, their intracranial pressure were continuously monitored by Konigsberg extradural transducer. The most common electrocardiographic changes that were observed in the patients with raised intracranial pressure were prominent U waves, ST changes, tachycardia, qt prolongation. (40)

The changes in blood pressure, heart rate, respiration following injury to the central nervous system was first described by Cushings .(41)

These brain heart relation was further observed in many studies, which demonstrated variety of cardiovascular changes including increase or decrease in heart rate, cardiac arrhythmia, alteration in systemic or pulmonary vascular resistance. All these reflect changes secondary raised ICP than injury to brain itself.(42)(43)

Glasgow coma scale

Glasgow coma scale is a neurological scoring scale applied to patients to assess the level of consciousness and severity of acute brain injury. Graham Teasdale and Bryan Jennett, published the Glasgow Coma Scale for the first time in 1974.(29)

The scale rates patients based on their eye-opening, muscular, and verbal responses—the three components of responsiveness. The Glasgow Coma Scale's response levels are "scored" on a scale of 1 for no response to 4 for eye-opening, 5 for verbal response, and 6 for total response (Motor response)

Thus, the overall Coma Score ranges from three to fifteen, with three being the lowest and fifteen being the greatest. (30)

best eye reaction (4)

1. No eye movement

2. Eyes open to discomfort 3. Eyes open to sound

4. Eyes open on their own

best response in speech (5)

1. There is no spoken reply

2. Sounds that are unclear

3. inappropriate language

4. Confused

5. Orientated

optimal motor reaction (6)

1. No movement reaction

2. abnormal flexion and extension in response to pain

3. abnormal flexion and withdrawal from discomfort

5. Pinpointing pain

6. Follows orders

Although the scale is widely used , to guide treatment in acute brain injury, there are conditions which can interfere with the proper assessment, Language barrier, speech impairment, intellectual deficit to name a few.

Gennarelli et al. demonstrated the existence of a continuous, progressive association between increasing mortality following a head injury and decreases in GCS Score from 15 to 3, illuminating the relationship between assessments of the GCS (typically reported as the total GCS Score) and the outcome.(31)

Electrocardiograph-a overview

An electrocardiogram, often known as an EKG or ECG, is a recording of the electrical activity of the heart. Willem Einthoven first invented it in 1902. The initial evaluation of a patient who may have cardiac-related problems must include an EKG. It is a non invasive diagnostic tool, widely used for assessing cardiac function and electrical abnormalities.

The conventional 12 lead ECG consists of 6 limb leads 3 precordial leads. The limb leads are called lead 1, lead 2, lead 3 and lead avL, avF, avR.

The six precordial leads are V1 , V2,V3,V4,V5,V6. Color-coding the limb leads helps prevent misplacing them.

Precordial leads are placed on the chest surface.

V1 is located to the right of the sternal boundary, and V2 is positioned to the left (47)

V4 is positioned at the level of the fifth intercostal gap on the mid-clavicular line. V4 should follow after V3.

Between V2 and V4 is V3, in that order.

V4 and V6 are next to each other, followed by V5.

V6 is positioned on the mid-axillary line at the level of the fifth intercostal gap.

V4 through V6 and the fifth intercostal gap should be aligned horizontally. The 25 mm/sec is the electrocardiograph's movement rate. Voltage and time are shown along the y-axis. One second is split into five substantial squares on the x-axis, each of which represents 0.2 seconds. Each box is further divided into 5 small boxes of 0.04s each.

The amplitude is 1mV for 1 small box and is 1mm in length.The best way to read a ECG is to read is systematically.

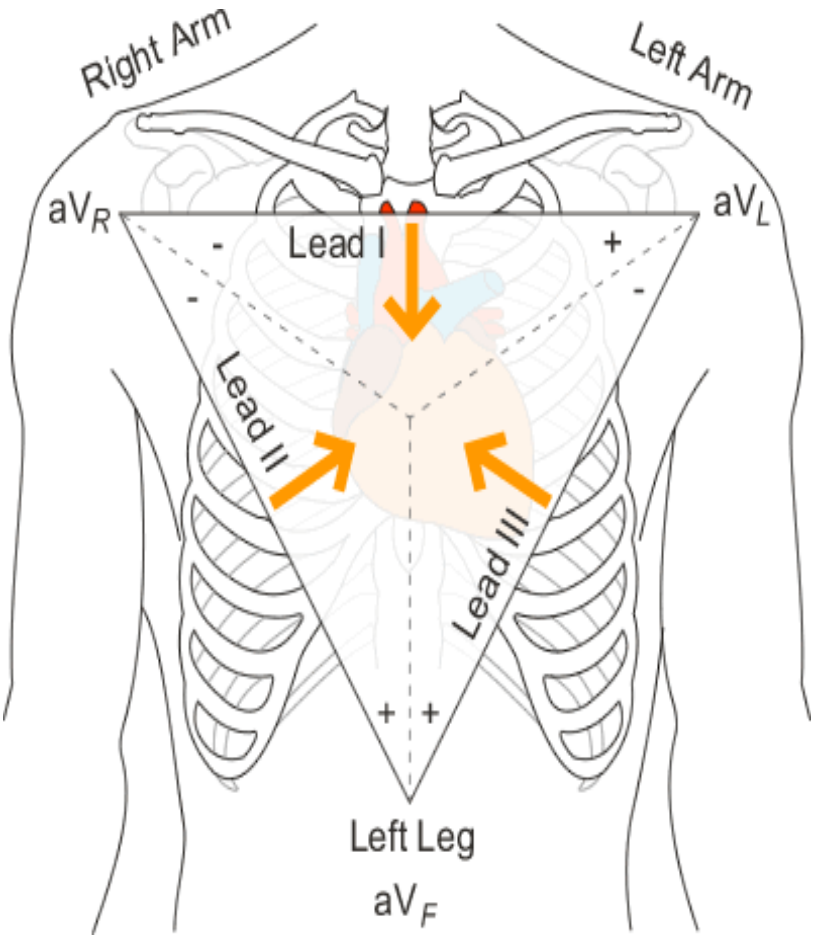


Fig 5: Einthoven triangle of electrocardiogram

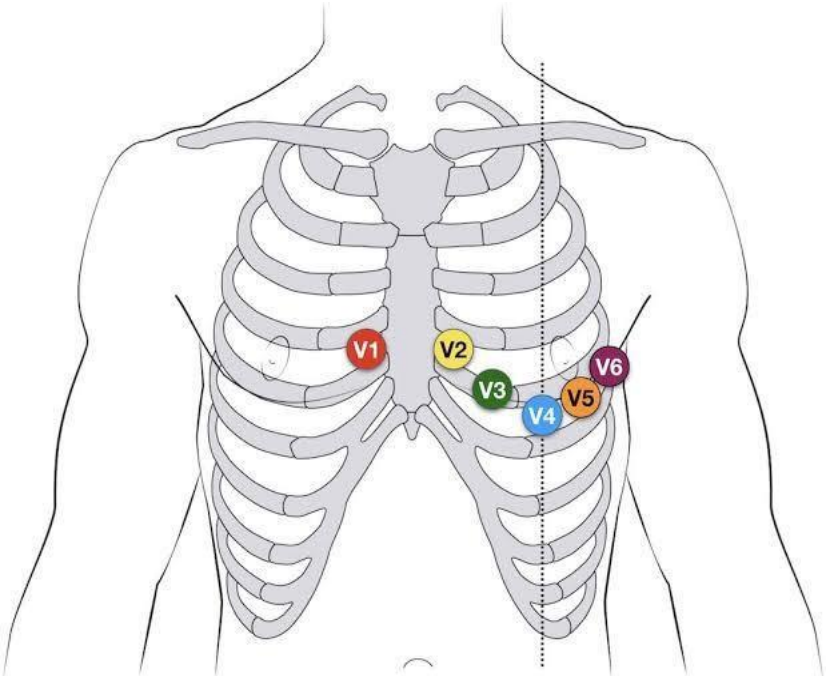


Fig 6: pictorial representation of precordial lead placement for ECG recording.

Rate

It denotes the number of times heart beats in a minute. Rate is calculated by counting the number of large boxes between two consecutive R waves , this number is then divided with 300.

If we are counting the number of small boxes between two consecutive R wave , the rate is obtained by dividing 1500 by number of small boxes. This

calculation of heart rate is correct if the patient has a regular rhythm. If the rhythm is irregular, then the number of beats in a ten second strip should be counted and multiplex by 6.(48)

The normal heart rate is between 60 to 99 beats per min. If the rate is more than or equal to 100 , it is termed tachycardia. If the rate is less than or equal to 59 then it is termed as bradycardia .

Rhythm

For interpretation of rhythm requires careful inspection of lead 1, 2 avF and V1. For accurate rhythm assessment, these 5 points to be considered. The presence or absence of regular P wave. The duration of Qrs complex ,whether it is narrow or broad. The relation between p wave and qrs complex. The morphology of p waves. The regularity of rhythm.

Normal sinus rhythm has positive p wave is lead 1, 2 , avF and suggests downward progression of cardiac impulse from sink atrial node. The change in position and morphology of p waves gives information about origin of the rhythm.

Cardiac axis

It denotes the direction of depolarisation of heart in the frontal plane. The normal cardiac axis is -30 to 90 degree. There are various methods for calculating the axis , the most common method used is the quadrant method. In this the leads 1 and avF are observed for predominant posited or negative deflection of the qrs complex.

If both 1and avF showing predominant positive deflection then it is normal axis.

If lead 1 is positive and lead avF is negative then it is left axis deviation (between 0 and -90 degrees).

If lead avF is positive and lead 1 is negative then it is right axis deviation (+90 and 180 degrees).

If both lead 1 and avF are negative then it is either extreme right axis deviation or intermediate axis (-90 to 180 degrees)(49).Apart from these there are other methods of axis calculation, like the three lead analysis, isoelectric lead analysis.

There are several conditions in which the axis can be deviated. Left axis deviation is found in left ventricular hypertrophy, old lateral wall myocardial infarction, left bundle branch block.

Right axis deviation can be seen in right ventricular hypertrophy, pulmonary hypertension, wolf Parkinson's white syndrome, hyperkalemia.

P wave

It denotes the initiation of normal atrial activity. It has two components, the first half of the p wave denotes right atrial depolarisation and the second half of the p wave denotes left atrial depolarisation.

P wave is best viewed in lead 2 and V1.

In lead 2, it appears as a pyramidal shaped wave with 3 small square width and 2.5mm high. It is always positive in lead 1 and 2, consistently negative in lead avR. P wave is biphasic in lead V1, having an initial positivity and a terminal negativity.

Duration of P wave - 0. 08 to 0. 10 sec in lead 2

The maximum amplitude is 2. 5 mm in lead 2. The amplitude of initial positive deflection is 1. 5mm. Duration is 0. 05 sec and does not exceed 0.

08sec.Terminal negative deflection should not exceed 1mm in depth and 0. 03 sec in duration.

P are tall in case of right atrial enlargement in which case it is called as the p pulmonale , the amplitude of the initial deflection more than 2.5mm with possible right axis deviation. P wave in duration and amplitude of the final component both increase with left atrial hypertrophy. It must be at least 0.04 seconds (40 ms) wide and dip at least 1 mm below the isoelectric line in lead V1. The left atrium does not exhibit any axis deviation because it is electrically dominant.It is termed as P mitrale.(50)

Conditions with tall p waves are

P Congenitale

COPD

Congenital heart diseases

PR segment

It denotes the ending of atrial depolarisation and beginning of ventricular depolarisation. It is the atrioventricular nodal delay in conduction of electrical impulse. This delays allows for complete atrial emptying into the ventricles.

The normal PR interval is between 3 to 5 small boxes and is 120 ms to 200ms in duration. Many conditions alter the duration of PR segment. Long PR segment indicates that there is conduction block .

Short PR interval is seen in accelerated AV conduction like wolf Parkinson's white syndrome, ventricular ectopic, Lown-Ganong-Levine syndrome. (51)

First degree heart block: Prolongation of PR interval to more than 20ms , every p wave is followed by a qrs complex. The duration of PR interval remains constant throughout. It might be normal is certain individuals but may also be denoting underlying structural heart disease or Hypokalemia, severe rheumatic fever, and transitory myocarditis or drug toxicity manifestations. Usually requires no treatment.

Second degree heart block: it is further of two types the Mobitz type 1 also called the wenkebach phenomenon and Mobitz 2.

In Mobitz type 1 there is progressive prolongation of PR interval and subsequently a missed qrs beat. It can be due to myocardial ischemia or infarction. It usually does not require any intervention but can progress to third degree as well , in which case permanent pacemaker is the option. In certain cases patient may suffer syncope in which case atropine or pacemaker is adviced.

Mobitz type 2 : In this type of second degree heart block , there is missed qrs complex, without any progressive prolongation of PR interval. It is all or none phenomena. It almost always progressed to third degree heart block and thus requires intervention like permanent cardiac pacemaker.(53)

Third degree heart block : There is complete dissociation between between p wave and qrs complex and there is no relation between the two. The atria is beating on its own, ventricle beats on its own. However the PP and RR interval remains constant. It is usually due to complete degeneration of the conducting system , inferior wall myocardial infarction . This requires placement of permanent cardiac pacemaker(53).

Bundle branch block: Conduction block of the left or right bundle branch leads to a bundle branch block. It is identified by looking at the width and arrangement of the QRS complexes. On the ECG, the right bundle branch block is

denoted by a broadened QRS complex longer than 0.12 seconds and an RSR pattern in V1 and V2. Leads V5, V6, I, and aVL may also exhibit reciprocal alterations, ST-segment depression, T wave inversions, and other abnormalities. (52)

Bundle branch block is the outcome of the left or right bundle branches' conduction block. The width and arrangement of the QRS complexes are analysed to diagnose it. A broadened QRS complex longer than 0.12 seconds and an RSR pattern in V1 and V2 are indicators of the right bundle branch block on the electrocardiogram (ECG). Additionally, leads V5, V6, I, and aVL may exhibit reciprocal alterations, T wave inversions, and ST-segment depression.

(52)

Qrs complex

It denotes the ventricular depolarisation. The duration is usually less than 3 small squares that is 120 ms. (60-100ms)

Qrs complex has the initial negative deflection called the q wave is a tiny negative deflection in leads I, aVL, V5, and V6 . It denotes interventricular septum depolarisation. ECG pathological Q-waves may indicate an old infarct.

Pathologic Q-waves are those that last longer than 40 ms (one tiny box), are deeper than 1 mm, or are larger than 25% of the amplitude of the QRS complex.The initial positive deflection R-wave , it progresses from right to left in the precordial leads and gradually increases in amplitude. The R wave is weakest in lead V1 and biggest in lead V5. Several factors, such as anteroseptal MI in the past, left ventricular hypertrophy, and improper lead placement, might result in a decreased R-wave progression. The following adverse deflection The Purkinje fibres' complete depolarization is represented by the letter S. wave, it is any downward deflection. It might not be found in every ECG lead. altogether ismaller n the lead V6.(53)

R wave is because the left ventricle is thicker in width and the lateral precordial leads pick up the depolarisation coming toward then hence the positive. While the precordial leads V1-V3 see the charge going away from them , hence there is predominant S wave. This explains the R wave progression. (53)(54)

ST segment

It represents the termination of ventricular depolarization and the start of ventricular repolarization . The ST segment lasts, on average, less than two to three small squares (80-120 ms). The PR-interval and ST segments both lie at the same level on an isoelectric line. A 1 mm or greater elevation or depression of the ST segment, measured at the J point, is considered abnormal. The space between the QRS complex and the ST segment is known as a J point.

It is clinically relevant for the diagnosis of acute myocardial infarction if the vertical distance between the ECG trace and the baseline after the J-point is at least 1 mm in a limb lead or 2 mm in a precordial lead. Except for leads V1 and aVR, ST-elevations are diffuse in acute pericarditis and linked to PR depression about TP segments.

ST depression greater than 1 mm is often a sign of myocardial ischemia or angina.ST depression is also seen in non-ischemic causes, including digoxin toxicity, hypokalemia, hypothermia, and tachycardia.(55)

T WAVE

Deflection is produced by ventricular repolarization usually upright in left orientated leads. T wave in lead v6 > v1.

Tall T waves are seen in lead v2-v4. Inverted T waves in lead v1 to v3 in adulthood constitute persistent juvenile pattern. Amplitude of T wave > 5 mm is considered significant.

TALL PEAKED T WAVES

1) Acute sub endocardial ischemia or infarction

2) Hyperkalemia

3) Resolving inferior infarction

INVERTED T WAVES

Coronary heart disease

Pericardial effusion

Myxoedema

QT segment

QT indicates the Normal QT interval is 0.40s to 0.44s. The heart rate and a QT interval are inversely relatedQT indicates the starting of ventricular depolarisation till the end of depolarisation of ventricles . entricular tachycardia, ventricular fibrillation, and Torsades de Pointes.Common causes for QT prolongation are hypocalcemia and hypomagnesemia, and congenital long QT syndrome.(57)

Less than 360 milliseconds in length, the short QT interval can be caused by hyperkalemia, acidosis, hypercalcemia, hyperthermia, or short QT syndrome.

PROLONGED QTC

Sleep

Hypocalcaemia,

Acute myocardial infarction,

Procainamide effect,

Cerebral injury,

Hypothermia,

Jervell-lange neilson syndrome

Romono-ward syndrome

SHORTENED QTC

Digitalis effect

Hypercalaemia

Hyperthermia

Vagalstimulation

MEASUREMENT OF QT INTERVAL:

May at time present some difficulty because it may be difficult to determine the exact beginning and end of interval. The beginning of QRS complex is best determined in lead1, 11, avL, v5, v6. When QT interval is measured from a lead where U is prominent the dip or notch between T and U wave is taken as the end of T wave.

QT interval shortens with tacycardia and lengthens with bradycardia. This is QT shortens with diminution of R-R INTERVAL and lengthens with increase of R-R interval.

CORRECTION OF QT INTERVAL

Corrected QT interval is known as the QTC interval various formulae have been proposed for correction of QT interval. the most frequently used it the bazett.

BAZETS FORMULAE : QTc =QT/√(RR) QTc may be regarded as K constant

K=QT/√RR.

Normal value for k is 0. 39 to 0. 04 sec. Normal range is 0. 35 to 0.43 sec.

The value of QTC corresponds to QT duration at a heart rate of 60/m.

U WAVES

Normal U wave is small rounded deflection 1mm that follows T wave usually has polarity as T wave. An abnormal increase in u wave amplitude is most common due to drugs or hypokalemia. Very prominent U waves are marker of increased susceptibility to torsades de pointes type of ventricular tachycardia.

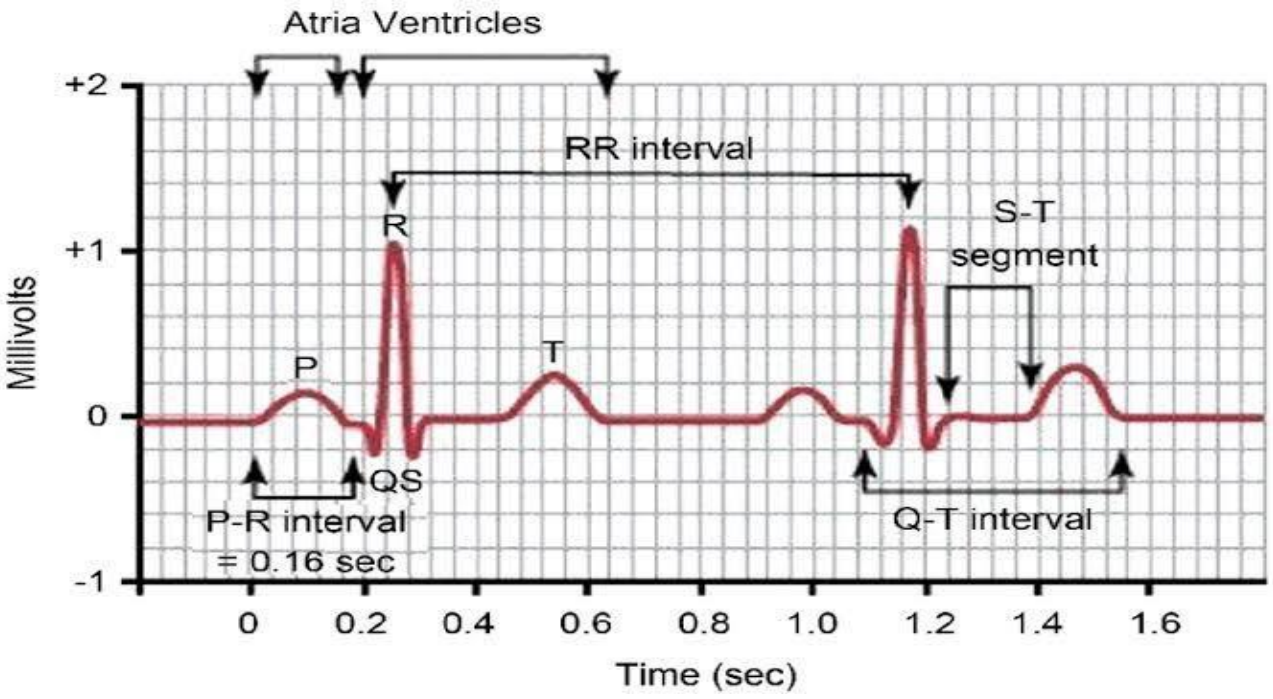


Fig 7: picture showing the components of ECG.

Glasgow Outcome scale

Glasgow outcome scale primarily used for disability and social participation assessment in patients with neurological injury. It is the most commonly used outcome measure in brain injury studies.

Bryan Jennett and Michael Bond first released the Glasgow Outcome Scale (GOS) in 1975. It is the most widely used evaluation scale for neurological injuries because to its simplicity, quick administration time, reliability and validity, stability, flexibility of administration, cost-free availability, and accessibility of access.The GOS can be used in a number of different contexts, including face-to-face or telephone interviews, mail, and, with a modified version, inpatient settings; this flexibility results in high rates of follow-up.(43)

Instead of focusing on the specific deficiencies and symptoms brought on by the injury, the scale concentrates on how functioning in significant areas of life has been affected. GOS is used on both adults and chlidren. The GOS's validity when used on kids, however, is unknown.

The GOS criteria appear to be essentially irrelevant for very young children. The present strategy is intended for usage with individuals 16 years of age and older.

The scale is meant to be used after hospital discharge, and in particular, good recovery and mild disability cannot be assessed until after discharge.

The Extended Glasgow Result Scale and the Disability Rating Scale were both based on this scale, which contains five degrees of outcome. (44)

Good recovery : A successful recovery means that the patient can return to their old job. Although the patient may still have a few small limitations, they are able to live independently.

Moderate recovery: A patient with a moderate impairment can live independently and use public transportation, but they frequently still have significant, ongoing physical or neurological deficits.

There will be clear deficit in the form of either limb weakness, deafness, loss of vision, cranial nerve paralysis. However these patients can perform most of the activities independently. There can even travel with some help or public transportation. (46)

Severe disability: A patient with a severe disability is conscious, but they need someone else's help every day.

These patients constantly require some persons help for the daily activities.Dependence varies from being bedridden to wanting a persons help to go out or to use bathroom.

Dependence is typically necessary due to a combination of a mental disease and physical impairment, such as paralysis or speech loss.46)

Some patients may have no physical deficits but they may have severe menta

Dependence is typically necessary due to a combination of a mental disease and physical impairment, such as paralysis or speech lossental disability is more than that in physical disability.

Persistent vegetative state: The person is bed ridden, has normal respiratory and periods of eye opening and closing, that suggest sleep wake cycle. He may have blank stare, look into direction of bright light or loud noise but does not , and remains unaware of the surroundings and the environment . There may be spontaneous movement of face muscles, trunk muscles.He also withdraws from painful limb stimuli.(44)

There will be severe damage of the bilateral cerebral hemisphere.

The persistent vegetative state is termed permanent when the irreversibility is established with high degree of certainty, that is if a patient with traumatic brain injury remains in the persistent vegetative state beyond 6 months of insult and patient with non traumatic brain injury remains in coma for more than 12 months.(45)

At one month after *trauma*, about 1/3 of patients in the vegetative state will show some improvement over the subsequent year. After *non-traumatic coma*, outcome is much worse; only about 7% show some improvement and have severe disability.(45)

Death: The outcome of a patient with neurological injury is graded 1 (dead), if he succumbs anytime within 1 month of insult.

The scale is further narrowed to favourable outcome and unfavourable outcome.

Glasgow outcome scale of 1 to 3 , that is death , persistent vegetative state, severe disability are considered to be unfavourable outcomes . While the Glasgow outcome scale of 4 and 5 that is moderate disability and good recovery are considered as favourable outcomes.

However glasgow outcome scale has it own limitations. It is considered too broad scale and cannot be applied to head injury secondary to concussion.Nonetheless, the Glasgow outcome scale still remains to widely accepted standard global outcome assessment post brain injury.

MATERIALS AND METHODS

MATERIALS AND METHODS

1. SOURCE OF DATA: The information for the study will be collected from patients coming to Emergency Medicine department BLDEU'S Shri B.M Patil Medical College Hospital and Research Centre, Vijayapur from January 2021 to June 2022.

2. METHOD OF COLLECTION OF DATA:

Information will be collected through prepared pro forma from patients with Head Injury. Qualifying patients will be undergoing detailed history, clinical examination and laboratory investigations

Inclusion Criteria:

1. Patients with head injury aged more than 18 years.

2. The patients with a diagnosis of traumatic brain injury with documented CT/MRI.

Exclusion Criteria:

1. K/C/O Hypertension

2. K/C/O Ischemic heart disease.

3. K/C/O Cardiomyopathy.

4. K/C/O Valvular Heart Disease,

5. Patients taking drugs that affect cardiovascular System

6. Patients with previously documented abnormal ECG

3. TYPE OF STUDY:

Single centre prospective study.

Sample size calculation

With the anticipated proportion of the ECG abnormality among head injury 80% [2] , the study would require a sample size of 471 with 97% confidence level and 4% absolute precision

By using the formula:

$$n = \frac{z^2 p(1-p)}{d^2}$$

Where

Z= z statistic at 4% level of significance

d is margin of error

p is anticipated prevalence rate (80%)

Statistical analysis

The data obtained will be entered in a Microsoft excel sheet, and statistical analysis will be performed using statistical package for the social sciences (verson20)

Results will be presented as mean(median)+/- SD, counts, percentage and diagrams

For normally distributed continuous variables between two groups will be compared using independent t test

For normally distributed variables Mann Whitney U test will be used.

Categorical variables will be compared using Chi square test

Relationship between variables will be analyzed using Pearson's / Spearman's correlation

$P<0.05$ will be considered statistically significant. All statistical tests will perform two tailed

INVESTIGATIONS

Patients presenting to the emergency medicine department with history of traumatic head injury, who satisfy the inclusion criteria will undergo detailed history, clinical examination and following set of investigations.

Investigation :

1. 12 lead electrocardiograph will be recorded within 12 hours of admission to the hospital.
2. Radio imaging of the brain: CT scan of the brain or MRI scan of the brain- All patients with traumatic brain injury satisfying the inclusion and exclusion criteria will undergo either CT or MRI scan of the brain for the diagnosis and confirmation of type of brain injury.

Supplementary investigation:

- 2D echo
- Serum troponin I
- Serum Electrolytes
- CBC

RESULTS OF THE STUDY

A detailed analysis of electrocardiograph was done for all the patients.

The severity of brain injury was correlated with the electrocardiograph changes.

One month outcome of the patients assessed by Glasgow outcome scale, was correlated with the electrocardiograph changes.

A number of observations were made, which are given in the following tables from table 1-20

TABLE -1

BASELINE CHARACTERISTICS OF THE STUDY POPULATION

Sex		Frequency	Percent
Valid	Female	184	39.1
	Male	287	60.9
	Total	471	100.0

A total of 471 patients were followed up in our study.

39% of patients were females.

60.9% of patients were males.

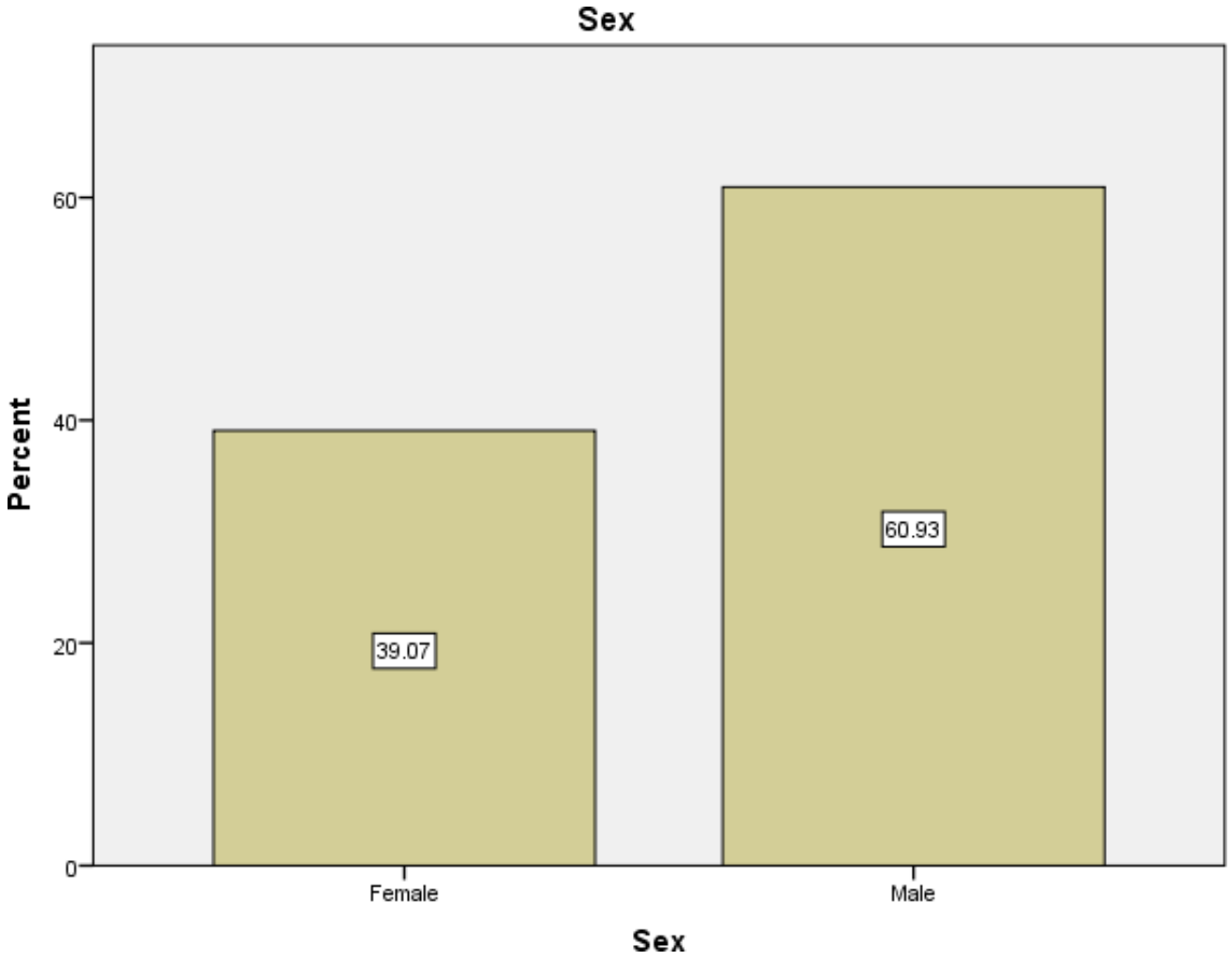


TABLE-2

INCIDENCE OF TYPES OF TRAUMATIC BRAIN INJURY

Type of traumatic brain injury		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Contusion	132	28.0	28.0	28.0
	Diffuse axonal injury	14	3.0	3.0	31.0
	Diffuse cerebral edema	25	5.3	5.3	36.3
	EDH	69	14.6	14.6	51.0
	IC Bleed	36	7.6	7.6	58.6
	IC bleed and SAH	3	.6	.6	59.2
	Intraventricular bleed	14	3.0	3.0	62.2
	SAH	115	24.4	24.4	86.6
	SDH	59	12.5	12.5	99.2
	SDH and SAH	4	.8	.8	100.0
	Total	471	100.0	100.0	

28% of patients suffered contusion.
24.4% of patients had subarachnoid hemorrhage.
14.6% of patients had extradural hemorrhage.
12.5% of patients had subdural hemorrhage.
7.6% of patients had intracerebral bleed
5.3% of patients had diffuse cerebral edema.
3% of patients had diffuse axonal injury.
3% of patients had intraventricular bleed.
0.8% of patients had subdural hemorrhage with subarachnoid hemorrhage.
0.6% of patients had intracerebral bleed with subarachnoid hemorrhage.

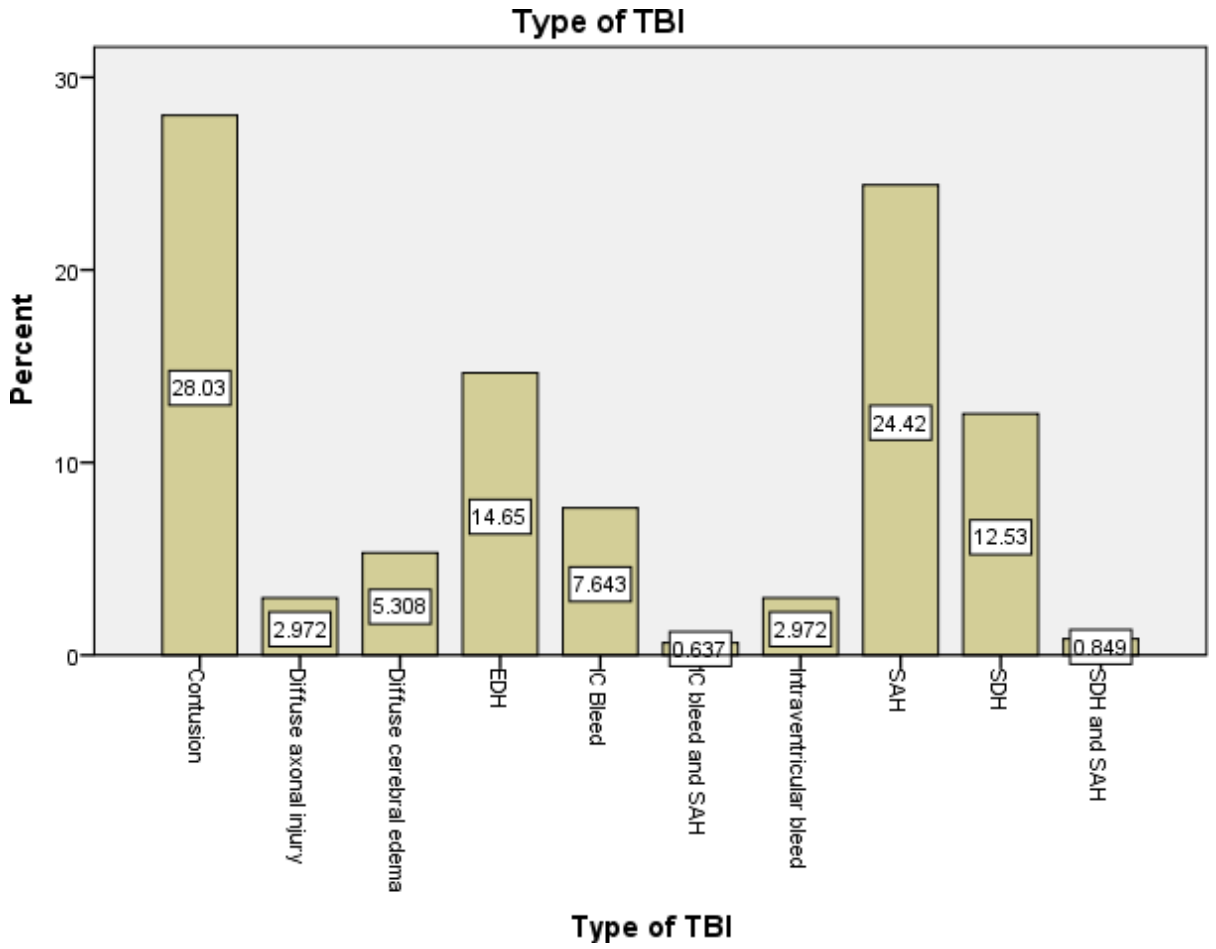


TABLE -3

INCIDENCE OF SEVERITY OF HEAD INJURY

SEVERITY OF HEAD INJURY GLASGOW COMA SCALE		Frequency	Percent	Valid Percent
Valid	mild	129	27.4	27.4
	Severe	156	33.1	33.1
	Moderate	186	39.5	39.5
	Total	471	100.0	100.0

27% of patients had mild head injury, indicating GCS score of 13 to 15

39.5% of patients had moderate head injury, indicating GCS score between 12 to 9

33.1% of patients had severe head injury, GCS less than or equal 8

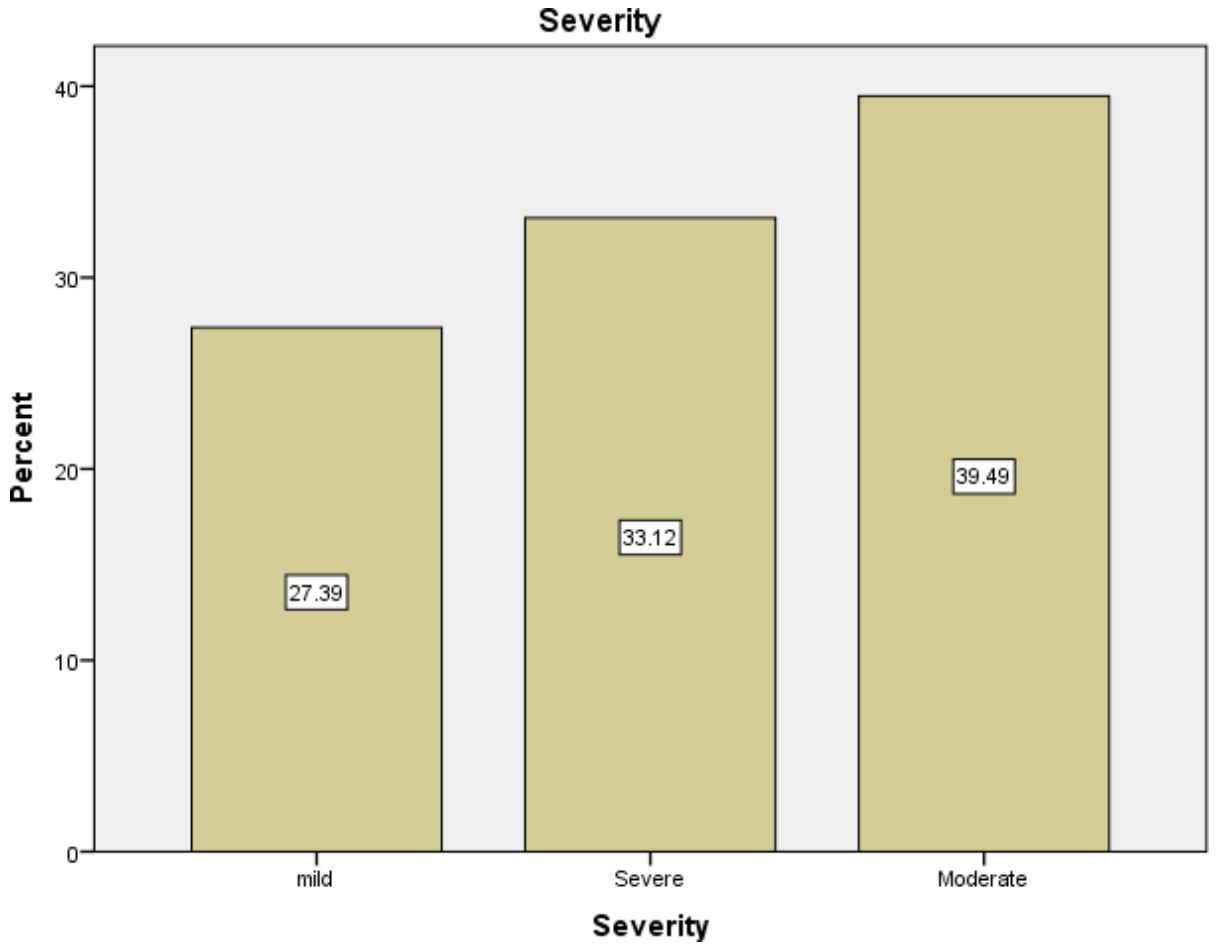


TABLE – 4

INCIDENCE OF ABNORMAL RHYTHM IN THE STUDY GROUP

			Severity			Total	Chi-square value	p-value
			mild	Severe	Moderate			
Rhythm	Atrial fibrillation	Count	0	0	2	2	68.880	0.000
		% within Severity	0.0%	0.0%	1.1%	0.4%		
	Complete heart block	Count	0	1	0	1		
		% within Severity	0.0%	0.6%	0.0%	0.2%		
	Junctional rhythm	Count	0	2	0	2		
		% within Severity	0.0%	1.3%	0.0%	0.4%		
	Normal	Count	97	74	113	284		
		% within Severity	75.2%	47.4%	60.8%	60.3%		
	Sinus bradycardia	Count	1	35	8	44		
		% within Severity	0.8%	22.4%	4.3%	9.3%		
	Sinus tachycardia	Count	31	42	63	136		
		% within Severity	24.0%	26.9%	33.9%	28.9%		
	Ventricular tachycardia	Count	0	2	0	2		
		% within Severity	0.0%	1.3%	0.0%	0.4%		
Total		Count	129	156	186	471		
		% within Severity	100.0%	100.0%	100.0%	100.0 %		

75.2% of patients with mild head injury had normal sinus rhythm.
24% of patients with mild head injury had sinus tachycardia.
0.8% of patients with mild head injury had sinus bradycardia.
60.8% of patients with moderate head injury had normal sinus rhythm.

33.9% of patients with moderate head injury had sinus tachycardia.
4.3% of patients with moderate head injury had sinus bradycardia.
1.1% of patients with moderate head injury had atrial fibrillation.
47.4% of patients with severe head injury had normal sinus rhythm.
26.9% of patients with severe head injury had sinus tachycardia.
22.4% of patients with severe head injury had sinus bradycardia.
1.3% of patients with severe head injury had junctional rhythm.
1.3% of patients with severe head injury had ventricular tachycardia.
0.6% of patients with severe head injury had complete heart block.

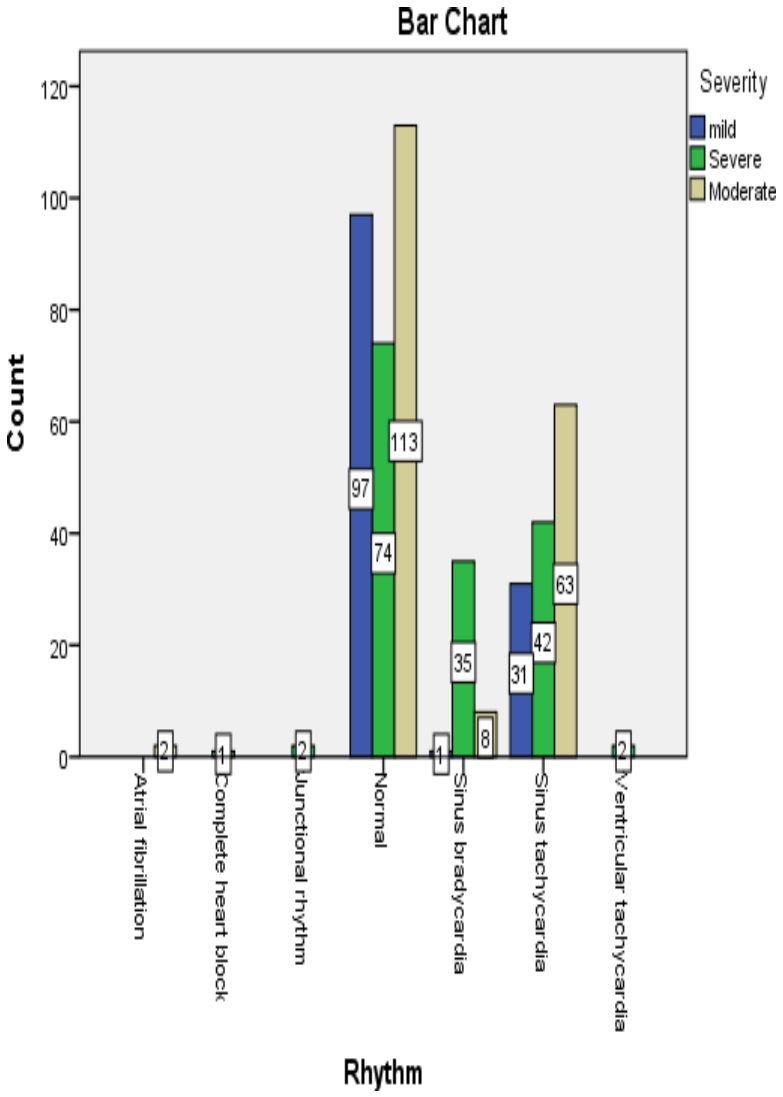


TABLE – 5

INCIDENCE OF ABNORMAL P WAVES IN STUDY POPULATION

			Severity			Total
			mild	Severe	Moderate	
P wave	Absent	Count	0	4	1	5
		% within Severity	0.0%	2.6%	0.5%	1.1%
	Fibrillatory p waves	Count	0	0	1	1
		% within Severity	0.0%	0.0%	0.5%	0.2%
	Normal	Count	129	152	184	465
		% within Severity	100.0%	97.4%	98.9%	98.7%
Total		Count	129	156	186	471
		% within Severity	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	6.748[a]	4	.150
Likelihood Ratio	7.601	4	.107
N of Valid Cases	471		

100% of patients with mild head injury had normal p waves.

98.9% of patients with moderate head injury had normal p waves.

0.5% of patients with moderate head injury had fibrillatory waves.

0.5% of patients with moderate head injury had absent p waves.

97.4% of patients with severe head injury had normal p waves.

2.6% of patients with severe head injury had absent p waves.

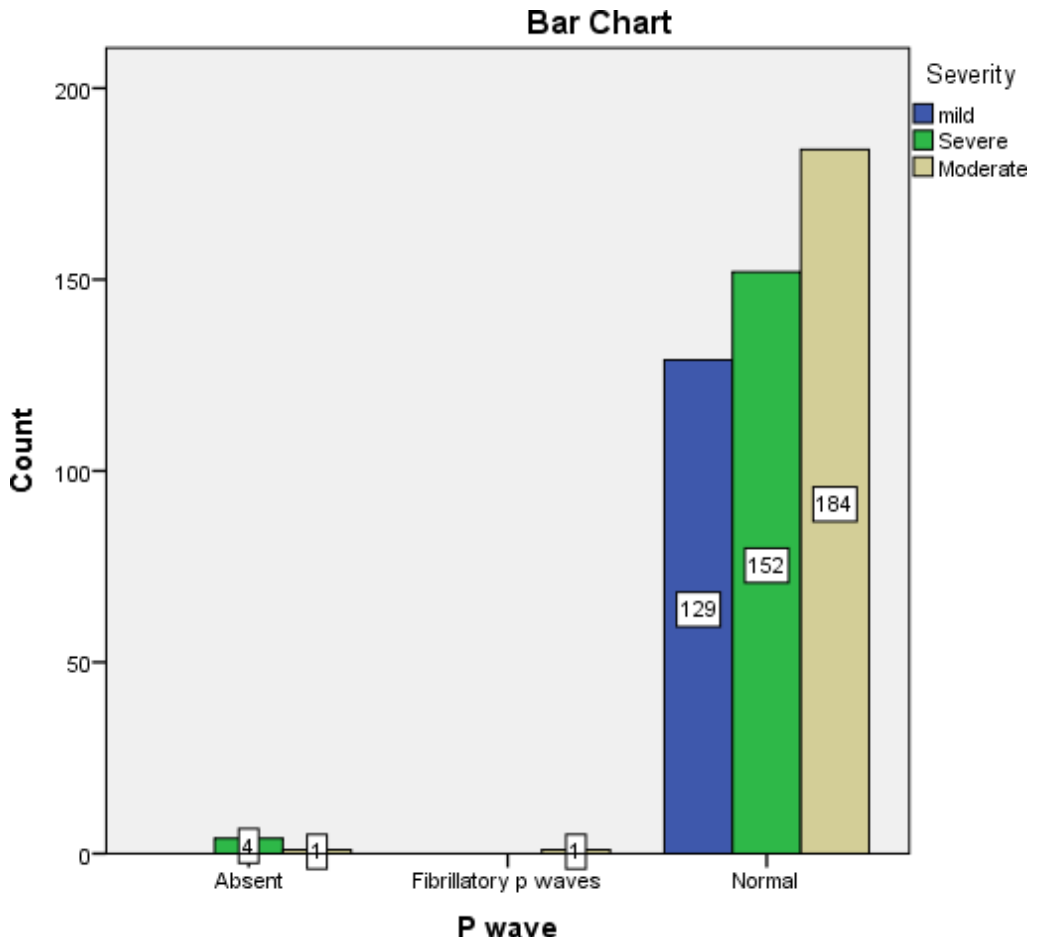


TABLE - 6

INCIDENCE OF ABNORMAL PR INTERVAL IN THE STUDY GROUP

PR interval* Severity Crosstabulation

			Severity			Total
			mild	Severe	Moderate	
PR interval	0	Count	0	3	2	5
		% within Severity	0.0%	1.9%	1.1%	1.1%
	Irregular	Count	0	1	0	1
		% within Severity	0.0%	0.6%	0.0%	0.2%
	Normal	Count	129	146	181	456
		% within Severity	100.0%	93.6%	97.3%	96.8%
	prolonged pr interval	Count	0	3	2	5
		% within Severity	0.0%	1.9%	1.1%	1.1%
	Short PR interval	Count	0	3	1	4
		% within Severity	0.0%	1.9%	0.5%	0.8%
Total		Count	129	156	186	471
		% within Severity	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	10.674[a]	8	.221
Likelihood Ratio	13.740	8	.089
Normal of Valid Cases	471		

100% of patients with mild head injury had normal PR interval

97.3% of patients with moderate head injury had normal PR interval

1.1% of patients with moderate head injury had prolonged PR interval

0.5% of patients with moderate head injury had short PR interval

93.6% of patients with severe head injury had normal PR interval

1.9% of patients with severe head injury had prolonged PR interval

1.9% of patients with severe head injury had short PR interval

TABLE -7

INCIDENCE OF ABNORMAL QRS INTERVAL IN THE STUDY GROUP

Crosstab

			Severity		
			mild	Severe	Moderate
QRS complex	Broad qrs complex	Count	0	2	0
		% within Severity	0.0%	1.3%	0.0%
	Left bundle branch block	Count	0	3	1
		% within Severity	0.0%	1.9%	0.5%
	Normal	Count	129	150	184
		% within Severity	100.0%	96.2%	98.9%
	Right bundle branch block	Count	0	1	1
		% within Severity	0.0%	0.6%	0.5%
Total		Count	129	156	186
		% within Severity	100.0%	100.0%	100.0%

Crosstab

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	8.359[a]	6	.213
Likelihood Ratio	9.824	6	.132
N of Valid Cases	471		

100% of patients with mild head injury had normal qrs complex.
98.9% of patients with moderate head injury had normal qrs complex.
0.5% of patients with moderate head injury had left bundle branch block .
0.5% of patients with moderate head injury had right bundle branch block .
96.2% of patients with severe head injury had normal qrs complex.
1.9% of patients with severe head injury had left bundle branch block .
0.6 % of patients with moderate head injury had right bundle branch block .

			Severity			Total
			mild	Severe	Moderate	
st segment	STelevation	Count	10	6	10	26
		% within Severity	7.8%	3.8%	5.4%	5.5%
	0	Count	1	0	1	2
		% within Severity	0.8%	0.0%	0.5%	0.4%
	Normal	Count	118	148	174	440
		% within Severity	91.5%	94.9%	93.5%	93.4%
	st depression	Count	0	2	1	3
		% within Severity	0.0%	1.3%	0.5%	0.6%
Total		Count	129	156	186	471
		% within Severity	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	5.012[a]	6	.542
Likelihood Ratio	6.150	6	.407
N of Valid Cases	471		

91.5% of patients with mild head injury had normal ST segment .

7.8% of patients with mild head injury had ST segment elevation .

93.5% of patients with moderate head injury had normal ST segment .

5.4 % of patients with moderate head injury had ST segment elevation .

0.6% of patients with moderate head injury had ST segment depression .

94.9% of patients with severe head injury had normal ST segment .

3.8 % of patients with severe head injury had ST segment elevation .

1.3% of patients with severe head injury had ST segment depression .

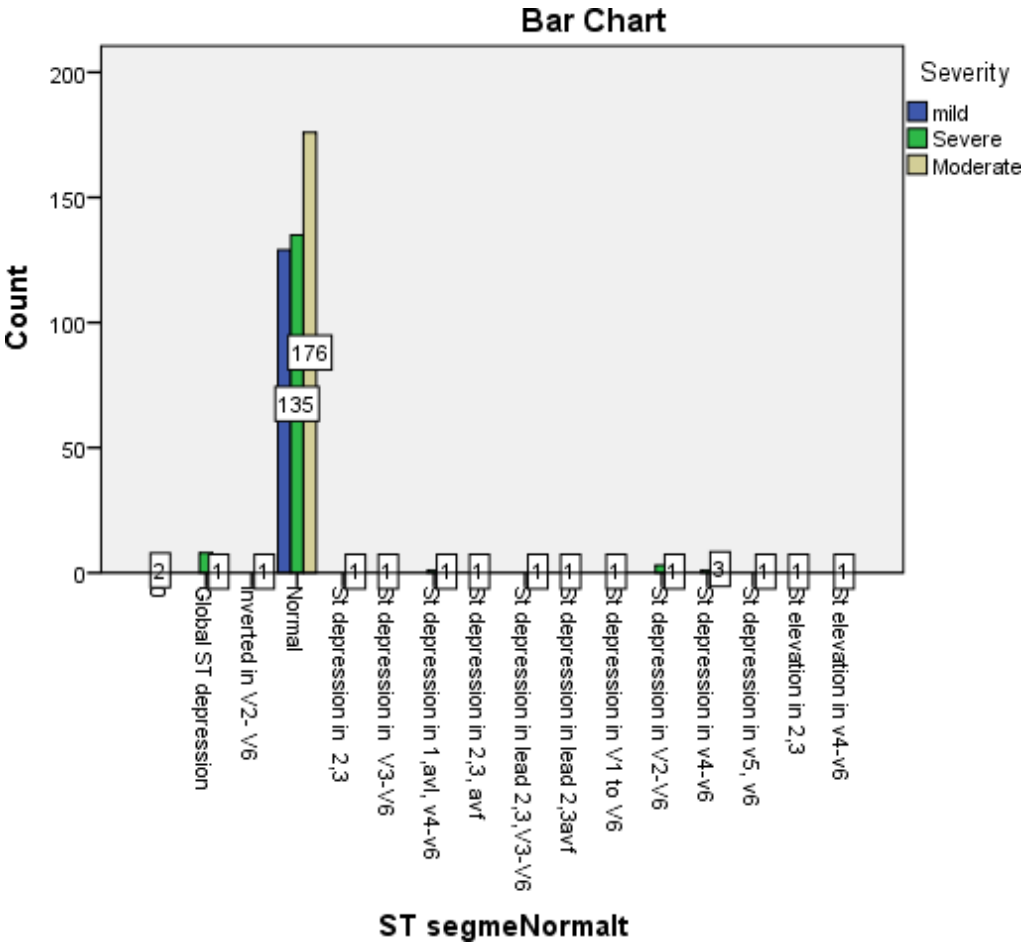


TABLE 8

CORRELATION BETWEEN SEVERITY OF HEAD INJURY AND T WAVE CHANGES

Crosstab

			Severity			Total
			mild	Severe	Moderate	
T wave	0	Count	0	2	0	2
		% within Severity	0.0%	1.3%	0.0%	0.4%
	Normal	Count	128	133	181	442
		% within Severity	99.2%	85.3%	97.3%	93.8%
	t wave inversion	Count	0	14	1	15
		% within Severity	0.0%	9.0%	0.5%	3.2%
	Tall T waves	Count	1	7	4	12
		% within Severity	0.8%	4.5%	2.2%	2.5%
Total		Count	129	156	186	471
		% within Severity	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	34.530[a]	6	.000
Likelihood Ratio	35.950	6	.000
N of Valid Cases	471		

99.2% of patients with mild head injury has normal T waves .

0.8% of patients with mild head injury has tall T waves .

97.3% of patients with moderate head injury had normal T waves .

0.5% of patients with moderate head injury has T wave inversion ..

2.2% of patients with moderate head injury had tall T waves .

85.3% of patients with severe head injury has normal T waves .

9.0% of patients with severe head injury has T wave inversion .

4.5% of patients with severe head injury has T wave inversion .

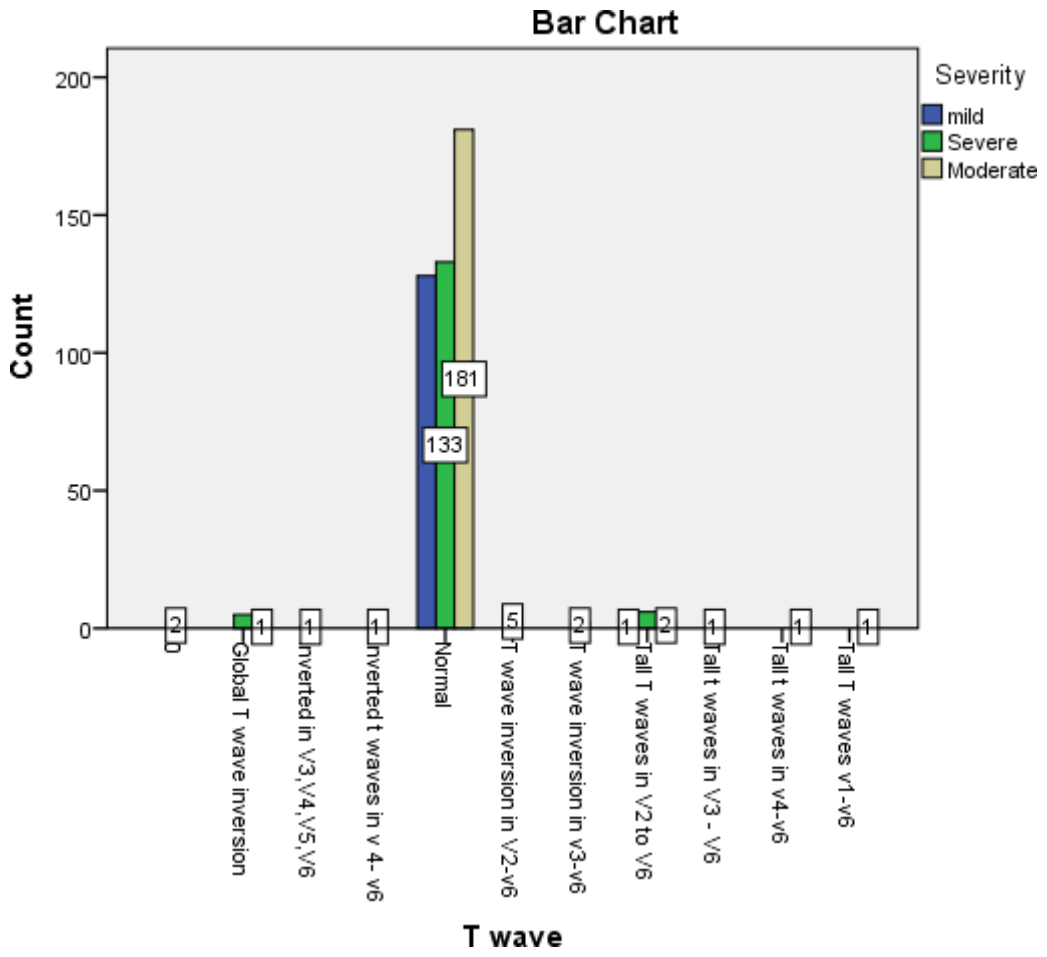


TABLE 9

CORRELATION BETWEEN U WAVE AND SEVERITY OF HEAD INJURY

Crosstab

			Severity			Total
			mild	Severe	Moderate	
	Normal	Count	129	151	182	462
		% within Severity	100.0%	96.8%	97.8%	98.1%
	Prominent	Count	0	3	4	7
		% within Severity	0.0%	1.9%	2.2%	1.5%
Total		Count	129	156	186	471
		% within Severity	100.0%	100.0%	100.0%	100.0%

100% of patients with mild head injury has normal U waves .

97.8% of patients with moderate head injury had normal U waves .

2.2% of patients with moderate head injury has prominent U waves .

96.8% of patients with severe head injury had normal U waves .

1.9% of patients with severe head injury had prominent U waves .

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	6.784[a]	4	.148
Likelihood Ratio	8.998	4	.061
N of Valid Cases	471		

TABLE 10

CORRELATION BETWEEN QTc INTERVAL AND SEVERITY OF HEAD INJUR.Y

Crosstab

			Severity			Total
			mild	Severe	Moderate	
QTc interval	Long QTC	Count	4	96	37	137
		% within Severity	3.1%	61.5%	19.9%	29.1%
	Normal	Count	123	57	147	327
		% within Severity	95.3%	36.5%	79.0%	69.4%
	Short QTC	Count	2	3	2	7
		% within Severity	1.6%	1.9%	1.1%	1.5%
Total		Count	129	156	186	471
		% within Severity	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	131.511[a]	4	.000
Likelihood Ratio	141.243	4	.000
N of Valid Cases	471		

95.3% of patients with mild head injury has normal QTc interval .

3.1% of patients with mild head injury has long QTc interval .

1.6% of patients with mild head injury has short QTc interval .

79.0% of patients with moderate head injury has normal QTc interval .

19.9% of patients with moderate head injury has long QTc interval .

1.1% of patients with moderate head injury has short QTc interval .

36.5% of patients with severe head injury has normal QTc interval .

61.5% of patients with severe head injury has long QTc interval .

1.9% of patients with severe head injury has short QTc interval .

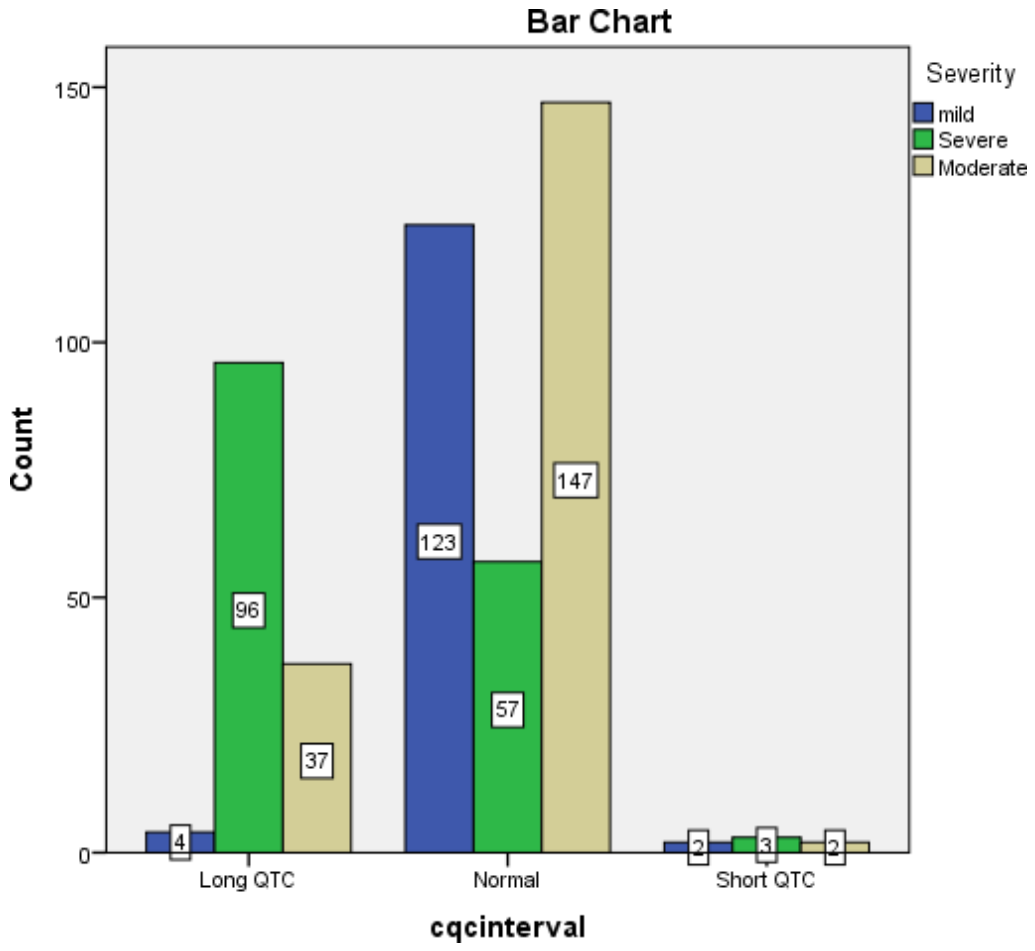


TABLE 11

CORRELATION BETWEEN SEVERITY OF HEAD INJURY AND GLASGOW OUTCOME SCORE.

			Severity			Total
			mild	Severe	Moderate	
Glasgow Outcome scale	1	Count	0	27	2	29
		% within Severity	0.0%	17.3%	1.1%	6.2%
	2	Count	0	40	2	42
		% within Severity	0.0%	25.6%	1.1%	8.9%
	3	Count	2	69	50	121
		% within Severity	1.6%	44.2%	26.9%	25.7%
	4	Count	1	19	104	124
		% within Severity	0.8%	12.2%	55.9%	26.3%
	5	Count	126	1	28	155
		% within Severity	97.7%	0.6%	15.1%	32.9%
Total		Count	129	156	186	471
		% within Severity	100.0%	100.0%	100.0%	100.0%

97.7% of mild head injury patients had a Glasgow Outcome Score of 5 .

1.6% of mild head injury patients had a Glasgow Outcome Score of 3 .

0.8% of mild head injury patients had a Glasgow Outcome Score of 4.

32.9% of moderate head injury patients had a Glasgow Outcome Score of 5 .

26.3% of moderate head injury patients had a Glasgow Outcome Score of 4 .

25.7% of moderate head injury patients had a Glasgow Outcome Score of 3.

8.9% of moderate head injury patients had a Glasgow Outcome Score of 2 .

6.2% of moderate head injury patients had a Glasgow Outcome Score of 1 .

44.2% of severe head injury patients had a Glasgow Outcome Score of 3 .

25.6% of severe head injury patients had a Glasgow Outcome Score of 2.

17.3% of severe head injury patients had a Glasgow Outcome Score of 1 .

12.2% of severe head injury patients had a Glasgow Outcome Score of 4 .

0.6% of severe head injury patients had a Glasgow Outcome Score of 5 .

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	508.243[a]	8	.000
Likelihood Ratio	535.952	8	.000
N of Valid Cases	471		

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 7.94.

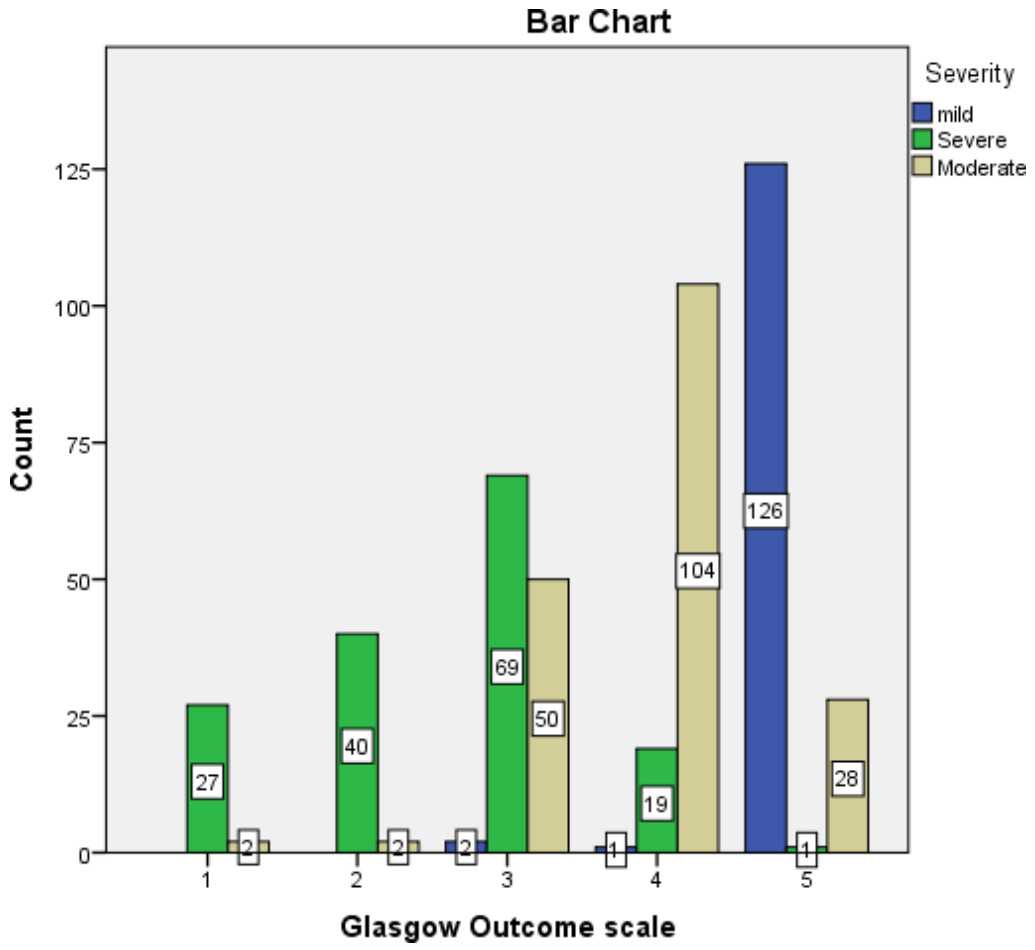


TABLE 12

CORRELATION BETWEEN RHYTHM AND SEVERITY OF HEAD INJURY.

Crosstab

			Glasgow Outcome scale			
			1	2	3	
Rhythm	Atrial fibrillation	Count	0	0	1	
		% within Glasgow Outcome scale	0.0%	0.0%	0.8%	
	Complete heart block	Count	1	0	0	
		% within Glasgow Outcome scale	3.4%	0.0%	0.0%	
	Junctional rhythm	Count	2	0	0	
		% within Glasgow Outcome scale	6.9%	0.0%	0.0%	
	Normal	Count	10	18	66	
		% within Glasgow Outcome scale	34.5%	42.9%	54.5%	
	Sinus bradycardia	Count	9	13	10	
		% within Glasgow Outcome scale	31.0%	31.0%	8.3%	
	Sinus tachycardia	Count	5	11	44	
		% within Glasgow Outcome scale	17.2%	26.2%	36.4%	
	Ventricular tachycardia	Count	2	0	0	
		% within Glasgow Outcome scale	6.9%	0.0%	0.0%	
Total		Count	29	42	121	
		% within Glasgow Outcome scale	100.0%	100.0%	100.0%	

Glasgow Outcome scale		Total
4	**5**	
1	0	2
0.8%	0.0%	0.4%

0	0	1
0.0%	0.0%	0.2%
0	0	2
0.0%	0.0%	0.4%
72	118	284
58.1%	76.1%	60.3%
10	2	44
8.1%	1.3%	9.3%
41	35	136
33.1%	22.6%	28.9%
0	0	2
0.0%	0.0%	0.4%
124	155	471
100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	144.008[a]	24	.000
Likelihood Ratio	89.715	24	.000
N of Valid Cases	471		

TABLE 13

CORRELATION BETWEEN P WAVES AND OUTCOME IN TBI

Crosstab

			Glasgow Outcome scale			
			1	2	3	4
P wave	Absent	Count	4	0	1	0
		% within Glasgow Outcome scale	13.8%	0.0%	0.8%	0.0%
	Fibrillatory p waves	Count	0	0	0	1
		% within Glasgow Outcome scale	0.0%	0.0%	0.0%	0.8%
	Normal	Count	25	42	120	123
		% within Glasgow Outcome scale	86.2%	100.0%	99.2%	99.2%
Total		Count	29	42	121	124
		% within Glasgow Outcome scale	100.0%	100.0%	100.0%	100.0%

Crosstab

			Glasgow Outcome scale	Total
			5	
P wave	Absent	Count	0	5
		% within Glasgow Outcome scale	0.0%	1.1%
	Fibrillatory p waves	Count	0	1
		% within Glasgow Outcome scale	0.0%	0.2%
	Normal	Count	155	465
		% within Glasgow Outcome scale	100.0%	98.7%
Total		Count	155	471
		% within Glasgow Outcome scale	100.0%	100.0%

TABLE 14

CORRELATION BETWEEN PR INTERVAL AND ECG CHANGES

Crosstab

			PR iNormalterval		
			0	Irregular	Normal
Glasgow Outcome scale	1	Count	3	1	24
		% within PR interval	60.0%	100.0%	5.3%
	2	Count	0	0	38
		% within PR interval	0.0%	0.0%	8.3%
	3	Count	1	0	117
		% within PR interval	20.0%	0.0%	25.7%
	4	Count	1	0	122
		% within PR interval	20.0%	0.0%	26.8%
	5	Count	0	0	155
		% within PR interval	0.0%	0.0%	34.0%
Total		Count	5	1	456
		% within PR interval	100.0%	100.0%	100.0%

Crosstab

			PR interval		Total
			prolonged pr interval	Short PR interval	
Glasgow Outcome scale	1	Count	0	1	29
		% within PR interval	0.0%	25.0%	6.2%
	2	Count	3	1	42
		% within PR interval	60.0%	25.0%	8.9%
	3	Count	1	2	121
		% within PR interval	20.0%	50.0%	25.7%
	4	Count	1	0	124
		% within PR interval	20.0%	0.0%	26.3%
	5	Count	0	0	155
		% within PR interval	0.0%	0.0%	32.9%
Total		Count	5	4	471
		% within PR interval	100.0%	100.0%	100.0%

TABLE 15

CORRELATION BETWEEN QRS COMPLEX AND OUTCOME IN TBI.

			Glasgow Outcome scale		
			1	2	3
QRS complex	Broad qrs complex	Count	2	0	0
		% within Glasgow Outcome scale	6.9%	0.0%	0.0%
	Left bundle branch block	Count	0	2	2
		% within Glasgow Outcome scale	0.0%	4.8%	1.7%
	Normal	Count	27	40	118
		% within Glasgow Outcome scale	93.1%	95.2%	97.5%
	Right bundle branch block	Count	0	0	1
		% within Glasgow Outcome scale	0.0%	0.0%	0.8%
Total		Count	29	42	121
		% within Glasgow Outcome scale	100.0%	100.0%	100.0%

Crosstab

			Glasgow Outcome scale		Total
			4	5	
QRS complex	Broad qrs complex	Count	0	0	2
		% within Glasgow Outcome scale	0.0%	0.0%	0.4%
	Left bundle branch block	Count	0	0	4
		% within Glasgow Outcome scale	0.0%	0.0%	0.8%
	Normal	Count	123	155	463
		% within Glasgow Outcome scale	99.2%	100.0%	98.3%
	Right bundle branch block	Count	1	0	2
		% within Glasgow Outcome scale	0.8%	0.0%	0.4%
Total		Count	124	155	471
		% within Glasgow Outcome scale	100.0%	100.0%	100.0%

TABLE 16
CORRELATION BETWEEN ST SEGMENT AND OUTCOME IN TBI

Crosstab

			st segment		
				0	Normal
Glasgow Outcome scale	1	Count	1	0	28
		% within st segment	3.8%	0.0%	6.4%
	2	Count	0	0	40
		% within st segment	0.0%	0.0%	9.1%
	3	Count	9	1	111
		% within st segment	34.6%	50.0%	25.2%
	4	Count	3	0	120
		% within st segment	11.5%	0.0%	27.3%
	5	Count	13	1	141
		% within st segment	50.0%	50.0%	32.0%
Total		Count	26	2	440
		% within st segment	100.0%	100.0%	100.0%

Crosstab

			st segment	Total
			st depression	
Glasgow Outcome scale	1	Count	0	29
		% within st segment	0.0%	6.2%
	2	Count	2	42
		% within st segment	66.7%	8.9%
	3	Count	0	121
		% within st segment	0.0%	25.7%
	4	Count	1	124
		% within st segment	33.3%	26.3%
	5	Count	0	155
		% within st segment	0.0%	32.9%
Total		Count	3	471
		% within st segment	100.0%	100.0%

TABLE 17

CORRELAOUTCOME IN TBI TION BETWEEN T WAVE CHANGES AND OUTCOME IN TBI

Crosstab

			T wave		
			0	Normal	t wave inversion
Glasgow Outcome scale	1	Count	2	20	4
		% within T wave	100.0%	4.5%	26.7%
	2	Count	0	34	6
		% within T wave	0.0%	7.7%	40.0%
	3	Count	0	113	5
		% within T wave	0.0%	25.6%	33.3%
	4	Count	0	121	0
		% within T wave	0.0%	27.4%	0.0%
	5	Count	0	154	0
		% within T wave	0.0%	34.8%	0.0%
Total		Count	2	442	15
		% within T wave	100.0%	100.0%	100.0%

Crosstab

			T wave	Total
			Tall T waves	
Glasgow Outcome scale	1	Count	3	29
		% within T wave	25.0%	6.2%
	2	Count	2	42
		% within T wave	16.7%	8.9%
	3	Count	3	121
		% within T wave	25.0%	25.7%
	4	Count	3	124
		% within T wave	25.0%	26.3%
	5	Count	1	155
		% within T wave	8.3%	32.9%
Total		Count	12	471
		% within T wave	100.0%	100.0%

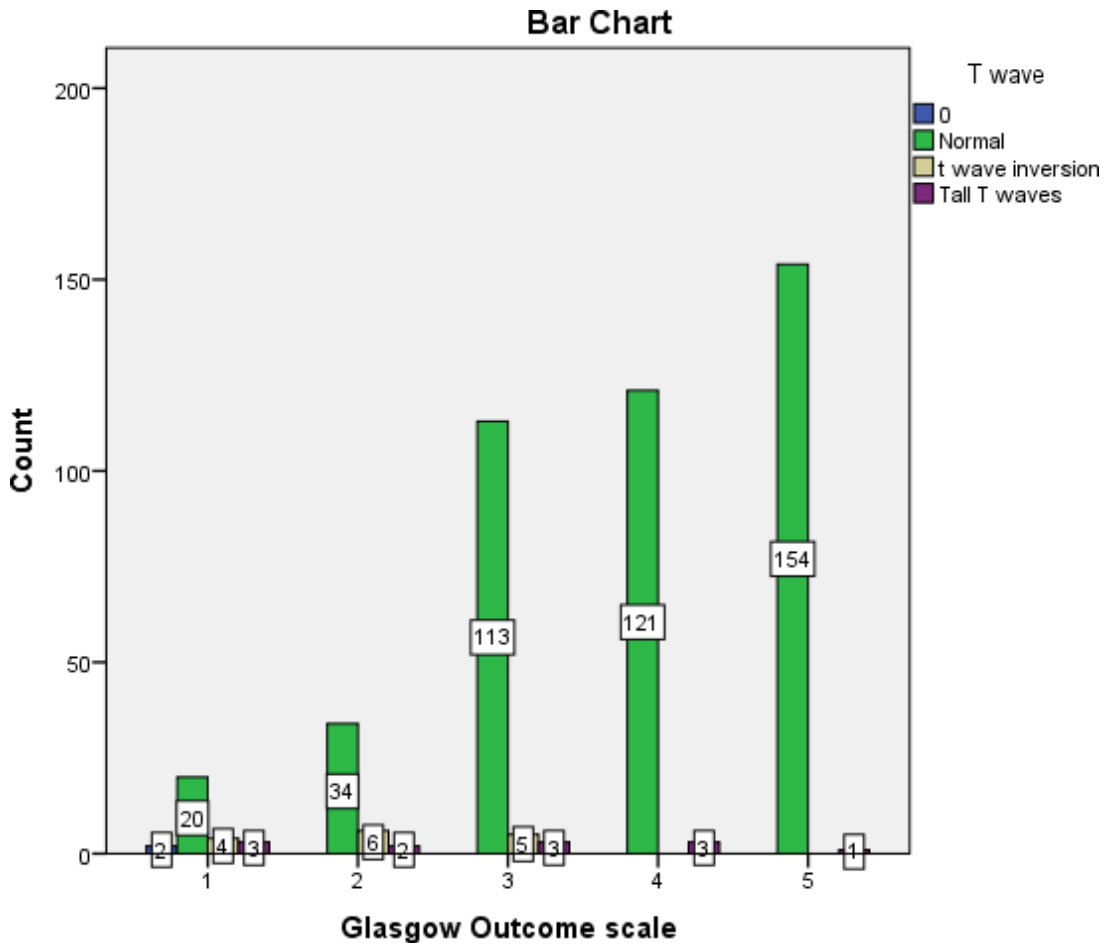


TABLE 18
CORRELATION BETWEEN U WAVES AND TBI

Crosstab

			Glasgow Outcome scale			
			1	2	3	4
U wave	0	Count	2	0	0	0
		% within Glasgow Outcome scale	6.9%	0.0%	0.0%	0.0%
	Normal	Count	26	41	118	122
		% within Glasgow Outcome scale	89.7%	97.6%	97.5%	98.4%
	Prominent	Count	1	1	3	2
		% within Glasgow Outcome scale	3.4%	2.4%	2.5%	1.6%
Total		Count	29	42	121	124
		% within Glasgow Outcome scale	100.0%	100.0%	100.0%	100.0%

Crosstab

			Glasgow Outcome scale	Total
			5	
U wave	0	Count	0	2
		% within Glasgow Outcome scale	0.0%	0.4%
	Normal	Count	155	462
		% within Glasgow Outcome scale	100.0%	98.1%
	Prominent	Count	0	7
		% within Glasgow Outcome scale	0.0%	1.5%
Total		Count	155	471
		% within Glasgow Outcome scale	100.0%	100.0%

TABLE 19\

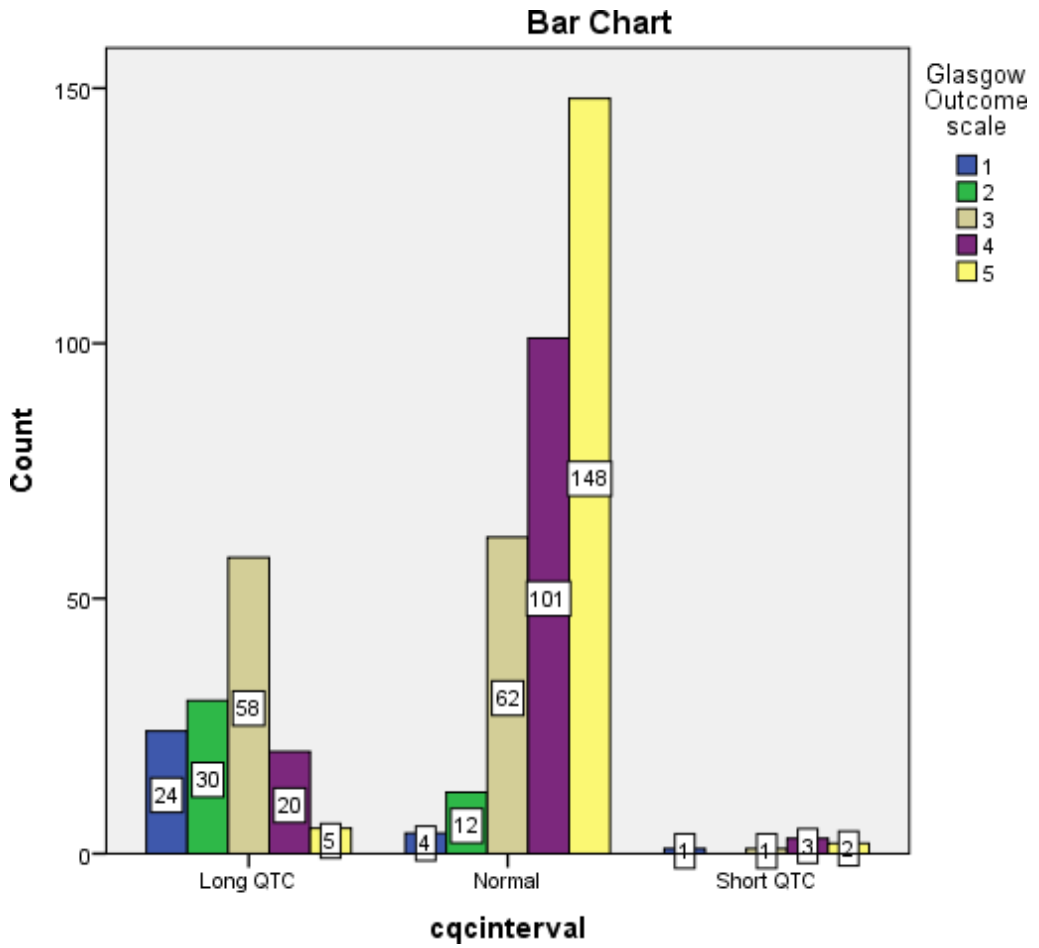
CORRELATION BETWEEN QTc interval and outcome in TBI.

Crosstab

			Glasgow Outcome scale			
			1	2	3	4
QTc interval	Long QTC	Count	24	30	58	20
		% within Glasgow Outcome scale	82.8%	71.4%	47.9%	16.1%
	Normal	Count	4	12	62	101
		% within Glasgow Outcome scale	13.8%	28.6%	51.2%	81.5%
	Short QTC	Count	1	0	1	3
		% within Glasgow Outcome scale	3.4%	0.0%	0.8%	2.4%
Total		Count	29	42	121	124
		% within Glasgow Outcome scale	100.0%	100.0%	100.0%	100.0%

Crosstab

			Glasgow Outcome scale	Total
			5	
QTc interval	Long QTC	Count	5	137
		% within Glasgow Outcome scale	3.2%	29.1%
	Normal	Count	148	327
		% within Glasgow Outcome scale	95.5%	69.4%
	Short QTC	Count	2	7
		% within Glasgow Outcome scale	1.3%	1.5%
Total		Count	155	471
		% within Glasgow Outcome scale	100.0%	100.0%



DISCUSSION

The incidence of road traffic accident and resultant traumatic brain injury has increa globally and especially in India. Most of the patients who suffer traumatic brain injury remain bed ridden and dependent on others for life or are dead, this increases the socioeconomic burden of the country.Outcome of patients with neurological injury is affected by many factors including age, associated premorbid conditions , operative interventions, however none of these factor can independently predict the outcome of a patient with traumatic brain injury. The electrocardiographic changes observed in patients with traumatic head injury has a major role in predicting the outcome such patients . Our study aimed to understand the association between the ECG changes and the severity of head injury, and correlate ECG changes with the one month outcome.

This present prospective study was conducted in Shri B.M Patil Medical college hospital and research centre, Vijayapur over a period of 2 years. The study was conducted on total of 471 patients after considering the inclusion and exclusion criteria. Initial electrocardiography was done for all patients and diagnostic imaging was done of the brain was done (CT or MRI). Patients were divided into three categories that is mild , moderate and severe head injury based on the Glasgow coma scale. Patients were then followed up after a month of the traumatic brain injury and the outcome was recorded as 1 : dead, 2: Persistent vegetative state, 3: severe disability , 4 : moderate disability, 5: good recovery.

In the present study it was noted that of the 471 patients, 184 were females and 287 were males. The mean age of the study population was 42.49 years Minimum age was 18 years , maximum was 86 years. Of these 28.03% had contusion, 24.42% had subarachnoid haemorrhage, 14.65% had extradural haemorrhage, 12.53% had subdural haemorrhage, 7.64% had intracerebral bleed, 5.30% had diffuse cerebral oedema, 2.92% had diffuse axonal injury, 2.92% had intraventricular bleed, 0.6% had

intracerebral bleed with SAH. In the study population, 39.9% had moderate head injury, 33.12% had severe head injury, 27.39% had mild head injury.

Among the 471 patients with TBI, minimum heart rate noted was 40 beats per minute, 200 was the maximum heart rate. Mean heart rate noted in the study population was 88 beats per minute. 60.3% had normal sinus rhythm, 28.87% had sinus tachycardia, 9.34% had sinus bradycardia, 0.425% had ventricular tachycardia, 0.425% had junctional rhythm, 0.2% had complete heart block.

In the present study group, 98.73% had normal p waves , 1% had absent p waves, 0.2% had fibrillatory p waves. In the study group, 96.82% had normal PR interval, 1% had prolonged PR interval, 0.84% had short PR interval, 0.2% irregular PR interval, 1% had absent PR interval. 98.3% of the study population had normal qrs complex, 0.84% had left bundle branch block, 0.42% had right bundle branch block.

Normal ST segment was noted in 93.4% of the patients, 0.21% had ST elevation in V4 -V6, 0.2% had elevation in lead 2 and 3 0.21% had St depression in V5 and V6, 0.8% had St depression in V4-V6, 0.8% had depression in V2-V6, 0.2% had depression in V1-V6, 0.42% had st depression in inferior leads,1.9% had global ST depression. 93.8% of the study population had normal T waves,1.2% had global T wave inversion, 2.5% had tall T waves, 1.8% had T wave inversion in various precordial leads.

Amongst the 471 patients with traumatic brain injury, 1.4% had prominent U waves. Repolarisation abnormalities was found in significant number of patients, 69.4% had normal QTc interval,29% had long QTc, 1.4% had short QTc .

Outcome was assessed based on Glasgow Outcome scale. 32.9% of patients had good recovery, 26.3% of patients had moderate disability , 25.6% of patients had severe disability, 8.9% had persistent vegetative state, 6.157% were dead at the end of one month of traumatic brain injury.

The severity of head injury was correlated with the ECG changes of the patients. Rhythm was found to be significantly associated with the severity of head injury with Chi- square value of 68.88 and p value of 0.00. Sinus tachycardia was found in 26% of patients with severe head injury, 33% of moderate head injury patients, 24% of mild head injury patients. Sinus bradycardia was found in 22.4% of severe head injury patients, 4.3% of moderate head injury patients, 0.8% of mild head injury patients. 2 patients with severe head injury had ventricular tachycardia.

There was no significant correlation between p wave and severity of head injury. PR interval was prolonged in 3 patients with severe head injury and 2 moderate head injury patients, p value noted was 0.22 and there was no significant correlation between severity and PR interval.With a p value of 0.22, correlation between Qrs complex and severity of head injury remained insignificant. ST segment was depressed in 1.3% of patients with severe head injury , 0.5% of patients with moderate head injury. P value was 0.542 and hence no significant correlation was noted between qrs complex and severity of head injury.

There was significant positive correlation between T wave changes and the severity of head injury. Chi square value 34.53 and P value was 0.00. T wave inversion was present in 3.2% of population, 9% in severe head injury patients, 0.5% in moderate head injury patients. Tall T waves were present in 2.5% of study group, 4.5% in severe head injury patients, 2.2% in moderate head injury patients.

U waves was prominent in 1.5% of study population, however with p value of 0.148 no significant correlation was noted between severity of head injury and U waves .

Of the 471 patients who were followed up after 1 month, significant correlation was noted between severity of head injury and the outcome. P value was 0.00. Chi square value was 508.2

There was significant positive correlation between rate and outcome, Chi square value was 144.0, P value of 0.00. Of the 29 patients who were dead by first month of injury, 31% had sinus bradycardia , 17% had sinus tachycardia , 6.9% had ventricular tachycardia. Of the 42 patients who were in persistent vegetative state, 31% had sinus bradycardia, 26% had sinus tachycardia. Of the 121 people who had severe disability, 8.3% had sinus bradycardia ,36.4% had sinus tachycardia. Of the 124 patients with moderate disability, 33% had sinus tachycardia, 8% had sinus bradycardia. Of the 155 patients with good recovery, 22% had sinus tachycardia, 1.3% had sinus bradycardia.

P waves absent in 1.1% of total study population. Chi square value 51.04 . 13.8% of patients who died at the end of one month, had absent p waves , 0.8% of patients with severe disability had absent p waves, 0.8% of patients with moderate disability had fibrillatory p waves.

Qrs complex was found to be associated with the outcome of the patients, Chi square value of 43.61 p value of 0.00. 6.9% of patients who were dead at end of one month broad complex qrs, 4.8% of patients with persistent vegetative state had left bundle branch block. Of the 121 patients with persistent vegetative state, 1.7% had left bundle branch block, 0.8% had right bundle branch block.0.8% of patients with moderate disability had right bundle branch block.

No significant correlation was present between ST segment changes and the outcome. Chi square value was 22.8, p value 0.029.

Significant positive correlation was present between QTc interval and Glasgow outcome scale, chi square value of 161.2 and p value of 0.00. 29.1% of study group had long QTc interval, 1.5% had short QTc interval, 69.4% had normal QTc interval.

There was significant positive correlation between T wave changes and the outcome. Chi square value of 79.495, p value of 0.00 . Most common T wave change noted

was that wave inversion, it was present in 26% of patients with GOS score 1, 40% patients with persistent vegetative state, 33.3% of patients with severe disability. Tall T waves were noted in 25% of patients with GOS 1, 16.7% of patients with persistent vegetative state, 25% of patients with severe disability, 25% of patients with moderate disability , 1 % with good recovery.

CONCLUSION

Electrocardiographic changes in patients significantly associated with the outcome in patients with traumatic brain injury. The most common ECG changes that are found are sinus tachycardia, sinus bradycardia, T wave changes, and prolongation of QT interval. Repolarization abnormalities such as prolongation of the QT interval and changes in the ST segment and T wave morphology are the most common ECG found in the study group . There was no specific changes noted in ECG for different types of traumatic brain injury.

Patients with good recovery at the end of month (Glasgow Outcome scale of 5) had least changes in the electrocardiograph. ECG changes are significant in predicting the outcome of patients with traumatic brain injury.

Electrocardiograph thus appears to have significant role determining the mortality of patients with traumatic brain injury .

BIBLIOGRAPHY

1. Krishnamoorthy V, Prathep S, Sharma D, Gibbons E, Vavilala MS. Association between electrocardiographic findings and cardiac dysfunction in adult isolated traumatic brain injury. Indian journal of critical care medicine: peer-reviewed, official publication of Indian Society of Critical Care Medicine. 2014 Sep;18(9):570.
2. Fan X, DU FH, Tian JP. The electrocardiographic changes in acute brain injury patients. Chinese Medical Journal. 2012 Oct 5;125(19):3430-3.
3. Padilla-Zambrano HS, Garcia-Ballestas E, Rojas AN, Moscote-Salazar LR, Ghosh A, Pal R, Agrawal A. "Broken heart syndrome" Cardiovascular manifestations of traumatic brain injury. Heart and Mind. 2018 Jan 1;2(1):12.
4. Astarabadi M, Khurrum M, Asmar S, Bible L, Chehab M, Castanon L, Ditillo M, Douglas M, Joseph B. The impact of non-neurological organ dysfunction on outcomes in severe isolated traumatic brain injury. Journal of Trauma and Acute Care Surgery. 2020 Aug 1;89(2):405-10.
5. Collier BR, Miller SL, Kramer GS, Balon JA, Gonzalez III LS. Traumatic subarachnoid hemorrhage and QTc prolongation. Journal of Neurosurgical Anesthesiology. 2004 Jul 1;16(3):196-200.
6. Samudrala VD, Kumar A, Agrawal A. Electrocardiographic changes in patients with isolated traumatic brain injury and their correlation with outcome. Indian Journal of Neurotrauma. 2016 Aug;13(02):070-4.
7. Bramwell C. Can a head injury cause auricular fibrillation?. The Journal of Nervous and Mental Disease. 1934 Dec 1;80(6):716.

8. Singla SL, Garg P, Mehta RK. Electrocardiographic changes in craniocerebral trauma--could they serve as prognostic indicators?. Journal of the Indian Medical Association. 2002 Mar 1;100(3):188-90.
9. Coghlan LA, Hindman BJ, Bayman EO, Banki NM, Gelb AW, Todd MM, Zaroff JG, IHAST Investigators. Independent associations between electrocardiographic abnormalities and outcomes in patients with aneurysmal subarachnoid hemorrhage: findings from the intraoperative hypothermia aneurysm surgery trial. Stroke. 2009 Feb 1;40(2):412-8.
10. Wybraniec MT, Mizia-Stec K, Krzych Ł. Neurocardiogenic injury in subarachnoid hemorrhage: A wide spectrum of catecholamin-mediated brain-heart interactions. Cardiology Journal. 2014;21(3):220-8.
11. Syverud GE. Electrocardiographic changes and intracranial pathology. AANA journal. 1991 Jun 1;59(3):229-32.
12. Frontera JA, Parra A, Shimbo D, Fernandez A, Schmidt JM, Peter P, Claassen J, Wartenberg KE, Rincon F, Badjatia N, Naidech A. Cardiac arrhythmias after subarachnoid hemorrhage: risk factors and impact on outcome. Cerebrovascular diseases. 2008;26(1):71-8.
13. Rudehill A, Olsson GL, Sundqvist K, Gordon E. ECG abnormalities in patients with subarachnoid haemorrhage and intracranial tumours. Journal of Neurology, Neurosurgery & Psychiatry. 1987 Oct 1;50(10):1375-81.
14. Lacy PS, Earle AM. Central neural control of blood pressure and cardiac arrhythmias during subarachnoid hemorrhage in rats. Stroke. 1985 Nov;16(6):998-102.
15. HERSCH C. Electrocardiographic changes in head injuries. Circulation. 1961 Jun;23(6):853-60.

16. Chen Z, Venkat P, Seyfried D, Chopp M, Yan T, Chen J. Brain–heart interaction: cardiac complications after stroke. Circulation research. 2017 Aug 4;121(4):451-68.
17. Hashemian AM, Ahmadi K, Taherinia A, Sharifi MD, Ramezani J, Jazayeri SB, Saadat S, Rahimi-Movaghar V. ECG changes of cardiac origin in elderly patients with traumatic brain injury. Medical journal of the Islamic Republic of Iran. 2015;29:306.
18. Surawicz B. Electrolytes, hormones, temperature, and miscellaneous factors: electrophysiologic basis of ECG and cardiac arrhythmias. Baltimore, MD: Williams and Wilkins. 1995:426-53.
19. van der Ende MY, Siland JE, Snieder H, van der Harst P, Rienstra M. Population- based values and abnormalities of the electrocardiogram in the general Dutch population: the lifelines cohort study. Clinical cardiology. 2017 Oct;40(10):865-72.
20. van Bree MD, Roos YB, van der Bilt IA, Wilde AA, Sprengers ME, de Gans K, Vergouwen MD. Prevalence and characterization of ECG abnormalities after intracerebral hemorrhage. Neurocritical care. 2010 Feb;12(1):50-5.
21. Qureshi AI, Tuhrim S, Broderick JP, Batjer HH, Hondo H, Hanley DF. Spontaneous intracerebral hemorrhage. New England Journal of Medicine. 2001 May 10;344(19):1450-60.
22. Oppenheimer S. Cerebrogenic cardiac arrhythmias:: Cortical lateralization and clinical significance. Clinical Autonomic Research. 2006 Feb;16(1):6.
23. Lenstra JJ, Kuznecova-Keppel Hesselink L, la Bastide-van Gemert S, et al. The Association of Early Electrocardiographic Abnormalities With Brain Injury Severity and Outcome in Severe Traumatic Brain Injury. Frontiers in Neurology. 2020 ;11:597737. DOI: 10.3389/fneur.2020.597737. PMID: 33488498; PMCID: PMC7819976.

24. Modi NJ, Agrawal M, Sinha VD. Post-traumatic subarachnoid hemorrhage: A review. Neurology India. 2016 Feb 1;64(7):8.
25. Ullman JS, Morgan BC, Eisenberg HM. Traumatic subarachnoid hemorrhage. Subarachnoid hemorrhage: pathophysiology and management. Park Ridge: American Association of Neurological Surgeons. 1997.
26. Mattioli C, Beretta L, Gerevini S, Veglia F, Citerio G, Cormio M, Stocchetti N. Traumatic subarachnoid hemorrhage on the computerized tomography scan obtained at admission: a multicenter assessment of the accuracy of diagnosis and the potential impact on patient outcome. Journal of neurosurgery. 2003 Jan 1;98(1):37-42.
27. Chatterjee S. ECG changes in subarachnoid haemorrhage: a synopsis. Netherlands Heart Journal. 2011 Jan;19(1):31-4.
28. Yokobori S, Nakae R, Yokota H, Spurlock MS, Mondello S, Gajavelli S, Bullock RM. Subdural hematoma decompression model: a model of traumatic brain injury with ischemic-reperfusional pathophysiology: a review of the literature. Behavioural brain research. 2018 Mar 15;340:23-8.
29. Ndoumbe A, Ekeme MV, Jemea B, Simeu C, Takongmo S. Epidemiological analysis of surgically treated acute traumatic epidural hematoma. Open Journal of Modern Neurosurgery. 2016 Jun 30;6(3):89-97.
30. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. The Lancet. 1974 Jul 13;304(7872):81-4.
31. Jain S, Iverson LM. Glasgow coma scale.
32. Gennarelli TA, Champion HR, Copes WS, Sacco WJ. Comparison of mortality, morbidity, and severity of 59,713 head injured patients with 114,447 patients with extracranial injuries. The Journal of trauma. 1994 Dec 1;37(6):962-8.
33. Samuels MA. The brain–heart connection. Circulation. 2007 Jul 3;116(1):77-84.

34. Lowensohn R, Weiss M, Hon E. Heart-rate variability in brain-damaged adults. The Lancet. 1977 Mar 19;309(8012):626-8.

35. Berman M, Ali A, Ashley E, Freed D, Clarke K, Tsui S, Parameshwar J, Large S. Is stress cardiomyopathy the underlying cause of ventricular dysfunction associated with brain death?. The Journal of Heart and Lung Transplantation. 2010 Sep 1;29(9):957-65.

36. Sommargren CE, Warner R, Zaroff JG, Banki NM. Electrocardiographic abnormalities in patients with subarachnoid hemorrhage and normal adults: a comparison study. Journal of Electrocardiology. 2004;37:42.

37. Cropp GJ, Manning GW. Electrocardiographic changes simulating myocardial ischemia and infarction associated with spontaneous intracranial hemorrhage. Circulation. 1960 Jul;22(1):25-38.

38. Kono T, Morita H, Kuroiwa T, Onaka H, Takatsuka H, Fujiwara A. Left ventricular wall motion abnormalities in patients with subarachnoid hemorrhage: neurogenic stunned myocardium. Journal of the American College of Cardiology. 1994 Sep 1;24(3):636-40.

39. Lundberg N. The saga of the Monro-Kellie doctrine. InIntracranial pressure V 1983 (pp. 68-76). Springer, Berlin, Heidelberg.

40. Rodríguez-Boto G, Rivero-Garvía M, Gutiérrez-González R, Márquez-Rivas J. Basic concepts about brain pathophysiology and intracranial pressure monitoring. Neurología (English Edition). 2015 Jan 1;30(1):16-22.

41. Jachuck SJ, Ramani PS, Clark F, Kalbag RM. Electrocardiographic abnormalities associated with raised intracranial pressure. Br Med J. 1975 Feb 1;1(5952):242-4.

42. Cushing H. Concerning a definite regulatory mechanism of the vasomotor centers which controls blood pressure during cerebral compression. Bull Johns Hopkins Hosp. 1901;12:290.

43. Shanlin RJ, Sole MJ, Rahimifar M, Tator CH, Factor SM. Increased intracranial pressure elicils hypertension, increased sympathetic activity, electrocardiographic abnormalities

and myocardial damage in rats. Journal of the American College of Cardiology. 1988 Jan 1;12(3):727-36.

44. McMillan T, Wilson L, Ponsford J, Levin H, Teasdale G, Bond M. The Glasgow Outcome Scale—40 years of application and refinement. Nature Reviews Neurology. 2016 Aug;12(8):477-85.
45. Kenneth W, Lindsay B, Bone I, Lindsay KW. Neurology and neurosurgery illustrated. Elsevier Health Sciences; 2010.
46. Busl KM, Raju M, Ouyang B, Garg RK, Temes RE. Cardiac abnormalities in patients with acute subdural hemorrhage. Neurocritical care. 2013 Oct;19(2):176-82.
47. Sommargren CE. Electrocardiographic abnormalities in patients with subarachnoid hemorrhage. American Journal of Critical Care. 2002 Jan;11(1):48-56.
48. Yang XL, Liu GZ, Tong YH, Yan H, Xu Z, Chen Q, Liu X, Zhang HH, Wang HB, Tan SH. The history, hotspots, and trends of electrocardiogram. Journal of geriatric cardiology: JGC. 2015 Jul;12(4):448.
49. Becker DE. Fundamentals of electrocardiography interpretation. Anesthesia progress. 2006;53(2):53-64.
50. Spodick DH, Frisella M, Apiyassawat S. QRS axis validation in clinical electrocardiography. The American journal of cardiology. 2008 Jan 15;101(2):268-9.
51. Batra MK, Khan A, Farooq F, Masood T, Karim M. Assessment of electrocardiographic criteria of left atrial enlargement. Asian Cardiovascular and Thoracic Annals. 2018 May;26(4):273-6.
52. Pipberger HV, Tanenbaum HL. The P wave, PR interval, and QT ratio of the normal orthogonal electrocardiogram. Circulation. 1958 Dec;18(6):1175-80.
53. Smiseth OA, Aalen JM. Mechanism of harm from left bundle branch block. Trends in cardiovascular medicine. 2019 Aug 1;29(6):335-42.

54. Alnsasra H, Ben-Avraham B, Gottlieb S, Ben-Avraham M, Kronowski R, Iakobishvili Z, Goldenberg I, Strasberg B, Haim M. High-grade atrioventricular block in patients with acute myocardial infarction. Insights from a contemporary multi-center survey. Journal of Electrocardiology. 2018 May 1;51(3):386-91.
55. Zema MJ, Kligfield P. ECG poor R-wave progression: review and synthesis. Archives of internal medicine. 1982 Jun 1;142(6):1145-8.
56. de Bliek EC. ST elevation: Differential diagnosis and caveats. A comprehensive review to help distinguish ST elevation myocardial infarction from nonischemic etiologies of ST elevation. Turkish journal of emergency medicine. 2018 Mar 1;18(1):1-0.
57. Sattar Y, Chhabra L. Electrocardiogram. InStatPearls [Internet] 2022 Jun 13. StatPearls Publishing.
58. Rudic B, Schimpf R, Borggrefe M. Short QT syndrome–review of diagnosis and treatment. Arrhythmia & electrophysiology review. 2014 Aug;3(2):76.

ANNEXURE 1

IEC/No-09/2021
Date-22/01/2021

B.L.D.E. (DEEMED TO BE UNIVERSITY)

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11-00 am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: A study on electrocardiographic changes in head injury and their correlation with the outcome in patients with traumatic brain injury

Name of PG student: DrPriyanka G R, Department of Emergency Medicine

Name of Guide/Co-investigator: Dr Babu Kattimani, Associate Professor of Emergency Medicine

DR .S.V.PATIL
CHAIRMAN, IEC

Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B. M. Patil Medical College,
VIJAYAPURA-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

ANNEXURE 2

INFORMED CONSENT

BLDEU'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR-586103

TITLE OF THE PROJECT - STUDY ON ELECTROCARDIOGRAPHIC CHANGES IN HEAD INJURY AND THEIR CORRELATION WITH THE OVERALL OUTCOME IN PATIENTS WITH TRAUMATIC BRAIN INJURY

PRINCIPAL INVESTIGATOR P.G.GUIDE NAME

-

Dr.BABU KATTIMANI

I have been informed about this study. I have also been given a free choice of participation in this study.

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedure of this study is not expected to exaggerate these feelings that are associated with the usual course of treatment.

outcome.

records and will be subject to the confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by code number. The code-key connecting name to numbers will be kept in a separate location.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photographs and videotapes and hear the audiotapes before giving this permission.

Dr.PRIYANKA .G.R.is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation.

If during the study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful reading.

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr.PRIYANKA.G.R. may terminate my participation in the study after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

procedures required and the possible risks and benefits to the best of my ability in patient's own language.

Dr. PRIYANKA .G.R.

Date

I confirm that Dr. PRIYANKA G.R has explained to me the purpose of research, the study procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read and I understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

_____

Participant / Guardian

_____

Witness to signature

ANNEXURE 2

PERFORMA

STUDY ON ELECTROCARDIOGRAPHIC CHANGES IN HEAD INJURY AND THEIR CORRELATION WITH THE OVERALL OUTCOME IN PATIENTS WITH TRAUMATIC BRAIN INJURY

Religion:

Residence:

DOD:

PHONE NUMBER

The Glasgow Coma Scale is a neurological scale which provides a reliable objective way of recording the conscious state of a person. It's comprises of 3 tests – Eye,Verbal,Motor

GCS >/=13. Minor

9- 12 = moderate

The outcome of the patient after 1 month of insult is assessed based on Glasgow outcome score

Glasgow outcome score

1- DEAD

2- PERSISTENT VEGETATIVE STATE

3- SEVERE DISABILITY

4- MODERATE DISABILITY

5- GOOD RECOVERY

This 1 month outcome of the patient is compared with the initial ECG changes.

Patient showing ST changes are subjected to 2D echocardiography and cardiac enzyme analysis to rule out Myocardial Infarction

MASTERCHART

Patient name	Age	Sex	Diagnosis	Type of TBI	Glasgow coma scale		Rate	Rhythm	P w
Suresh	44	Male	Right fronto-parietal SDH	SDH	11/15	Moderate	106	Sinus tachycardia	Nor
Sangappa	65	Male	Right occipital SAH	SAH	10/15	Moderate	120	Sinus tachycardia	Nor
Yellawwa	44	Female	Intracerebral bleed	IC BLeed	8/15	Severe	56	Sinus bradycardia	Nor
Sathish	82	Male	Right parietal SAH	SAH	11/15	moderate	111	Sinus tachycardia	Nor
Mahadev	45	Male	Diffuse axonal injury	Diffuse axonal injury	5/15	Severe	60	Normal	Nor
Rajamma	65	Female	Right parietal SAH with SDH	SAH and SDH	7/15	Severe	98	Normal	Nor
Somanna	44	Male	Multiple left parieto occipital contusion	contusion	10/15	moderate	112	Sinus tachycardia	Nor
Balla	23	Male	Left occipital SDH	SDH	12/15	moderate	108	Normal	Nor
Veena	19	Female	Diffuse cerebral edema	Diffuse cerebral edema	13/15	mild	98	Normal	Nor
Raghavendra	45	Male	Right frontal intracerebral bleed	IC bleed	5/15	Severe	50	Sinus bradycardia	Nor
Amaresh	36	Male	Left fronto-parietal SAH	SAH	10/15	moderate	120	Sinus tachycardia	Nor
Tippanna	80	Male	Intracerebral bleed with SAH	IC bleed with SAH	6/15	severe	113	Sinus tachycardia	Nor
Vinod	21	Male	Left Parietal EDH	EDH	9/15	moderate	110	Sinus tachycardia	Nor
Ramya	19	Female	Left parietal contusion	Contusion	13/15	mild	80	Normal	Nor
Mahesh	39	Female	Intraventricular bleed with midline shift	Intraventricular bleed	3/15	severe	68	Normal	Nor
Somappa	54	Male	Right parietal EDH	EDH	12/15	moderate	105	Sinus tachycardia	Nor
Sangameshwari	62	Female	Diffuse cerebral edema	Diffuse cerebral edema	14/15	mild	90	Normal	Nor
Ningappa	71	Male	Right parietal SAH	SAH	13/15	mild	60	Normal	Nor

Tiramisu	65	Female	Right fronto-parietal SAH with SDH	SAH and SDH	10/15	moderate	78	Normal	Nor
Yenkanna	35	Male	Right parietal SAH	SAH	8/15	severe	80	Normal	Nor
Satish	44	Male	Right frontal SDH	SDH	8/15	severe	110	Sinus tachycardia	Nor
Manohari	52	Female	Left frontal contusion	Contusion	11/15	moderate	88	Normal	Nor
Manish	19	Male	Multiple right parietal contusion	Contusion	10/15	moderate	94	Normal	Nor
Vishalakshi	20	Female	Left frontal contusion with SAH	SAH	9/15	moderate	58	Sinus bradycardia	Nor
Paranappa	35	Male	Diffuse axonal injury	Diffuse axonal injury	5/15	sevre	115	Sinus tachycardia	Nor
Shivanand	46	Male	Minimal right frontal contusion	Contusion	14/15	mild	90	Normal	Nor
Na Jawa	36	Female	Left parietal SAH	SAH	12/15	moderate	70	Normal	Nor
Nazar alla	62	Male	Multiple right parieto occipital contusion	contusion	11/15	moderate	90	Normal	Nor
Babulal	41	Male	Right frontal bleed	IC bleed	8/15	severe	58	Sinus bradycardia	Nor
Vikramappa	53	Female	Left frontal contusion	Contusion	12/15	moderate	86	Normal	Nor
Bhimsankar	32	Male	Right occipital contusion	Contusion	11/15	moderate	88	Normal	Nor
Vikramappa	62	Male	Intracerebral bleed with SAH	SAH	7/15	severe	92	Normal	Nor
Shivanand	45	Male	Right occipital contusion	Contusion	12/15	moderate	80	Normal	Nor
Tara Singh	51	Female	Left parietal SAH	SAH	10/15	moderate	110	Sinus tachycardia	Nor
Mallappa	41	Male	Right frontal intracerebral bleed	IC bleed	5/15	severe	59	Sinus bradycardia	Nor
Mallannna	24	Male	Large left hemisphere intracerebral bleed	IC bleed	4/15	severe	45	Sinus bradycardia	Nor
Sumitra	25	Female	Right frontal SDH	SDH	7/15	severe	70	Normal	Nor
Ning Anna	35	Male	Diffuse cerebral edema	Diffuse cerebral edema	13/15	mild	89	Normal	Nor
Venkatesh	46	Male	Left Parietal EDH	EDH	11/15	moderate	94	Normal	Nor
Janakibai	41	Female	Right frontal intracerebral bleed	IC bleed	8/15	moderate	56	Sinus bradycardia	Nor
Yamuna	35	Female	Right fronto-parietal SDH with SAH	SDH and SAH	7/15	severe	110	Sinus tachycardia	Nor
Tarabai	53	Female	Left frontal contusion	Contusion	13/15	mild	78	Normal	Nor
Komalppa	51	Male	Right temporal SDH	SDH	11/15	moderate	90	Normal	Nor
Chaitra	29	Female	Bilateral frontal contusion	Contusion	11/15	moderate	98	Normal	Nor
Kumar	28	Male	Right temporo-parietal SAH	SAH	10/15	moderate	102	Sinus tachycardia	Nor
Rajmalla	38	Male	Multiple left parietal contusion	SAH	8/15	severe	90	Normal	Nor
Sonubai	48	Female	Diffuse axonal injury	Diffuse axonal injury	5/15	severe	59	Sinus bradycardia	Nor
Rajkumar	65	Male	Right fronto-parietal SAH	SAH	12/15	moderate	120	Sinus tachycardia	Nor
Mahendra	32	Male	Left parietal Bleed with depressed skull fracture		6/15	severe	180	Sinus tachycardia	Nor
Kavya	19	Female	Left parietal contusion	Contusion	13/15	mild	70	Normal	Nor
Malaya	38	Male	Right fronto-parietal SDH with SAH	SDH and SAH	7/15	severe	140	Sinus tachycardia	Nor
Ninganna Gouda	54	Male	Left parietal SAH	SAH	11/15	moderate	120	Sinus tachycardia	Nor
Eshwaran	58	Female	Left frontal contusion	Contusion	13/15	mild	78	Normal	Nor
Mallanna	62	Female	Right occipital contusion	Contusion	13/15	mild	90	Normal	Nor
Mallappa	42	Male	Right fronto-parietal SAH	SAH	10/15	moderate	68	Normal	Nor
Vishalakshi	46	Female	Left parietal EDH	EDH	12/15	moderate	72	Normal	Nor
Selena	38	Male	Intraventricular bleed with midline shift	Intraventricular bleed	7/15	severe	200	Ventricular tachycardia	Abs
Maruti	51	Male	Bilateral frontal SAH	SAH	11/15	moderate	88	Normal	Nor
Chinaware	57	Female	Right parietal EDH	EDH	12/15	moderate	98	Normal	Nor
Paramappa	67	Male	Left occipital contusion	Contusion	13/15	mild	100	Sinus tachycardia	Nor
Gollawwa	27	Female	Multiple left temporal contusion with SAH	SAH	8/15	severe	62	Normal	Nor
Mitanni	51	Male	Right parietal bleed with SAH	IC bleed and SAH	8/15	severe	102	Sinus tachycardia	Nor
Sreedharan	26	Male	Left fronto parietal contusion with SAH	SH	10/15	moderate	110	Sinus tachycardia	Nor
Kamala	34	Female	Right frontal contusion	Contusion	13/15	mild	99	Normal	Nor
Ravi	28	Male	Left parietal bleed with midline shift	IC bleed	8/15	severe	40	Sinus bradycardia	Nor

Meenakshi	19	Male	Multiple left temporal contusion	Contusion	13/15	mild	88	Normal	Nor
Tarabai	37	Female	Diffuse cerebral edema	Diffuse cerebral edema	14/15	mild	98	Normal	Nor
Tippanna	62	Male	Right frontal EDH	EDH	12/15	moderate	104	Sinus tachycardia	Nor
Ningappa	47	Male	Left frontal contusion with SAH	SH	7/15	severe	60	Normal	Nor
Amijaan	72	Female	Right occipital bleed	IC bleed	8/15	severe	103	Sinus tachycardia	Nor
Kallappa	34	Male	Left fronto-parietal EDH	EDH	9/15	moderate	89	Normal	Nor
Yenkawwa	67	Female	Right fronto-parietal SDH	SDH	11/15	moderate	106	Sinus tachycardia	Nor
Abdul	51	Male	Right temporal contusion	Contusion	12/15	moderate	99	Normal	Nor
Shreedevi	20	Male	Right frontal bleed with SAH	SAH	6/15	severe	56	Sinus bradycardia	Nor
Parashuram	36	Male	Left temporal contusion	Contusion	13/15	mild	110	Sinus tachycardia	Nor
Kalavati	24	Female	Diffuse axonal injury	Diffuse axonal injury	5/15	severe	80	Normal	Nor
Bhimannagouda	24	Male	Right fronto-parietal SAH	SAH	11/15	moderate	98	Normal	Nor
Giddanna	26	Male	Left parietal SDH	SDH	11/15	moderate	103	Sinus tachycardia	Nor
Neela	28	Female	Right frontal contusion	Contusion	13/15	mild	90	Normal	Nor
Mohith	25	Male	Right occipital bleed	IC bleed	8/15	severe	59	Sinus bradycardia	Nor
Rangalakshmi	35	Female	Left parietal bleed with midline shift	IC bleed	7/15	severe	48	Sinus bradycardia	Nor
Ninganna Gouda	60	Male	Diffuse cerebral edema	Diffuse cerebral edema	13/15	mild	98	Normal	Nor
Dastagiri	44	Male	Right fronto-parietal SAH	SAH	11/15	moderate	80	Normal	Nor
Soumya	33	Female	Left parietal contusion	Contusion	13/15	mild	103	Sinus tachycardia	Nor
Buduma	25	Male	Right fronto-parietal EDH	EDH	8/15	severe	120	Sinus tachycardia	Nor
Ramyashree	64	Female	Right fronto-parieto-temporal EDH	EDH	7/15	severe	101	Sinus tachycardia	Nor
Vimala	20	Female	Left occipital contusion	Contusion	13/15	mild	90	Normal	Nor
Taranath	31	Male	Right fronto-parietal SDH	SDH	11/15	moderate	92	Normal	Nor
Guruprasad	60	Male	Right frontal contusion with SAH	SAH	8/15	severe	112	Sinus tachycardia	Nor
Rama Rao	51	Male	Right temporal contusion	Contusion	13/15	mild	89	Normal	Nor
Prema	28	Female	Right frontal multiple contusion	Contusion	10/15	moderate	100	Sinus tachycardia	Nor
Yallappa	37	Male	Right frontal SAH	SAH	8/15	severe	126	Sinus tachycardia	Nor
Vindhyamala	66	Female	Left occipital SDH	SDH	7/15	severe	89	Normal	Nor
Nagamma	34	Female	Left temporal contusion	Contusion	11/15	moderate	104	Sinus tachycardia	Nor
Sattwawwa	58	Female	Right parietal bleed	IC bleed	6/15	severe	130	Sinus tachycardia	Nor
Iriyya	49	Male	Intraventricular bleed with midline shift	Intraventricular bleed	5/15	severe	54	Sinus bradycardia	Nor
Mallanna Gouda	60	Male	Diffuse cerebral edema	Diffuse cerebral edema	13/14	mild	80	Normal	Nor
Seetha	22	Female	Right frontal EDH	EDH	11/15	moderate	111	Sinus tachycardia	Nor
Basavaraj	27	Male	Left occipital SDH	SDH	10/15	moderate	107	Sinus tachycardia	Nor
Venkatesh	61	Male	Right temporal contusion	Contusion	11/15	moderate	120	Sinus tachycardia	Nor
Dadusab	55	Maley	Left frontal SAH with contusion	SAH	10/15	moderate	106	Sinus tachycardia	Nor
Vittal	43	Male	Left fronto parietal contusion with SAH	SAH	9/15	moderate	104	Sinus tachycardia	Nor
Parashuram	24	Male	Right occipital contusion	Contusion	13/15	mild	70	Normal	Nor
Gundappa	62	Male	Diffuse cerebral edema	Diffuse cerebral edema	14/15	mild	80	Normal	Nor
Yellawwa	48	Female	Intraventricular bleed with midline shift	IC bleed	6/15	severe	56	Sinus bradycardia	Nor
Marutappa	65	Male	Right fronto parieto temporal EDH	EDH	7/15	severe	58	Sinus bradycardia	Nor
Gundappa	32	Male	Right parietal bleed with SAH	SAH	8/15	severe	42	Sinus bradycardia	Nor
Kallawwa	25	Female	Left parietal contusion	Contusion	13/15	mild	78	Normal	Nor
Basuvanth	54	Male	Multiple right frontal contusion	Contusion	14/15	mild	86	Normal	Nor
Himeshroa	38	Male	Left temporal bleed	IC bleed	11/15	moderate	90	Normal	Nor
Gnagawwa	45	Female	Right fronto parietal EDH	EDH	8/15	severe	92	Normal	Nor
Hanumant	46	Male	Bilateral fronto parietal SAH	SAH	6/15	severe	86	Normal	Nor
Ramesh	35	Male	Right frontal multiple contusion	Contusion	9/15	moderate	98	Normal	Nor
Trenetri Devi	41	Female	Left occipital SDH	SDH	7/15	severe	110	Sinus tachycardia	Nor

Ningawwa	52	Female	Right frontal contusion	Contusion	14/15	mild	72	Normal	Nor
Hemalatha	34	Female	Diffuse axonal injury	Diffuse axonal injury	5/15	severe	48	Sinus bradycardia	Nor
Rang Anna	51	Male	Bilateral parietal contusion with minimal SAH	SAH	8/15	severe	56	Sinus bradycardia	Nor
Bhimashankar	23	Male	Left parieto temporal EDH	EDH	9/15	moderate	102	Sinus tachycardia	Nor
Renuka	34	Female	Left temporo occipital SAH	SAH	10/15	moderate	100	Sinus tachycardia	Nor
Sankarappa	62	Male	Right frontal bleed	IC bleed	8/15	severe	78	Normal	Nor
Shivanand	51	Male	Left parietal contusion	Contusion	13/15	mild	98	Normal	Nor
Daneshwari	39	Female	Multiple temporal contusion with SAH	SAH	7/15	severe	56	Sinus bradycardia	Nor
Tippanna	57	Male	Right frontal EDH	EDH	10/15	moderate	102	Sinus tachycardia	Nor
Ningawwa	58	Female	Left fronto parietal EDH	EDH	7/15	severe	108	Sinus tachycardia	Nor
Balla	34	Male	Right temporal SDH	SDH	6/15	severe	58	Sinus bradycardia	Nor
Nawaz shah	38	Male	Left parietal Bleed with depressed skull fracture	IC bleed	6/15	severe	64	Normal	Nor
Medina	45	Female	Diffuse cerebral edema	Diffuse cerebral edema	14/15	mild	70	Normal	Nor
Ramesh	27	Male	Intraventricular bleed with midline shift	Intraventricular bleed	5/15	severe	50	Sinus bradycardia	Nor
Siddamma	46	Female	Large right hemisphere bleed with midline shift	IC bleed	4/15	severe	68	Normal	Nor
Shivaya	51	Male	Right temporal contusion	Contusion	13/15	mild	90	Normal	Nor
Veerubai	39	Female	Left fronto parieto temporal EDH	EDH	9/15	moderate	86	Normal	Nor
Tamara papa	46	Male	Right frontal SDH	SDH	9/15	moderate	68	Normal	Nor
Mahanatppa	52	Male	Right occipital contusion	Contusion	13/15	mild	90	Normal	Nor
Sonamma	42	Female	Left parietal contusion	Contusion	10/15	moderate	84	Normal	Nor
Krishna Prabhu	34	Male	Right temporal contusion	Contusion	14/15	mild	106	Sinus tachycardia	Nor
Parvati	21	Female	Left fronto parieto occipital bleed	IC bleed	5/15	severe	60	Normal	Nor
Gudusab	25	Male	Bilateral frontal contusion	Contusion	11/15	moderate	90	Normal	Nor
Afreen	22	Male	Diffuse axonal injury	Diffuse axonal injury	5/15	severe	60	Normal	Nor
Vindhya	34	Female	Right frontal bleed	IC bleed	8/15	severe	68	Normal	Nor
Ashok	36	Male	Left frontal SAH with contusion	SAH	13/15	mild	98	Normal	Nor
Rangamma	38	Female	Right fronto parietal EDH	EDH	9/15	moderate	68	Normal	Nor
Ravi Kumar	37	Male	Left temporal SAH	SAH	11/15	moderate	98	Normal	Nor
Guddusab	57	Male	Right temporo-parietal SAH	SAH	7/15	severe	100	Sinus tachycardia	Nor
Bouramma	26	Female	Left parietal contusion	Contusion	13/15	mild	102	Sinus tachycardia	Nor
Totteppa	47	Male	Left frontal bleed	IC bleed	10/15	mild	80	Normal	Nor
Shradda	20	Female	Left parietal contusion with SAH	SAH	8/15	severe	94	Normal	Nor
Appasab	24	Male	Right frontal contusion	Contusion	13/15	mild	98	Normal	Nor
Ningawwa	56	Female	Right fronto parietal EDH	EDH	9/15	moderate	98	Normal	Nor
Kantabai	24	Female	Left temporal contusion	Contusion	14/15	mild	90	Normal	Nor
Sanju	28	Male	Right fronto parietal SDH	SDH	8/15	severe	116	Sinus tachycardia	Nor
Appasabanna	39	Male	Right frontal contusion	Contusion	13/15	mild	108	Sinus tachycardia	Nor
Virajmallappa	54	Male	Left parietal bleed with SAH	SAH	6/15	severe	60	Normal	Nor
Hema	26	Female	Left temporal contusion	Contusion	12/15	moderate	88	Normal	Nor
Doddappa	54	Maley	Left fronto parietal SDH	SDH	8/15	moderate	96	Normal	Nor
Mahanatppa	50	Male	Left temporal SAH	SAH	12/15	moderate	111	Sinus tachycardia	Nor
Mahanth	26	Male	Right frontal multiple contusion	Contusion	13/15	mild	78	Normal	Nor
Timara	39	Female	Left parietal Bleed with depressed skull fracture	IC bleed	6/15	severe	46	Sinus bradycardia	Nor
Yanagappa	44	Male	Intraventricular bleed with midline shift	Intraventricular bleed	4/15	severe	58	Complete heart block	Nor
Yellaweshwari	60	Female	Diffuse axonal injury	Diffuse axonal injury	4/15	severe	89	Normal	Nor
Pramod	29	Male	Right parietal EDH	EDH	11/15	moderate	94	Normal	Nor
Namma	27	Female	Left temporal contusion	Contusion	13/15	mild	88	Normal	Nor

Parvathi	34	Female	Left occipital SDH	SDH	10/15	moderate	74	Normal	Nor
Nagesh	35	Male	Right frontal SAH	SAH	13/15	mild	68	Normal	Nor
Bindu	19	Female	Right frontal multiple contusion	Contusion	13/15	mild	104	Sinus tachycardia	Nor
Suresh	20	Male	Right fronto-parietal SDH	SDH	9/15	moderate	64	Normal	Nor
Shridhar	24	Male	Bilateral fronto parietal SAH	SAH	8/15	severe	54	Sinus bradycardia	Nor
Bhagya	28	Female	Left fronto-parietal EDH	EDH	10/15	moderate	66	Normal	Nor
Vikram	18	Male	Left parietal contusion with SAH	SAH	9/15	moderate	74	Normal	Nor
Shreepad	30	Male	Right fronto-parietal SDH	SDH	8/15	severe	120	Sinus tachycardia	Nor
Tippesh	41	Male	Right fronto parieto temporal SAH	SAH	9/15	moderate	126	Sinus tachycardia	Nor
Gowrawwa	46	Female	Left occipital contusion	Contusion	14/15	mild	98	Normal	Nor
Nirmala	34	Female	Diffuse cerebral edema	Diffuse cerebral edema	14/15	mild	82	Normal	Nor
Ravi Kumar	28	Male	Right occipital contusion	Contusion	13/15	mild	90	Normal	Nor
Soumya Patil	18	Female	Right occipital contusion	Contusion	13/15	mild	111	Sinus tachycardia	Nor
Allabaksh	34	Male	Left frontal bleed with SAH	SAH	8/15	severe	121	Sinus tachycardia	Nor
Chandappa	36	Male	Right fronto-parietal EDH	EDH	12/15	moderate	60	Normal	Nor
Amarappa	50	Male	Right temporo-parietal SAH	SAH	9/15	moderate	102	Sinus tachycardia	Nor
Normal lama	58	Female	Left temporo occipital SAH	SAH	10/15	moderate	58	Sinus bradycardia	Nor
Gormalabai	60	Female	Left occipital contusion	Contusion	14/15	mild	78	Normal	Nor
Mallanna Gouda	74	Male	Right fronto-parieto-temporal EDH	EDH	7/15	severe	84	Normal	Nor
Vidya	26	Female	Left occipital contusion	Contusion	13/15	mild	68	Normal	Nor
Ashwathy	29	Male	Left temporal contusion	Contusion	14/15	mild	94	Normal	Nor
Chandibai	41	Female	Right temporal SDH	SDH	9/15	moderate	62	Normal	Nor
Bheemappa	52	Male	Multiple right parietal contusion	Contusion	9/15	moderate	90	Normal	Nor
Chandrashekhar	56	Male	Right parietal bleed with SAH	SAH	7/15	severe	86	Normal	Nor
Kamla athi	37	Female	Intraventricular bleed with midline shift	Intraventricular bleed	5/15	Severe	54	Sinus bradycardia	Nor
Ningawwa	52	Female	Right occipital contusion	Contusion	13/15	mild	106	Sinus tachycardia	Nor
Allu Rathod	24	Male	Left temporal contusion	Contusion	14/15	mild	98	Normal	Nor
Jayanth	29	Male	Right parietal EDH	EDH	7/15	severe	104	Sinus tachycardia	Nor
Sanju Rathod	51	Male	Left parietal bleed	IC bleed	8/15	severe	68	Normal	Nor
Siddamma	60	Female	Right temporal SAH	SAH	10/15	moderate	74	Normal	Nor
Rajesh	20	Male	Left occipital SDH	SDH	9/15	moderate	84	Normal	Nor
Subhas	42	Male	Left occipital contusion	Contusion	13/15	mild	92	Normal	Nor
Mllappa	36	Male	Right parieto temporal EDH	EDH	8/15	severe	56	Sinus bradycardia	Nor
Yamunabai	54	Female	Left parieto occipital contusion with SAH	SAH	9/15	moderate	108	Sinus tachycardia	Nor
Marappagouda	58	Male	Right frontal SAH	SAH	12/15	moderate	112	Sinus tachycardia	Nor
Nazir	61	Male	Left occipital SAH	SAH	13/15	mild	68	Normal	Nor
Bande Nawaz	23	Male	Right parietal contusion	Contusion	11/15	moderate	75	Normal	Nor
Surukanna	51	Male	Right frontal contusion	Contusion	13/15	mild	62	Normal	Nor
Kamla	24	Male	Right parieto temporal EDH	EDH	9/15	moderate	94	Normal	Nor
Mitra	37	Female	Left parietal SAH	SAH	9/15	moderate	90	Normal	Nor
Shiva papa	28	Male	Right frontal SDH	SDH	8/15	severe	88	Normal	Nor
Shreeshaila	39	Female	Right parietal SAH	SAH	13/15	mild	84	Normal	Nor
Gundubai	42	Male	Left fronto parieto occipital bleed	IC bleed	7/15	severe	58	Sinus bradycardia	Nor
Shibanand	38	Male	Right frontal multiple contusion	Contusion	11/15	moderate	108	Sinus tachycardia	Nor
Farukha	25	Female	Left temporal contusion	Contusion	13/15	mild	98	Normal	Nor
Maruthi	34	Male	Left occipital SAH	SAH	10/15	moderate	68	Normal	Nor
Mallesh	36	Male	Intraventricular bleed with midline shift	Intraventricular bleed	5/15	severe	72	Normal	Nor
Siddamma	43	Male	Diffuse axonal injury	Diffuse axonal injury	4/15	severe	48	Sinus bradycardia	Nor

Meena	21	Female	Left fronto-parietal EDH	EDH	9/15	moderate	68	Normal	Nor
Kamalesh	29	Male	Right parieto temporal SDH	SDH	8/15	severe	92	Normal	Nor
Vikram	34	Male	Left parieto temporal SAH	SAH	10/15	moderate	68	Normal	Nor
Bandenamaj	45	Male	Right frontal SAH	SAH	11/15	moderate	74	Normal	Nor
Hemanthappa	56	Male	Left occipital SDH	SDH	8/15	severe	76	Normal	Nor
Revathi	24	Female	Right frontal contusion	Contusion	13/15	mild	82	Normal	Nor
Kamalabai	35	Female	Left temporal bleed	IC bleed	8/15	severe	84	Normal	Nor
Siddanagouda	45	Male	Right temporo-parietal SAH	SAH	11/15	moderate	88	Normal	Nor
Ambuja	61	Female	Right occipital contusion	Contusion	12/15	moderate	90	Normal	Nor
Tippesh	35	Male	Left occipital bleed	IC bleed	8/15	severe	60	Normal	Nor
Vishalakshi	38	Female	Left parieto occipital contusion with SAH	SAH	10/15	moderate	90	Normal	Nor
Rakesh	27	Male	Right parieto temporal SDH	SDH	8/15	severe	77	Normal	Nor
Someshwaragouda	64	Male	Right temporal contusion	Contusion	13/15	mild	106	Sinus tachycardia	Nor
Mallappa	48	Male	Left parietal SAH	SAH	10/15	moderate	76	Normal	Nor
Mahanadi	25	Male	Right occipital contusion	Contusion	14/15	mild	111	Sinus tachycardia	Nor
Sridevi	29	Female	Right parietal SDH	SDH	7/15	severe	84	Normal	Nor
Tim manna	27	Male	Left parieto occipital EDH	EDH	9/15	moderate	56	Sinus bradycardia	Nor
Vimala	45	Female	Right fronto parieto temporal EDH	EDH	8/15	severe	74	Normal	Nor
Gundappa	41	Male	Left temporal contusion	Contusion	13/15	mild	102	Sinus tachycardia	Nor
Manjula	35	Female	Bilateral parietal SAH	SAH	12/15	moderate	92	Normal	Nor
Siddesh	37	Male	Bilateral frontal contusion	Contusion	14/15	mild	68	Normal	Nor
Mohith	25	Male	Right temporal SAH	SAH	9/15	moderate	58	Sinus bradycardia	Nor
Tamanna	24	Male	Left parietal bleed	IC bleed	7/15	severe	92	Normal	Nor
Rangabai	46	Female	Right fronto parieto temporal SAH	SAH	8/15	severe	108	Sinus tachycardia	Nor
Rangalakshmi	35	Female	Left temporal EDH	EDH	9/15	moderate	120	Sinus tachycardia	Nor
Ramesh	21	Male	Right frontal contusion						

ANNEXURE 4