

An Audit of Moderate to Severe Acute Head Injury Patients in Chris Hani Baragwanath Academic Hospital

By

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Declaration

I declare that the master script, which I hereby submit for the degree Master of Medicine in Neurosurgery at the University of Witwatersrand, is my own work and has not been previously submitted for a degree at another university.

This study will also be submitted for journal publication.

Dr. Antony Thomas

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Dedication

This work is dedicated to my beautiful, loving and caring wife, Mrs. Monique Thomas for her steadfast support, unconditional love, prayers and dedication throughout the hurdles in my life. Thank you for standing by my side no matter what may come, for all your endless help, encouragement in this research work and during my studies.

To my parents Dr. and Mrs. Thomas, my brother Mr. Anil Thomas and my family for their prayers and support during my studies without them all this would have not been possible.

Abstract:

Objectives: Head injury is a devastating condition in developing countries like South Africa, contributing significantly to mortality and morbidity. The various factors affecting outcome like age, gender, mechanism of injury, clinical, radiological findings and treatment is reported. Their relation to outcome (Glasgow Outcome Score) of treatment in Chris Hani Baragwanath Academic hospital is analyzed.

Methods: This is a retrospective, descriptive and demographic profile study. The sample group consists of moderate to severe head injury patients admitted in the neurosurgical unit of Chris Hani Baragwanath Academic hospital from January 2011 to June 2012. The data includes age, gender, nature of head injury (scalp, skull, intracranial), mode of injury (fall from height, road traffic accident, fire arm injury, assault, blast injury), condition at presentation [Glasgow Coma Scale (GCS)], pupillary reaction, Computed Tomography (CT) scan findings, treatment received and outcome [Glasgow Outcome Score (GOS)] of treatment.

Results: A total of 292 patients was enrolled in the study, 258 males (88.3%) and 34 females (11.6%). In the age distribution 50 patients were below 19 years, 161 patients were between 20 to 39 years, 60 patients 40 to 59 years and 21 patients above 60 years. The various mechanisms of injury noted were assault in 127 patients, pedestrian vehicular accident in 50 patients, motor vehicular accident in 33 patients, motor bike accidents in 4 patients, train accidents in 2 patients, gunshot injury in 6 patients, fall from height in 35 patients and struck by heavy object in 5 patients. 123 patients had a GCS between 3-5, 72 patients GCS between 6-8 and 97 patients GCS 8-12. 192 patients had equal and reacting pupils after the head injury, 52 patients unilateral fixed pupils and 10 patients bilateral fixed pupils. The Computed tomography (CT) of the brain showed 287 patients with focal intracranial findings, 107 with diffuse brain injury and 168 patients with features of raised intracranial pressure. 129 patients (44.1%) were surgically treated and 163 patients (55.8%) treated conservatively with medical treatment. The variables age, mechanism of injury, GCS, pupillary reaction, raised intracranial pressure and type of management was compared to GOS and found to be statistically significant.

Conclusions: The outcome of patients with moderate to severe head injury has no effect on gender but has a significant relationship between age and mortality. The mechanism of head injury has a direct effect on the prognosis with gunshot head having the worst outcome. The important prognostic factors affecting the outcome include: age of patients, severity of head injury (GCS), pupillary reactivity to light and the pathology of the brain CT scan. The unfavorable prognostic factors are: old age, non-reacting pupils to light, severe head injury (low GCS) and raised ICP after head injury. Medical or surgical management have similar mortality rate.

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1	Chapter 1: Introduction and General Background
1.1	Introduction
1.2	Background
1.3	Aims and Objectives

Chapter 1: Introduction and General Background

1.1 Introduction:

This chapter gives the overview of the research project. It includes background of the study, the problem statement and the importance of conducting the study of this kind. The aims and objectives are defined.

1.2 Background:

Head injury is defined as injury that may damage the scalp, skull or brain. The most important consequence of head trauma is traumatic brain injury. The clinical assessment and classification of the severity of head injury is commonly based on the Glasgow Coma Scale (GCS), (Teasdale and Jennett, 1974).^[36] Where a score of 13-15 represents mild head injury, 9-12 moderate head injury and 3-8 severe head injury.

Traumatic brain injury is a major health and socioeconomic problem in the world.^[68] In developing countries like South Africa, head injuries contribute to significant mortality and morbidity. It could be the biggest cause of premature death in South Africa. It is the main cause of mortality and disability in young adults and the global incidence is rapidly rising. Among all traumatic deaths 50% are from traumatic brain injury. This requires an audit of present day patient demographics, mechanism of injury and outcome in the South African tertiary premier institutes.

For this reason this study is done in Chris Hani Baragwanath Academic hospital which serves mainly the Sowetan community but also far-lying communities such as Mafikeng, Klerksdorp, Potchefstroom, Sebokeng, Krugersdorp and others. There is a general increase in the incidence of head injuries on holidays and weekends. These are times when coverage in hospitals is often less than optimal. Thus, we are dealing with a very common problem that tends to occur at inconvenient times, demands a lot of time, attention and can lead to disastrous results if not handled rapidly and appropriately.

1.3 Aims and Objectives:

The aim of our study is to audit moderate to severe acute head injury patients in the neurosurgical unit of Chris Hani Baragwanath Academic Hospital retrospectively from January 2011 till June 2012. There is a high impact of Traumatic brain injury to the South African population and has devastating outcome. This study brings about better understanding of the present affected population group, their incidence and outcome which could lead to better methods of prevention and treatment protocols in the high risk groups. It may bring about positive impact on this significant health problem and identifies specific factors to trauma in this population subgroup. This is compared to published results.

Primary Objectives

1. Analyze the clinical profile of patients treated for moderate to severe head injury with respect to:

- * Gender
- * Age
- * Mechanism of injury
- * GCS (Glasgow Coma Scale) on admission
- * Pupillary reaction
- * Computed tomography scan findings
- * Management – Clinical Outcome

Secondary Objectives

2. Analyze the influence of the following variables on mortality and neurological outcome in patients treated for moderate to severe head injury:

- a. Gender
- b. Age
- c. GCS (Glasgow Coma Scale) on admission
- d. Pupillary reaction on admission

2	Chapter 2 : Literature review
2.1	Literature review

Chapter 2. Literature Review:

2.1 Literature Review

Traumatic brain injury (TBI) is a major public health problem causing a great burden to society. It costs billions per year for rehabilitation, support services and loss of income for injured patients. Despite significant research and improved treatment very little can be done to reverse the effect of injury to the brain. So it is logical that prevention of these injuries should take priority to reduce the magnitude of this health problem. The South African study by Nell and Ormond shows that there is an average of 316 per 100 000 incidents of brain-injuries per year.^[55] In the United States at least 1.4 million people sustain traumatic brain injury.^[64]

TBI can be described according to the primary or secondary injury. Primary injury occurs at the moment of insult and is caused by the initial mechanical forces generated by direct trauma to the head. During the primary injury, collision of the head with a surface or contact of the brain inside of the skull leads to epidural or subdural haematomas, subarachnoid or intraventricular hemorrhages, cerebral contusions or diffuse axonal injury. Subdural haematomas are much more common and are present in between 12% and 29% of patients who have sustained a severe TBI in a study by Bullock et al.^[11] Secondary injuries occur within hours to several days after the initial traumatic event and result from ongoing cellular damage from the release of calcium, excitatory amino acids and other neurotoxins in response to impaired cerebral blood flow, oedema or increased intracranial pressure.^[44]

It is known that there are various risk factors affecting outcome of patients with TBI. They are gender, age, mechanism of injury, GCS, pupillary reaction, CT scan brain findings and type of management.

Gender:

Males have increased incidence than females with brain injuries 5:1.^[55] In the United States males are about twice as likely as females to experience a traumatic brain injury. Female patients who sustain severe head injury, especially aged 50 years and younger are significantly more likely to experience brain swelling and intracranial hypertension than male patients with a comparable injury severity, suggesting that younger women may benefit from more aggressive monitoring and treatment of intracranial hypertension.^[22] However, gender has no clear prognostic effects in the multivariable analysis and so was not included in further prognosis table development in the study by Chantal et al.^[13]

Age:

In terms of age distribution African males in the age group 25 – 44 years are most susceptible to suffer brain injuries.^[10] The highest incidence of TBI occurs among males between the ages of 15 to 24 years and those 75 years of age and older.^[40] There are several other studies^[28,42,70] where it is observed that the proportion of survivors with poor outcomes (for example, severe disability or vegetative state) increased with age and that the proportion of patients with favorable outcomes declined. The age of a patient is one of the main prognostic factors and has a strong association with unfavorable outcome than mortality.^[13]

Mechanism of Injury:

In a study by Langlois et al in the United States falls was found to be 28% especially in children 0 to 4 years of age and in adults over the age of 75, this is followed by motor vehicle collisions 20%, assaults 11%, sports-related injuries and other penetrating traumas 13%.^[40] The cause of injury has no clear prognostic effects in the multivariable analysis and so was not included in further prognosis table development in the study by Chantal et al.^[13]

Glasgow Coma Score:

It is generally accepted that the neurological status and age of the patient are the two most important factors in prediction of outcome.^[47,48,62,63] The Glasgow Coma Score is a good objective measure of level of consciousness and today it is the most widely used clinically utility for measurement of severity of head injury. The first GCS score by the neurosurgeons after clinical stabilization and resuscitation is taken into account. This is however complicated by the pre-hospital and hospital treatment (sedation, intubation, pharmacological paralysis). An Australian^[23] and United States^[46] study has shown an inverse relationship between the Glasgow Coma Score and poor outcome.

The Glasgow Coma Score has been extensively tested as a means of rapidly assessing a patient with head injury and making an early and accurate prediction of outcome.^[48,63] However the GCS is not an absolute predictor as there are patients with poor scores who may improve as also patients with good scores who may not show expected improvement.

Pupillary reaction:

Pupillary reaction is a good predictor of outcome. There are class 1 studies showing bilateral absent pupil reaction has greater than 70% positive predictive value for a poor outcome.^[36,46] There is a prospective study of severe head injury where bilaterally absent pupillary light reflex is noted in 35% and a poor outcome (dead, vegetative, or severely disabled) is found in 70% of these patients.^[54] The pupillary reactivity has stronger association with unfavorable outcome in the study by Chantal et al.^[14]

Imaging:

TBI can be classified based on morphological characteristics on computed tomographic (CT) or magnetic resonance imaging (MRI) investigations. In the acute phase CT remains the first choice of investigation. It helps in differentiating focal and diffuse lesions which guides in the management of the patient. Conventional classification of CT findings in severely head-injured patients differentiates between focal (extradural, subdural, intracerebral haematomas or space occupying contusions) and diffuse head injuries.^[26] There are many studies which increasingly uses CT findings and classification as a strong predictor of outcome like the international guidelines on prognosis

include the CT classification as a major predictor based on class 1 evidence.^[16] The study of James S. Heiden shows that, intracranial haematomas is associated with the worst outcome.^[33] A higher mortality appears to occur in patients under 20 years of age when an intracranial haematoma is present in comparison with patients of the same age with a diffuse brain injury.^[34] A. Wani found that 16.7% patients with epidural haematoma had good functional outcome as compared to 11.1% and 12.5% patients having contusions and acute SDH respectively.^[1] Increased intracranial pressure that does not respond to medical treatment is the main cause of death for patients with severe head injury. With severe intracranial hypertension, over 40 mm Hg, cerebral perfusion decreases and ischemia occurs causing severe neurological dysfunction.^[51] Damage that is sufficiently severe to produce moderately increased ICP also produces CT scan appearances of obliteration of the 3rd ventricle and the cisterns at the tentorium.^[2] Adams and Graham reports that when the ICP >40 mmHg, the brain at necropsy shows evidence of focal necrosis in the parahippocampal gyrus. In the absence of haematoma a minority of head injured patients have raised ICP.^[2] Patients with absent cisterns are likely to have raised ICP but only 40% have clinical signs of tentorial herniation and severe midbrain dysfunction.^[21] Radiologic criteria for DAI are small hemorrhagic lesions at the corticomedullary junction, in the corpus callosum, in the midbrain and in the brain stem, sometimes in conjunction with some intraventricular bleeding. DAI can sometimes be superimposed by generalized brain swelling.^[3] In the study of Dereck A. Bruce,^[19] brain edema can be intracellular (cytotoxic) or extracellular as a result of damage to the blood-brain barrier (vasogenic). Vasogenic edema is uncommon in the first 24-48 hours after trauma except surrounding an intracerebral haematoma. The early low-density changes seen in the brain soon after trauma (focal low density on CT scan) are probably the result of ischemia and hypoxia that represent cytotoxic edema. In older children it is more common to see the loss of gray/white matter differentiation occurring 3-5 days after injury.^[19]

Management:

The management of head injury begins immediately after trauma at the accident site and during transit to the hospital. The aim of early treatment is to anticipate and prevent secondary brain damage, which is responsible for increased morbidity and mortality. Triage, resuscitation of airway restoration and circulation should proceed simultaneously with other diagnostic evaluation. All unconscious critical patients require in-field intubation and resuscitation to keep airway patent and prevent further hypoxic brain injury.^[71] On arrival to the hospital the goal of treatment is to continue the brain resuscitation, prevention of secondary injury, to treat life threatening systemic injuries, to initiate imaging studies for diagnosis of cerebral and spinal injuries. The patients are intubated and ventilated if the GCS is equal to or below 8. Documentation of the neurological examination provides the base line for observing changes in the central nervous system status.

At Chris Hani Baragwanath Academic Hospital trauma patients are initially managed and resuscitated by the trauma surgery department before isolated head injuries are transferred to the neurosurgery department for further assessment and specialised care. Patients requiring surgery are transferred rapidly from the CT scanner to the operating theatre, others are transported to the intensive care unit for monitoring and medical treatment.

Good cerebral perfusion pressure is maintained by fluid therapy with normal saline or blood transfusion, if necessary. Prevention of seizures is done by the administration of an antiepileptic. Normothermia and adequate glucose control is maintained. In certain cases ICP is monitored with an ICP catheter. If the ICP is raised then it is treated appropriately by mannitol and furosemide or by cerebrospinal fluid drainage. Any major intracranial haematoma causing mass effect with raised intracranial pressure is surgically evacuated. This is a clinical and radiological decision made by the treating surgeon. There are guidelines outlined by Bullock et al ^[11] on the management of head injury patients which are followed at our institution. Surgical candidates are patients with an EDH greater than 30cm³ with midline shift on CT scan, an SDH with thickness greater than 10mm or midline shift greater than 5mm, parenchymal lesions greater than 50cm³ or more than 20cm³ with GCS below 8, basal cisternal compression with progressive neurological decline, posterior fossa mass lesions with mass effect (distortion, obliteration of fourth ventricle, compression of

basal cisterns, obstructive hydrocephalus) or neurological dysfunction or deterioration and lastly open compound depressed skull fracture greater than the thickness of the cranium with possible dural penetration or pneumocephalus or frontal sinus involvement or gross contamination or wound infection.^[11] Patients with lesions and no significant mass effect on CT with no neurological dysfunction can be managed by close observation and serial imaging.^[11]

In a study in Nepal by Shrestha et al the mortality was found to be higher in severe head injury patients managed conservatively.^[65] But it's known that aggressive management strategy is associated with a decreased mortality rate but no significant difference in functional outcome at discharge among patients.^[20]

Glasgow Outcome Score:

The first description of a Glasgow Outcome Score (GOS) was in 1975 by Jennett and Bond. It allows a degree of standardised description of objective degree of recovery. The outcome predictors are categorized as dead, vegetative, severely disabled or capable of independent survival, based on the best Glasgow Coma Score (GCS) scores obtained within 24 hours of injury by Kaufman et al.^[38] The correct prognosis of outcome is estimated in only about 56%.^[38]

In this study GOS is assessed at discharge of the patient home, back to the referral hospital or rehabilitation centre. Patients with moderate to severe head injury stay in for a week to a few months. The measure of outcome is done using the Glasgow Outcome Score (Appendix-C), this is also used by many international studies.^[54]

3	Chapter 3 : Research Methodology
3.1	Study Designs
3.2	Study Setting
3.3	Sample and Materials
3.4	Method
3.5	Statistical Data Analysis
3.6	Ethical considerations

Chapter 3: Research Methodology

3.1 Study Design

This is a retrospective, descriptive and demographic profile study. The sample group consists of head injury patients admitted in the neurosurgical unit of Chris Hani Baragwanath Academic hospital from January 2011 to June 2012.

3.2 Study Setting

They are patients in the neurosurgery ward of Chris Hani Baragwanath Academic Hospital. This is a tertiary institute which serves mainly the Sowetan community but also a referral for far-lying communities such as Mafikeng, Klerksdorp, Potchefstroom, Sebokeng, Krugersdorp and others.

3.3 Sample and Materials

The data is from the case folders of patients admitted to the neurosurgery unit with head injuries. The information is from patients with moderate to severe head injury. Data regarding age, gender, nature of head injury (scalp, skull, intracranial), mode of injury (fall from height, road traffic accident, fire arm injury, assault, blast injury), condition at presentation [Glasgow Coma Scale (GCS)], treatment received and outcome of the treatment is collected.

3.4 Method

The patients are grouped into mild, moderate and severe injury, based on post-resuscitation admission Glasgow Coma Score (GCS) of 13 - 15, 9 - 12 and 3 - 8, respectively. Only patients with a GCS between 3 and 12 are included in the study.

The following parameters are analyzed and their influence on outcome - age, gender, mode of injury, GCS, CT scan findings, pupillary reaction and

treatment. The primary outcome is assessed by the Glasgow Outcome Scale (GOS) at discharge. Good recovery or moderate disability is considered as a favorable outcome, severe disability, persistent vegetative state or death is considered as an unfavorable outcome.

3.5 Statistical data analysis:

The statistical analysis on the data is to assess the demographic trends in traumatic brain injury admissions. Descriptive statistics for variables including age, gender, severity of injury, mode of injury, GCS on admission, CT scan findings, pupillary reaction and treatment modality is reviewed.

The Pearson's Chi-square Goodness-of-fit tests is explored using version R 3.0.1, whether the differences between the observed versus expected frequency scores for categorical variables is statistically significant. Statistical significance is indicated by a probability score of less than 0.05. The statistics department of the North-West University is engaged with this study.

3.6 Ethical considerations:

The application of this study is approved by the committee for research on human subjects of the University of Witwatersrand and permission is given by the Chief Executive Officer of Chris Hani Baragwanath Academic Hospital to access records. The ethics clearance certificate is no. M1211104 on 30/11/2012 (Appendix D)

Funding's/ Conflicts of Interest

No financial grant applied for or obtained.

The author has no conflicts of interest to declare.

Chapter 4: Research Findings

Results:

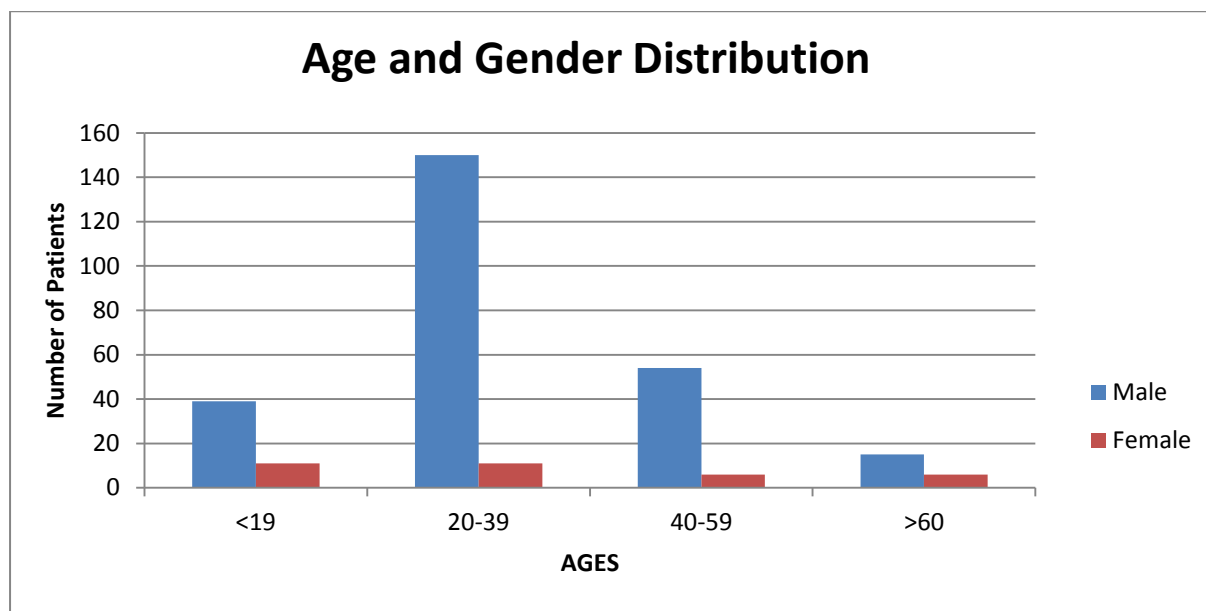
The Age and Gender Distribution of Patients:

A total of 292 patients were enrolled in the study having satisfied the criteria, 258 males (88.3%) and 34 females (11.6%). The age distribution 50 (17.1%) patients (39 males and 11 females) were below 20 years, 161 (55.1%) patients (150 males and 11 females) between 20 to 39 years, 60 (20.5%) patients (54 males and 6 females) were injured between ages 40 to 59 years and 21 (7.1%) patients (15 males and 6 females) above 60 years. As shown in Table (1) and Figure (1) below.

Table no.1 Age and Gender Distribution

Age	Male	Female	Total
<19	39	11	50 (17.1%)
20-39	150	11	161 (55.1%)
40-59	54	6	60 (20.5%)
>60	15	6	21 (7.1%)
Total	258	34	292

Figure no. 1 Age and Gender Distribution

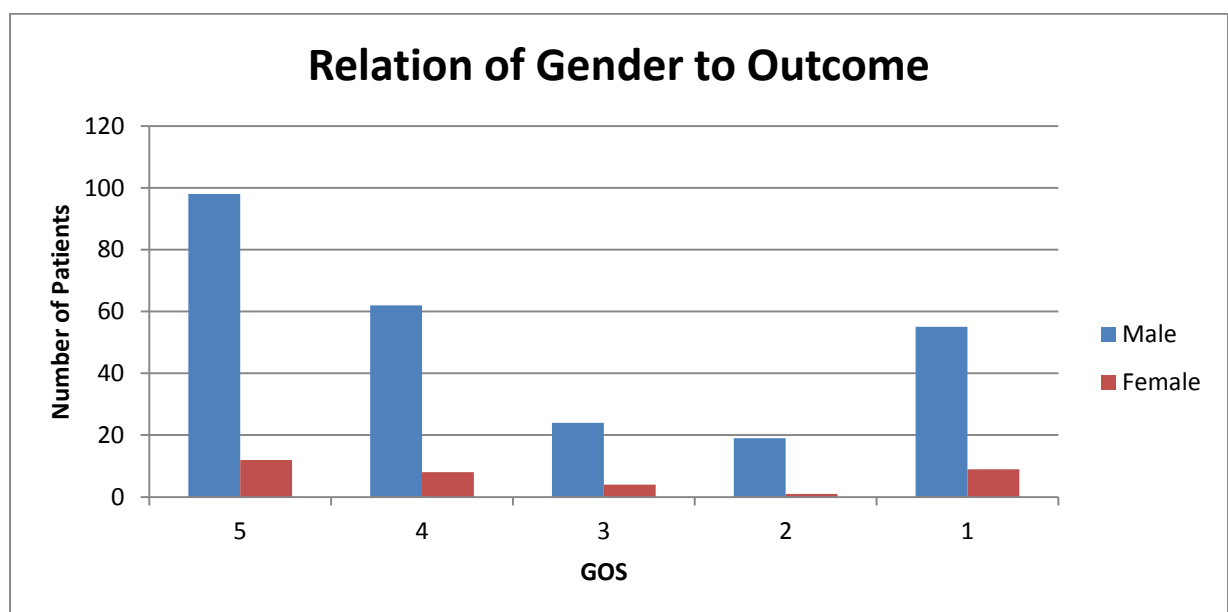


Relation of Gender to Outcome:

Table no. 2 Relation of Gender to Outcome

GOS Outcome	Male	Female	Total
5 Good recovery	98 (37.9%)	12 (35%)	110 (37.6%)
4 Moderate disability	62 (24%)	8 (23%)	70 (23.9%)
3 Severe disability	24 (9.3%)	4 (11.7%)	28 (9.5%)
2 Vegetative state	19 (7.3%)	1 (2.9%)	20 (6.8%)
1 Death	55 (21.3%)	9 (26.4%)	64 (21.9%)
Total	258	34	292 (100%)

Figure no. 2 Relation of Gender to Outcome



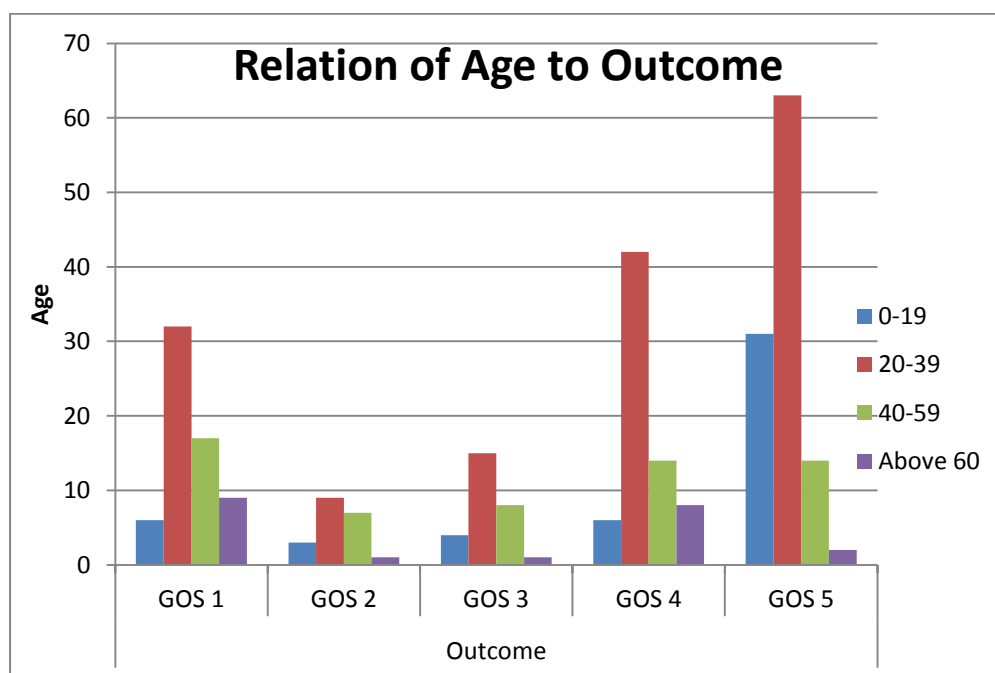
The relation of gender to outcome: the good recovery group was 110 patients: 98 males and 12 females, the moderate disability group was 70 patients: 62 males and 8 females, the severe disability group was 28 patients: 24 males and 4 females, the vegetative group 20 patients: 19 males and 1 female and there were 64 dead patients: 55 males and 9 females. As shown in Table (2) and Figure (2) above. Pearson's Chi-square Goodness-of-fit test shows, Pearson $\chi^2 = 1.4728$ and $Pr = 0.831$.

Relation of Age to Outcome:

Table no.3 Relation of Age to Outcome

Age	Outcome					
	No	GOS 1	GOS 2	GOS 3	GOS 4	GOS 5
0-19	50	6(12%)	3	4	6	31(62%)
20-39	161	32(19%)	9	15	42	63(39%)
40-59	60	17(28%)	7	8	14	14(23%)
Above 60	21	9(42%)	1	1	8	2(0.09%)
Total	292	64(21.9%)	20(6.8%)	28(9.5%)	70(23.9%)	110(37.6%)

Figure no. 3 Relation of Age to Outcome



The relation of age with outcome: the good recovery group was 110 (37.6%), 31 patients was below 19 years, 63 patients was between ages 20 to 39 years, 14 patients between ages 40 to 59 years and 2 patients above 60 years. 70 patients were moderately disabled, 6 patients below 20 years, 42 patients between ages 20 to 39 years, 14 patients between ages 40 and 59 years and 8 patients above 60 years. 28 patients were severely disabled, 4 patients below 20 years, 15 patients between ages 20 to 39 years, 8 patients between 40 to 59 years and one patient above 60 years. There were 20 patients in a vegetative state, 3 patients below 20 years, 9 patients between 20 to 39 years, 7 patients between 40 to 59 years and one patient above 60 years. Death occurred in 64 patients, 6 patients

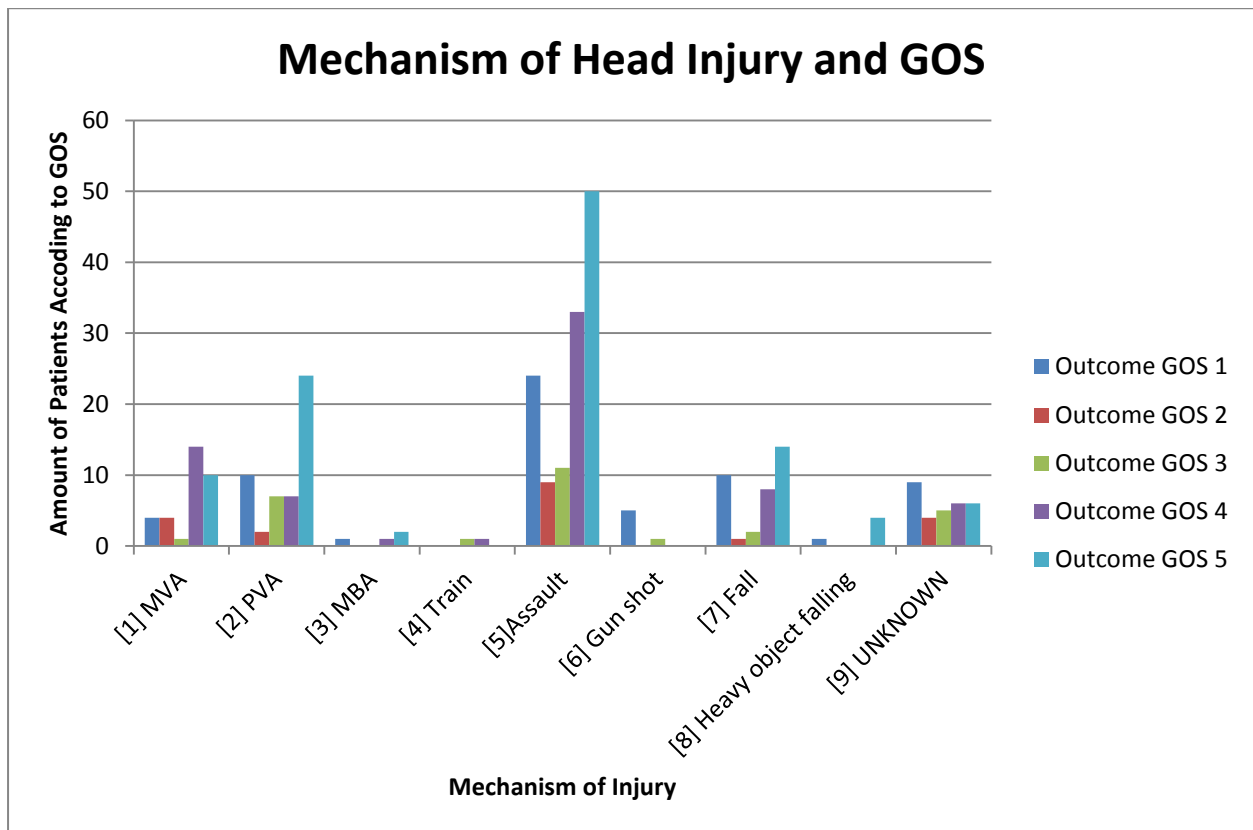
below 20 years, 32 patients below 20 to 39 years, 17 patients between 40 to 59 years and 9 patients above 60 years. As shown in Table (3) and Figure (3) above. Pearson's Chi-square Goodness-of-fit tests shows between age and outcome, the Pearson $\chi^2 = 31.6691$, $df = 12$, $p\text{-value} = 0.001556$. The p value is smaller than 0.05, therefore it is statistically significant.

Mechanism of Head Injury and Outcome:

Table no.4 Mechanism of Head Injury and Outcome

Mechanism of Injury		Outcome				
	No	GOS 1	GOS 2	GOS 3	GOS 4	GOS 5
[1] MVA	33 (11.3%)	4	4	1	14	10
[2] PVA	50 (17.1%)	10	2	7	7	24
[3] MBA	4 (1.3%)	1	0	0	1	2
[4] Train	2 (0.6%)	0	0	1	1	0
[5] Assault	127 (43.4%)	24	9	11	33	50
[6] Gun shot	6 (2%)	5 (83%)	0	1	0	0
[7] Fall	35 (11.9%)	10	1	2	8	14
[8] Heavy object falling	5 (1.7%)	1	0	0	0	4 (80%)
[9] UNKNOWN	30 (10.2%)	9	4	5	6	6
Total	292	64	20	28	70	110

Figure no. 4 Mechanism of Head Injury and Outcome



The mechanism of injury: Assault was the cause of head injury in 127 (43.4%) patients, pedestrian vehicular accident in 50 (17.1%) patients, motor vehicular accident in 33 (11.3%) patients, motor bike accidents in 4 (1.3%) patients, train accidents 2 (0.65%) patients, gunshot injury in 6 (2%) patients, fall from height 35 (11.9%) patients and struck by heavy object in 5 (1.7%) patients.

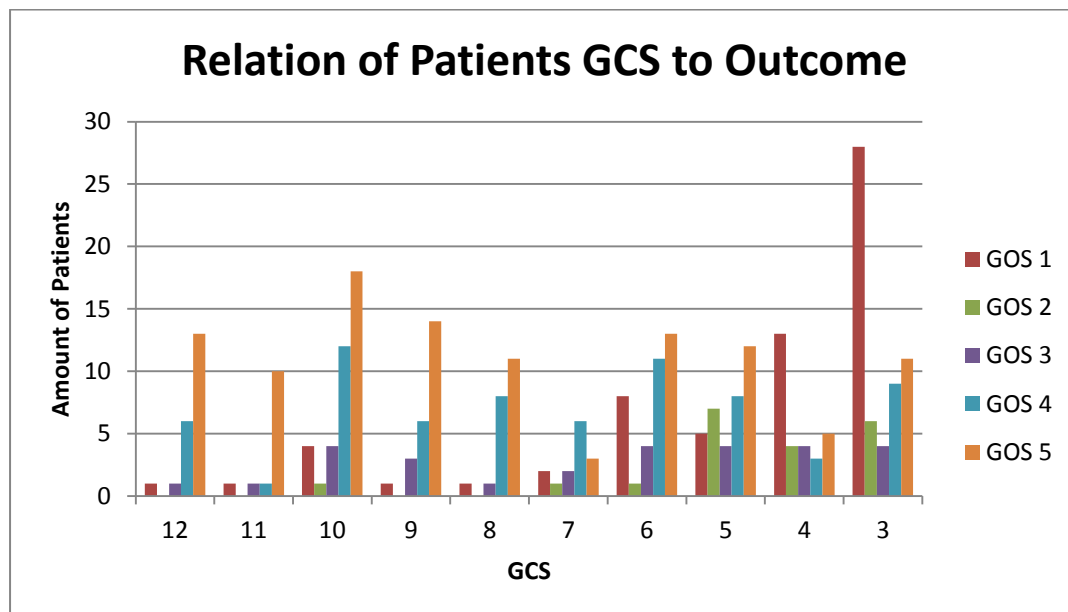
As demonstrated in Table (4) and Figure (4). Pearson's Chi-square Goodness-of-fit tests shows between mechanism of injury and outcome the, Pearson $\chi^2 = 49.9913$, $df = 32$, $p\text{-value} = 0.02234$. The $p\text{-value}$ is smaller than 0.05, therefore it is statistically significant.

Relation of Glasgow Coma Scale (GCS) and Outcome:

Table no.5 Relation of Glasgow Coma Scale (GCS) and Outcome

GCS	No	Outcome				
		GOS 1	GOS 2	GOS 3	GOS 4	GOS 5
12	21	1	0	1	6	13
11	13	1	0	1	1	10
10	39	4	1	4	12	18
09	24	1	0	3	6	14
08	21	1	0	1	8	11
07	14	2	1	2	6	3
06	37	8	1	4	11	13
05	36	5	7	4	8	12
04	29	13	4	4	3	5
03	58	28	6	4	9	11
Total	292	64(21.9%)	20(6.8%)	28(9.5%)	70(23.9%)	110(37.6%)

Figure no. 5 Relation of Glasgow Coma Scale (GCS) and Outcome



In this study, 123 patients (42.1%) had head injury with GCS 3-5, 72 patients (24.6%) with GCS 6-8 and 97 patients (33.2%) with GCS 8-12. The relation of GCS score to outcome is shown in Table (5) and Figure (5). Pearson's Chi-square Goodness-of-fit tests between GCS and GOS shows, the Pearson $\chi^2 = 96.5258$, $df = 40$, $p\text{-value} = 1.419e-06$. The $p\text{-value}$ is smaller than 0.0001, so it is statistically significant.

Pupillary Reaction:

Figure no. 6 Patients and Pupillary Reaction

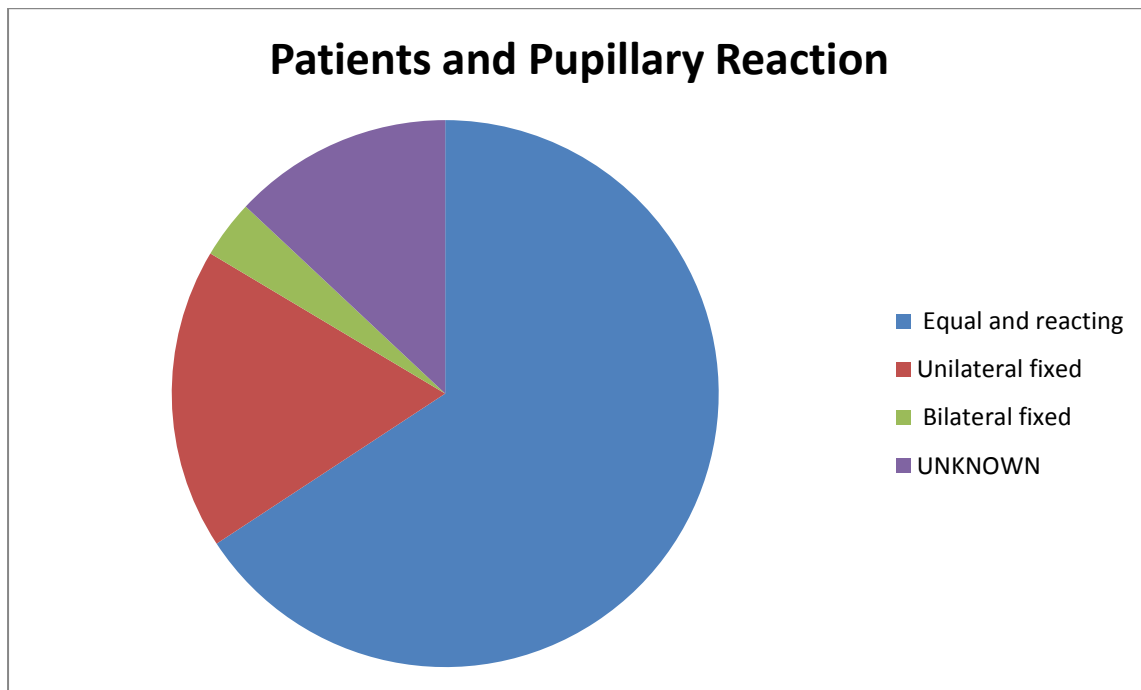
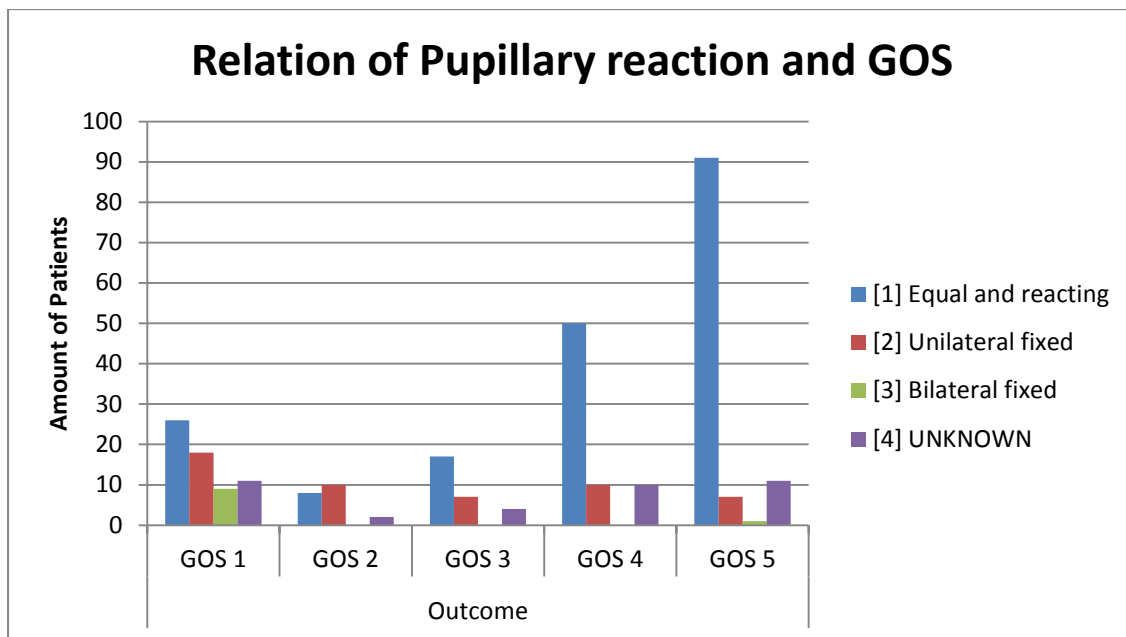


Table no.6 Relation of Pupillary Reaction and Outcome

Pupils	NO	Outcome				
		GOS 1	GOS 2	GOS 3	GOS 4	GOS 5
[1] Equal and reacting	192 (65.7%)	26 (13%)	8	17	50	91 (47%)
[2] Unilateral fixed	52 (17.8%)	18 (34%)	10	7	10	7 (13%)
[3] Bilateral fixed	10 (3.4%)	9 (90%)	0	0	0	1 (11%)
[4] UNKNOWN	38 (13%)	11 (28%)	2	4	10	11 (28.9%)

Figure no. 7 Relation of Pupillary Reaction and Outcome



In this study, 192 patients (65.7%) had equal and reacting pupils after the head injury, 52 patients (17.8%) unilateral fixed pupils and 10 patients (3.4%) bilateral fixed pupils. The relation of pupillary reaction to outcome is shown in Table no (6) and Figure (6). Pearson's Chi-square Goodness-of-fit tests between pupillary reaction and GOS shows the Pearson $\chi^2 = 67.7221$, $df = 12$, $p\text{-value} = 8.524e-10$. The p-value is smaller than <0.001 , so it is statistically significant.

CT scan Brain Findings in Males and Females:

Table no.7 CT scan Brain Findings in Males and Females

CT scan	Gender		
	No	MALE	FEMALE
[1] EDH	65	60	5
[2] SDH	110	95	15
[3] ICH	25	22	3
[4] IVH	15	13	2
[5] PF H	1	1	0
[6] CONTUSION	86	78	8
[7] TSAH	64	59	5
[8] INFARCT	3	3	0
[9] NO BLEED	21	17	4

Figure no. 8 CT scan Brain Findings in Males and Females

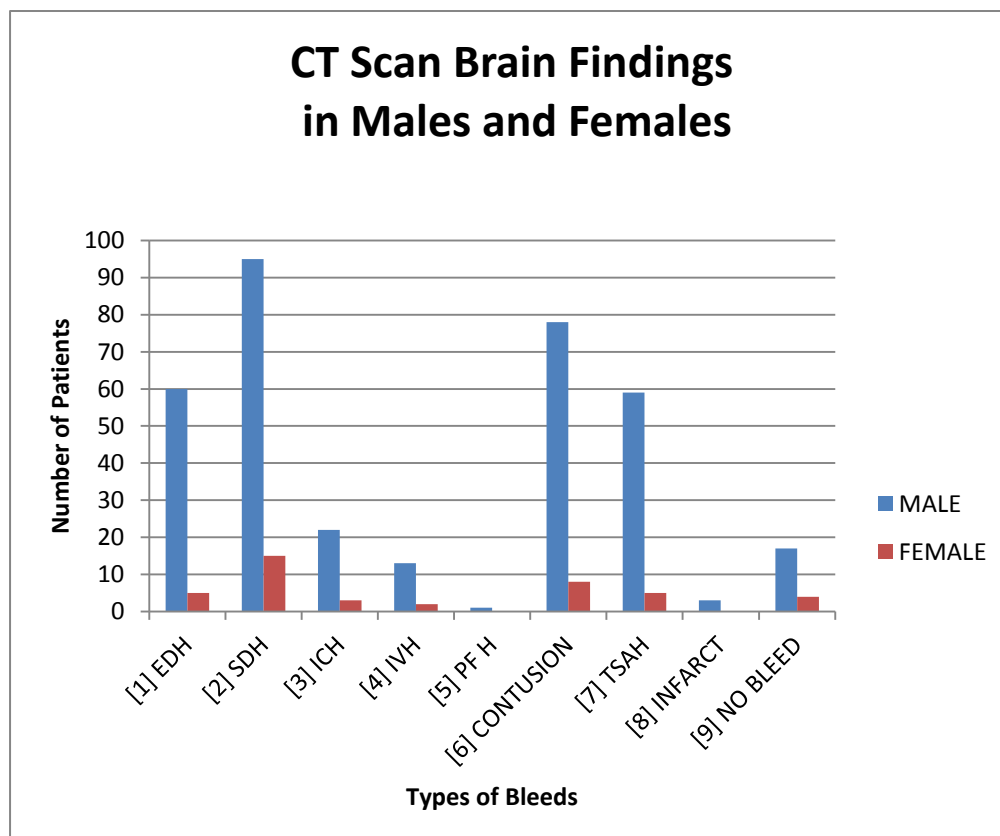


Figure no. 9 CT scan Brain Findings

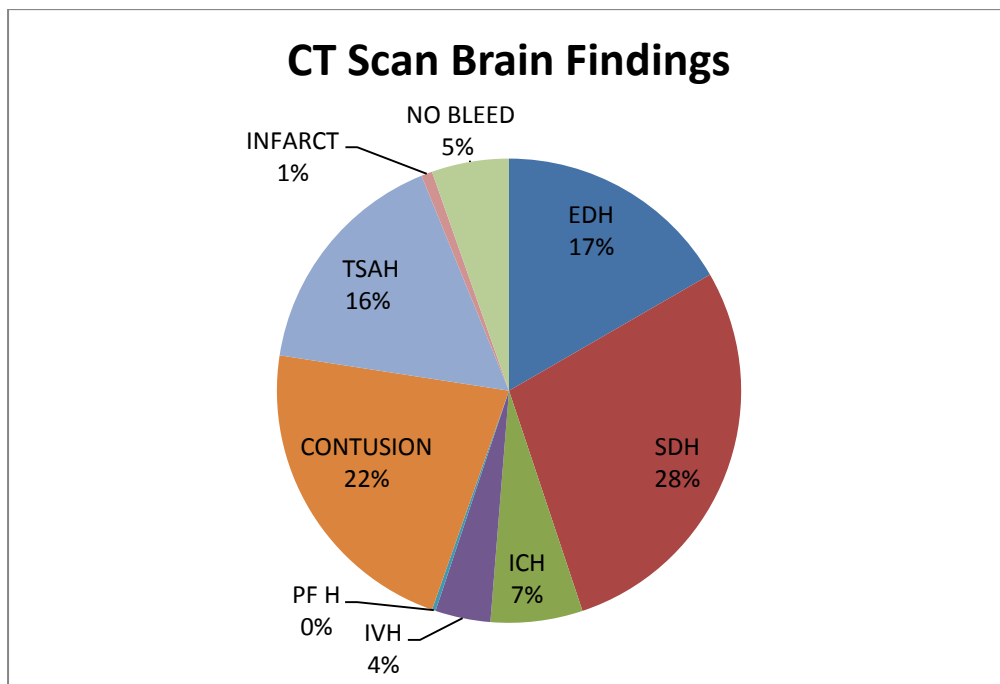
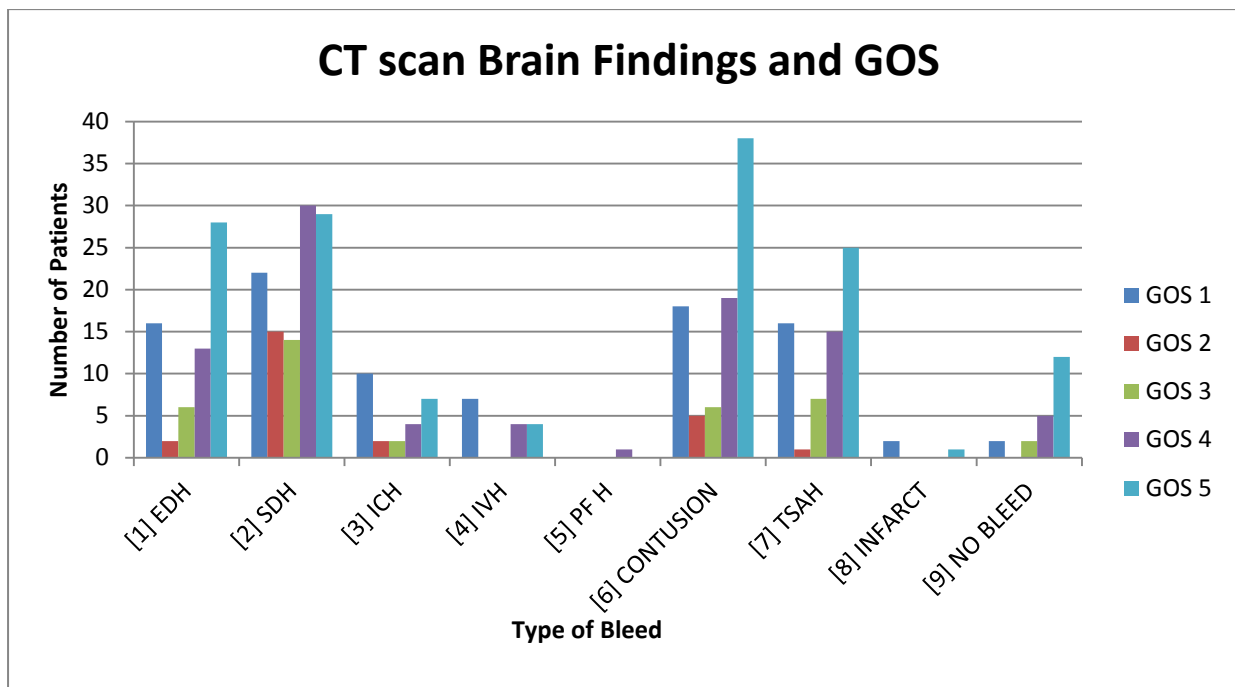


Table no.8 CT scan Brain Findings and Relation to Outcome

CT scan (pathology)	Outcome					
	No	GOS 1	GOS 2	GOS 3	GOS 4	GOS 5
[1] EDH	65	16	2	6	13	28
[2] SDH	110	22	15	14	30	29
[3] ICH	25	10	2	2	4	7
[4] IVH	15	7	0	0	4	4
[5] PF H	1	0	0	0	1	0
[6] CONTUSION	86	18	5	6	19	38
[7] TSAH	64	16	1	7	15	25
[8] INFARCT	3	2	0	0	0	1
[9] NO BLEED	21	2	0	2	5	12

Figure no. 10 CT scan Brain Findings and Relation to Outcome



The Computed tomography (CT) of the brain identified the pathology of the head injury, there were 390 overlapping intracranial findings in 292 patients as demonstrated separately in Table (7), figure (8) in males and females. The various findings were Acute Extradural Haematoma (EDH No.65), Acute Subdural Haematoma (SDH No.110) with the highest incidence, Acute Intracranial Haematoma (ICH No.25), Intraventricular Haematoma (IVH No.15), Posterior Fossa Haematoma (PFH No.1), Hemorrhagic Contusion (No.86), Traumatic Subarachnoid Hemorrhage (TSAH No.64), Ischemic Infarct (No.3), No Bleed (No.21). These are shown in Figure (9).

The relation of CT scan findings to outcome is shown in Table (8) and Figure (10) above. Pearson’s Chi-square Goodness-of-fit tests was done between CT scan intracranial finding and outcome, the Pearson $\chi^2(32) = 44.2784$, $df = 32$, $p\text{-value} = 0.07292$. This was a border line $p\text{-value}$. It is larger than 0.05, therefore it is not statistically significant.

CT Scan Findings of Skull Fractures:

Table no.9 CT scan Findings of Skull Fractures and Outcome

CT scan		Outcome				
	No	GOS 1	GOS 2	GOS 3	GOS 4	GOS 5
[1] Linear fracture	36	7	0	3	11	15
[2] Depressed fracture	23	4	1	1	8	9
[3] Base of skull fracture	11	3	0	0	3	5
[4] No fracture	223	51	19	24	48	81

The CT scan finding of 293 overlapping findings of skull fracture in 292 patients and its relation to outcome is shown in table (9). The various findings were: Linear fracture (No.36), Depressed fracture (No.23), Base of skull fracture (No.11) and No fracture (No.223). Pearson's Chi-square Goodness-of-fit tests between CT scan finding of fracture and outcome shows the Pearson $\chi^2(32) = 9.8254$, $df = 12$, $p\text{-value} = 0.6313$. This $p\text{-value}$ is larger than 0.05, therefore it is not statistically significant.

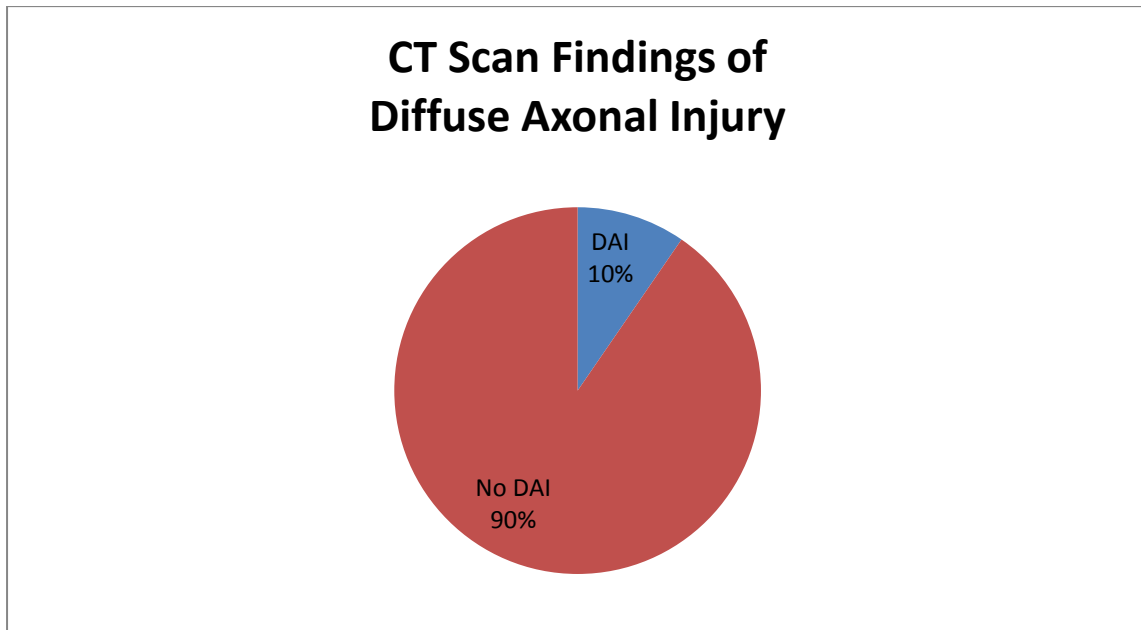
CT scan Finding of Diffuse Axonal Injury:

Table no.10 CT scan Finding of Diffuse Axonal Injury and Outcome

CT scan		Outcome				
	No	GOS 1	GOS 2	GOS 3	GOS 4	GOS 5
DAI	28	4	4	3	8	9
No DAI	264	60	16	25	62	101

The CT scan finding of diffuse axonal Injury (DAI) in 292 patients is shown in Figure (11) below and outcome is shown in table (10) above. Pearson's Chi-square Goodness-of-fit tests between CT scan finding of DAI and outcome shows the Pearson $\chi^2(32) = 3.8889$, $df = 4$, $p\text{-value} = 0.4213$. This $p\text{-value}$ is larger than 0.05, therefore it is not statistically significant.

Figure no. 11 CT scan Finding of Diffuse Axonal Injury



Intracranial Pressure in CT scan:

Figure no. 12 CT scan Finding of Normal and Increased Intracranial Pressure

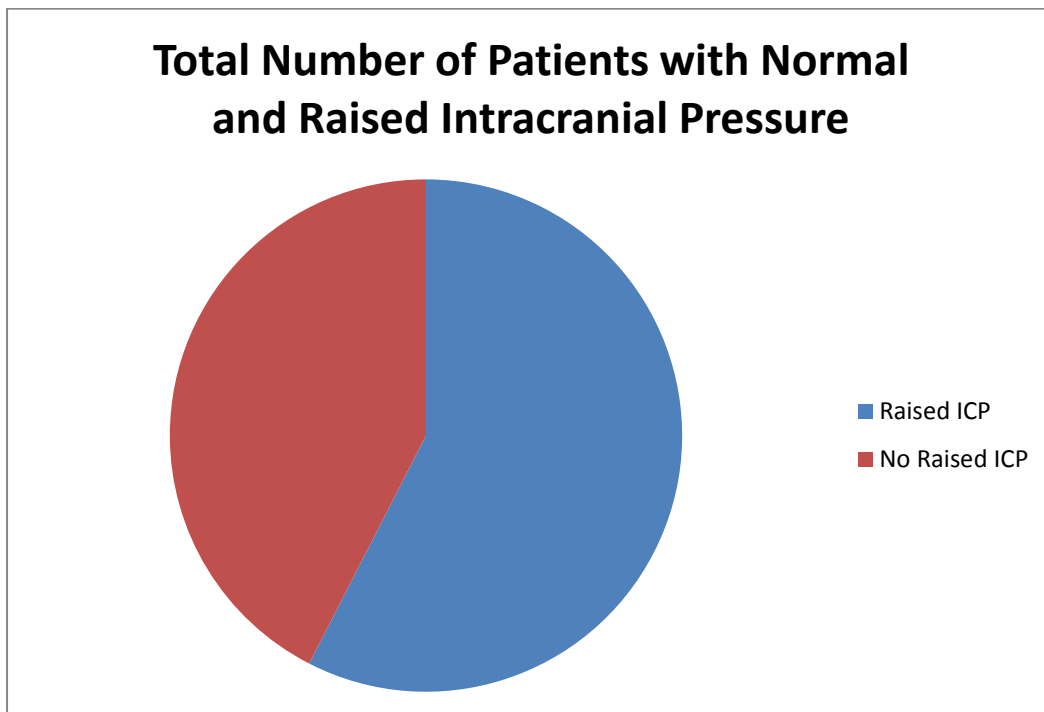
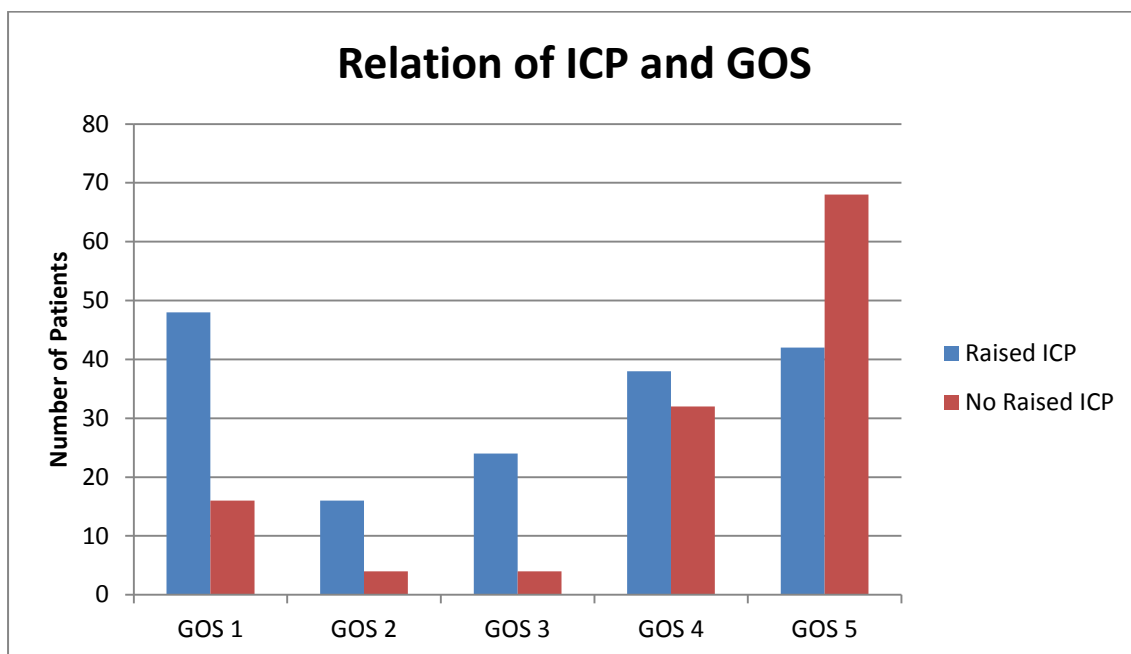


Table no.11 Relation of Increased Intracranial Pressure in CT scan and Outcome

CT scan	Outcome					
	No	GOS 1	GOS 2	GOS 3	GOS 4	GOS 5
Raised ICP	168 (57.5%)	48 (28%)	16	24	38	42 (25%)
No Raised ICP	124 (42.4%)	16 (12%)	4	4	32	68 (54%)

Figure no. 13 Relation of Increased Intracranial Pressure in CT scan and Outcome



In this study, there were 168 patients (57.8%) with features of raised intracranial pressure shown in the CT scan and 124 (42.4%) with no such features as shown in Figure (12).

Their relation to outcome is given in table (11) and Figure (13). Pearson's Chi-square Goodness-of-fit tests between raised intracranial pressure and outcome shows the Pearson $\chi^2(32) = 38.3869$, $df = 4$, $p\text{-value} = 9.324e-08$. The $p\text{-value}$ is smaller than 0.001. Therefore it is statistically significant.

Management Type:

Table no. 12 Management Type

Treatment	Gender		
	No	MALE	FEMALE
Medical	163 (55.8%)	143	20
Surgical	129 (44.1%)	115	14

Figure no. 14 Management Type

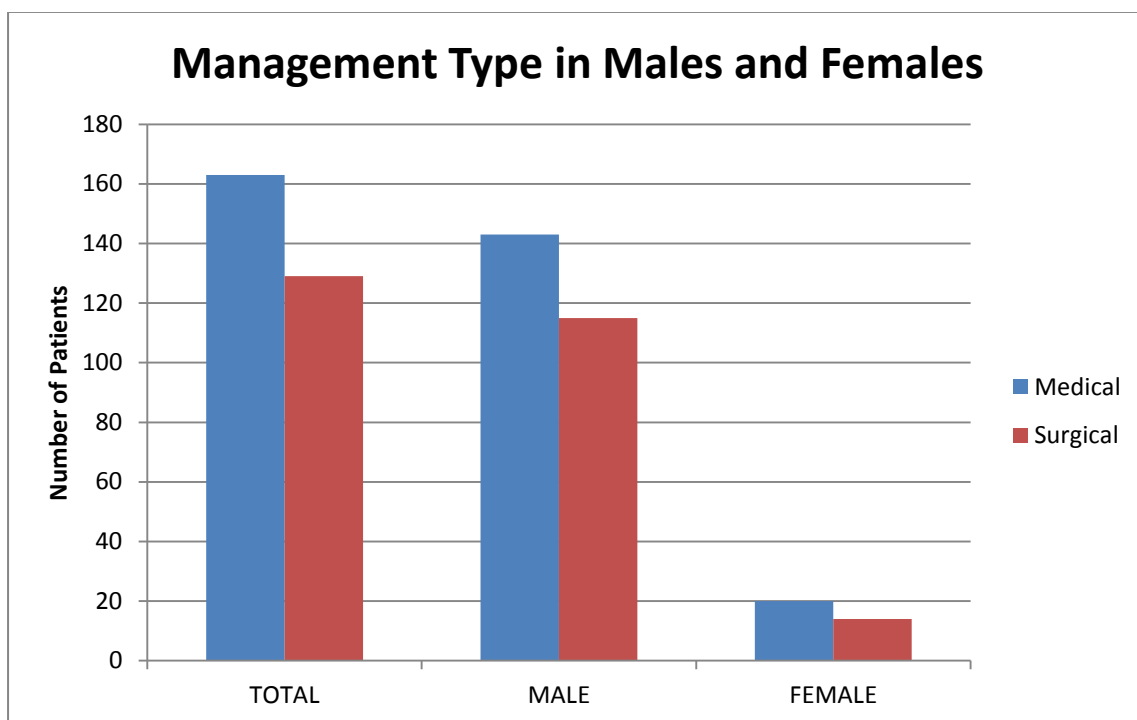


Figure no. 15 Management Type

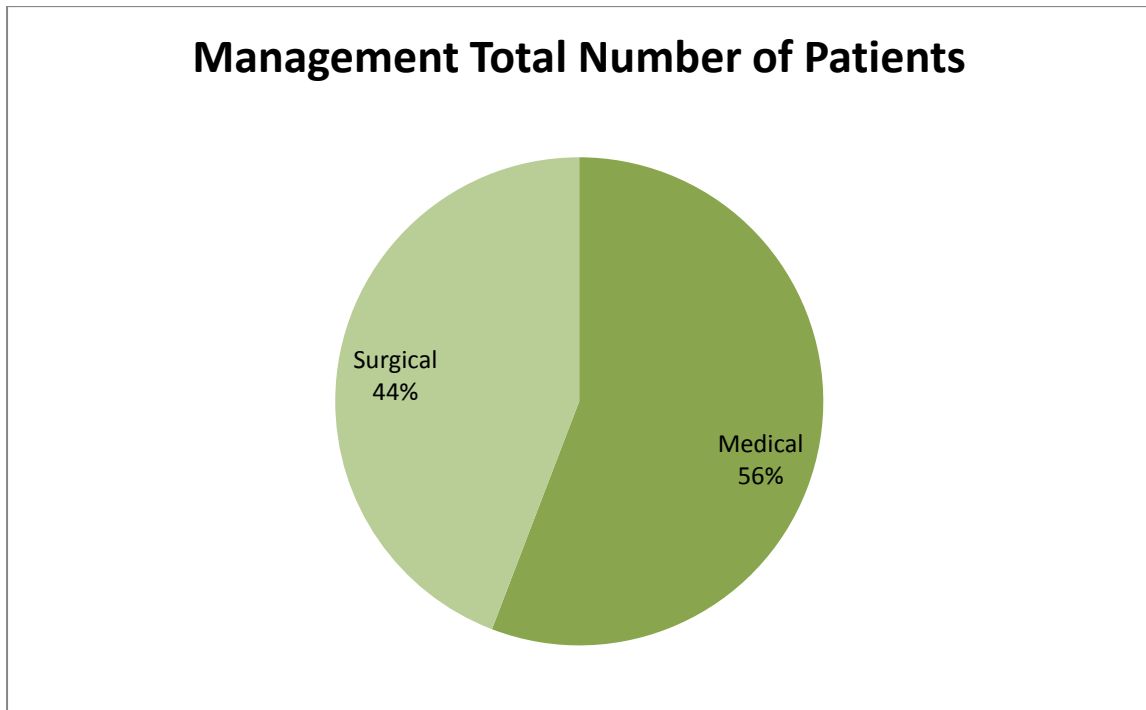
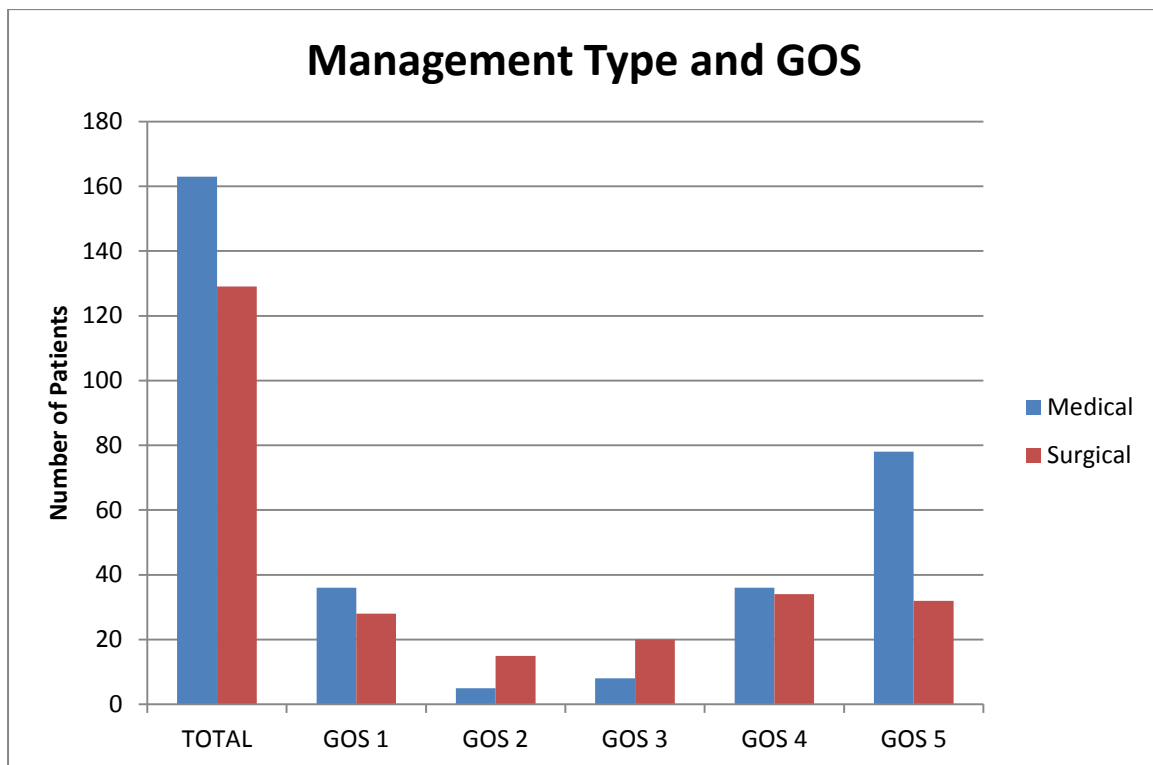


Table no.13 Management Type and Outcome

Management	No	Outcome				
		GOS 1	GOS 2	GOS 3	GOS 4	GOS 5
Medical	163	36	5	8	36	78
Surgical	129	28	15	20	34	32

Figure no. 16 Management Type and Outcome



In this study, 129 patients (44.1%) were surgically treated and 163 patients (55.8%) treated conservatively with medical treatment.

This is shown in table (12), Figure (14) and (15).

The management type and GOS is shown in table (13) and figure (16).

Pearson's Chi-square Goodness-of-fit tests showed the Pearson $\chi^2 = 26.8414$, $df = 4$, $p\text{-value} = 2.14e-05$. The $p\text{-value}$ is smaller than 0.001. Therefore this is statistically significant.

5	Chapter 5 : Discussion and Conclusion
5.1	Discussion
5.2	Limitations
5.3	Conclusion
5.4	Recommendation

Chapter 5: Discussion and Conclusion

5.1 Discussion:

Gender:

In this study, the male: female ratio is approximately 7:1, there were 258 males (88.3%) and 34 females (11.6%). There were more males than the study of A.Wani in Kashmir, India^[1] (39 males and 9 females (M:F ratio 4.3:1). The studies from Europe have shown the male: female ratio varies from 1.2:1 to 2.7:1 in Sweden and Spain respectively. In the United States, the exposure of males to violence and RTAs leads to a male : female ratio of head injury incidence of about 4:1 in the study by Langlois et al.^[41] In the study of Jess F. Kraus,^[37] of 313 individuals, 263 were males (84.0%) and 50 were females (16.0%) making a ratio of 5:1. Males in developing countries have a much higher risk of TBI compared to those in developed countries as shown in this study and others.^[31] In the European Brain Injury Consortium (EBIC) study of severe head injuries, 74% of the patients were males.^[53] In the Traumatic Coma Data Bank of patients with severe head injury, about 77% were males.^[24] In the CRASH study, 81% were males.^[17] All the above studies agree with this study (male predominance). The male excess in TBI is attributed to greater exposure and more risk-taking behavior during occupation or life.

Regarding the relation of gender to outcome in this study, there is no conclusive gender effect on the outcome of head injury. The good recovery group GOS 5 was 37 % in males and 35% in females, while death and the vegetative state group GOS 1 and 2 is 28.6% in males and 29% in females. In the study of Jess F. Kraus^[37] the GOS scores at discharge does not show a significant trend by gender, 60% of females and 51.4% of males had poor outcomes (persistent vegetative state or severe disability). But, after controlling for age, admission GCS, blunt or penetrating injury and multiple trauma high mortality rates, poorer outcomes was found in females in comparison to males by Jess et al.^[37]

In other studies gender had no prognostic value like in study by Chantal et al.^[13] The number of female patients were small, which may not give an accurate statistical result in our study as well as others.^[67]

Age:

The highest incidence in this study is between ages 20 to 39 years, 161 patients about 55 % with higher number of male patients. This may be due to the risk taking behavior of this age group and socio-economic divide in South Africa. This study agrees with the findings of European Brain Injury Consortium (EBIC) study in which patients were admitted to neurosurgical centers in 12 European countries, the median age of the subjects is 38 years with a higher preponderance of male patients.^[31] In other studies highest incidence of head trauma was reported in adolescents and young adults. In the study by Langlois J et al, TBI was more likely in children aged 0–4 years due to falls and adolescents aged 15–19 due to motor vehicular incidents.^[41] Among those attending Accident and Emergency departments in the UK with head injuries the highest rates were observed in urban males aged 15–19 years.^[31]

Regarding the relation of the age with the outcome in this study, the results demonstrates that the best outcome occurs in the age below 20 years (good to moderate recovery were 74%), to a lesser extent in patients between 20-40 years (good to moderate recovery were 65%), while between 40-60 years (46%) had good to moderate recovery and above 60 years (47%) had good to moderate recovery. The younger patients had better outcome to treatment than older patients who had worse prognosis. This is in agreement with international literature. The chances of survival in patients with intracranial haematomas decrease with advancing age.^[5] Age is found to be an independent predictor after other factors are excluded. The proportion of survivors in the Glasgow Outcome Scale scores of good recovery (GCS scores 5, 4, and 3) all declined with age.^[37] The result in this study agrees with the study of James S. Heiden.^[33] who demonstrated that the age had an adverse effect on outcome following a severe head injury. In the study of Randall M^[60] the prognosis for recovery from head trauma as one ages is a function of the type of injury that occurs in each age group. In the last few decades, several authors have identified age as a strong prognostic indicator following injury to the brain.^[8] One group indicated that the outcomes tend to be better in children under ten years of age,^[32] while others reported that children under 5 have a higher mortality rate.^[8] Several

large pediatric head injury series have reported that children have a lower mortality than adults, while others report that the primary mortality rate does not differ between children and adults. Additionally, some investigations report better outcomes below the age range of 40-50 years.^[8] A prospective study of age and outcome from the TCDB reveals that patients older than 60 had a significantly worse outcome, six months after severe head injury, 92% were dead, vegetative or severely disabled. Several studies demonstrated a mortality of greater than 75% in severely brain injured patients older than 60.^[7] Gutterman and Shenkin found that among the patients who decerebrate after head injury, younger patients did better than older ones.^[30] Age effects the outcome in many ways and the common one was mechanism of injury and association of medical illnesses.^[52] In the study of Chantal W.P.M. Hukkelhoven^[14] the proportion of survivors with poor outcomes increases with age and that the proportion of patients with favorable outcomes declines.

These results support the hypothesis that the adult brain has a decreased capacity for repair as it ages,^[50] because of a decreasing number of functioning neurons and a greater exposure to minor repetitive (often subclinical) insults to the brain as age increases. In adults, however diminished cognitive or behavioral function may be influenced beneficially by regeneration or plasticity of the brain.^[18] The patients age is thought to be a strong predictor of morbidity and mortality following severe closed head injury.^[32] The older patients are more likely to have intracranial mass lesions, particularly subdural haematoma regardless of injury mechanism. The reasons for this haemorrhagic tendency may include cerebral atrophy with change in the viscoelastic properties of the brain, alterations in the mechanical properties of the bridging veins and stress on venous structures secondary to cerebral atrophy. Some authors have suggested that one of the pathophysiological mechanisms behind this effect may be due to increased sensitivity to ischaemic brain damage associated with mitochondrial dysfunction shown both with advancing age and severe head injury.^[61] Several medical conditions are more prevalent in old age, such as ischaemic heart disease, hypertension, arrhythmia's, chronic obstructive airway disease, gait disturbances and diabetes mellitus. Such illnesses are known to impact negatively on outcome in elderly trauma victims.^[59]

Mechanism of injury:

Assault (43%) is the most common mechanism of head injury in this study followed by pedestrian vehicular accident (17%). This could be due to the present social and economic conditions in South Africa. This was not in agreement to studies from developed nations and by comparing the mechanism of head injury in this study to others, in a review of European studies, 21%–60% of TBIs were caused by RTAs (from 21% in Norway & UK to 60% in Sweden and Spain); 15% (in Italy) 62% (in Norway) were caused by falls.^[68] The Glasgow and Scotland study reported violence/assault (28%) as the second most common cause after falls (46%).^[69] In Europe, TBIs caused 40% RTAs, 37% falls, 7% violence/assault and 16% by other activities.^[68] In the EBIC study of patients with GCS \leq 12, 51% were involved in RTA, 12% in falls and 5% in assaults.^[31] In the USA, RTAs accounts for 50%, falls for 23%–30% and assaults for 20% of head injuries. In the USA gunshot wounds to the head is now a more frequent cause of serious head injury than RTA with a case fatality of about 90%.^[9] In a study from Canada, RTAs accounted for 43% and assault for 11% of head injuries. In the CRASH trial, the RTAs accounted for 64% and falls 13% of all head injuries.^[39] Sports may account for up to 5%–10% of head injuries.^[35]

In a study of TBI in children, the most common cause of injury was accidents involving children as pedestrians (36%), followed by falls (24%), cycling accidents (10%), motor vehicle occupants (9%) and assault (6%).^[56] The distribution of causes of head injury in children varies according to severity, with falls predominant for accident and emergency attenders and admissions, and RTA is the major cause for neurosurgical unit transfers, severe injuries, and deaths.^[58,59] The distribution of victims of RTA with head injuries are different for children, with fewer car occupants, more pedestrians and cyclists. Among fatal RTAs concerning children, pedestrians were more common, 69% in the Newcastle series.^[27] In the study of the Traumatic Coma Data Bank (TCDB), motor-vehicle accidents were the cause of injury in 55% of patients ages 15–25, whereas only about 5% suffered falls. However, in the age range above 55, 45% suffered falls and only about 15% were in motor-vehicle crashes.^[24] An examination of injury type with respect to age demonstrates an increasing proportion of injuries secondary to falls and pedestrian accidents with advancing age.^[5] The etiology of head injury changes across the age spectrum.

Falls and pedestrian injury are more common in older age groups while the incidence of RTA declines. An increasing incidence of sensory deficit, muscle weakness, gait unsteadiness and arrhythmia contribute to the higher risk of falls in older patients.^[28]

In this study the relationship of mechanism of injury and outcome to treatment, the worst prognosis is for gunshot head. When compared to international literature, the cause of injury had no clear prognostic effects in the multivariable analysis and so was not included in further prognosis table development in the study by Chantal et al.^[13]

GCS (Glasgow Coma Scale):

This study shows that low GCS leads to poor outcome to treatment and patients with a higher GCS had better prognosis. It is directly related and is a significant prognostic indicator. This is in agreement with all international studies. In the Jennett and Teasdale study, functional outcome (GOS 4,5) was only in 7% of patients having GCS of 3 or 4. Motor response was an important predictor of outcome and outcome improves with increasing GCS^[6,14], the GCS shows a clear linear relation with mortality. Increasing age was associated with worse outcomes but this association was apparent only after age 40.^[30,66] The GCS identifies favorable neurological signs. These are eye opening, motor responses such as localizing. Negative signs such as absent eye opening, no motor response or motor responses of extension are prognostic signs of poor recovery and are associated with a mortality rate of 85-91%. The presence of intact brainstem reflexes within 24 hours after head injury, improves the prognosis for recovery.^[33] In a prospective study by Narayan, a positive predictive value of 77% for a poor outcome (dead, vegetative state or severely disabled) was shown for patients with a GCS score of 3-5 and 26% poor predictive value for a GCS score of 6-8.^[54] In a United States study on 746 patients by Marshall et al, the interval from the injury to outcome assessment was variable and ranged from 11 to 1199 days with a median of 674 days. The mortality rate for those with an initial post traumatic GCS score of 3 was 78.4%, initial GCS score of 4 was 55.9%, initial GCS score of 5 was 40.2%. Of note was that 4.1%, 6.3% and 12.2% of the three groups, respectively had a good outcome.^[46] In this study the mortality rate for those with an initial post traumatic GCS score of 3 is 48%, initial GCS score of 4 is 44% and initial GCS score of 5 is 13%. Good outcome is seen in 18% with an initial GCS score of 3, 17% with GCS of 4 and 33% with

GCS of 5. So, in moderate to severe head injury low GCS is an important factor prognosticating the outcome.

Pupillary reaction:

In this study, 141 patients with reacting pupils have good to moderate recovery while one patient without reacting pupils has good/moderate recovery. In the death group, 90% of patients have non-reacting pupil and only 13% patients have reacting pupils. This means that reacting pupils are a favorable prognostic factor. This result agrees with the study of James S. Heiden,^[33] who had found that reacting pupils are a favorable prognostic sign; 49 percent of these patients have moderate disability to good recovery, 15% are in the severe disability group, and 36% died or are in a vegetative state. Nonreactive pupils indicate a worse situation; only 3% have moderate disability or good recovery, 6% are in the severe disability group, and 91% died or in a vegetative state.^[33] Bilateral unreactive pupils occur in 20%–30% of severe TBI patients and predict a 70%–90% chance of poor outcome. Asymmetrical pupils predict the presence of an operable mass lesion in about 30% of cases.^[17] In the study of A. Wani,^[1] only 3 (6.2%) patients had normal reacting pupils, 32 (66.6%) patients with fixed dilated pupils and anisocoria was seen in 13 (27.0%) patients. Patients with normal pupils have better outcome than those with anisocoria and the worst outcome is seen in patients with fixed dilated pupils ($p < 0.05$). The incidence of pupillary abnormalities in patients with severe head injury by the studies of Jennett, Braakman, Narayan and others within 24 hours, post-resuscitation, demonstrated that an average of 65% of patients with severe head injury had normally reactive pupils after resuscitation, one abnormal pupil in 12% and bilateral pupillary nonreactivity in 28%.^[57] There is a significant interaction between pupillary reactivity and the Glasgow Coma Scale (GCS)^[57] hypotension, and CT basal cisterns. In the study of Lawrence F. Marshall^[46] among patients who had reactive pupils throughout their hospital course, only 8.5% were dead or vegetative at last contact. In contrast, among patients who had reactive pupils following resuscitation and then develop one pupillary abnormality, 9 (50%) of 18 were dead or vegetative. When both pupils were fixed and unreactive immediately following resuscitation, 151 (74%) of 209 died or were vegetative.^[43] In moderate to severe head injury non-reactive pupils is an important factor prognosticating the outcome.

Computed tomography scan findings:

In this study the SDH (28%) is the most common CT brain intracranial finding followed by EDH. In skull fracture, linear is the most common followed by depressed skull fracture. In the study by Gutman MB et al acute SDH was the most common encountered operable lesion^[29]. The Computed tomography (CT) of the brain in this study identifies 287 patients with focal intracranial findings, 107 with diffuse brain injury and 168 patients with features of raised intracranial pressure. In the CT scan brain findings 28% have SDH, 17% have EDH, contusion is found in 22%, TSAH in 16%, IVH in 4%, ICH in 7%, PFH 0%, Infarct 1% and no bleed in 5%. In the study of Abrar Ahad Wani, the CT finding in 48 patients demonstrated ICH in 14 patients, contusion in 18 and brain oedema in 14 and normal 2 patients.^[1]

In reference to recovery and the brain CT scan in this study shows that the best prognosis is with intracranial hemorrhage, epidural hemorrhage 43% and in base of skull fracture. This study did not show any statistically significant result with CT scan findings and outcome of treatment. The study of James S.Heiden showed that, intracranial haematomas has been associated with the worst outcome.^[33] Patients with diffuse injuries are found to have an intermediate prognosis when compared to patients with epidural or subdural haematomas. While acute subdural haematomas with low GCS scores have a high mortality, diffuse injuries with higher GCS scores showed a low mortality and a high incidence of good recovery. Outcome is significantly better in extradural haematoma without concomitant brain swelling, simple brain contusion, generalized swelling and in the absence of lesions.^[45]

In this study there are 168(57.5%) patients with raised ICP. Increased intracranial pressure was associated with poor recovery, with a greater percentage of patients having classifications of severe disability, vegetative state, or death in the study by Miller at al^[51] which was also shown in this study. Patients with raised intracranial pressure in the CT scan had a 28% mortality rate while 25 % showed good functional recovery, in comparison 54% with no signs of raised ICP had good recovery.

In moderate to severe head injury increased ICP is an important factor prognosticating the outcome.

Management:

In this study, 129 patients (44%) were treated surgically and 163 patients (56%) conservatively. The surgically patients were those patients with intracranial haematoma, whether extradural, subdural or intraparenchymal in addition to those patients with depressed skull fractures. The decision to operate on a head-injured patient was based on: premorbid state, the severity of initial injury, the rapidity of neurological deterioration and patient assessment on arrival at the neurosurgical unit.^[25] Dereck A. Bruce stated that if the epidural or subdural haematomas was removed before the onset of coma, rapid and almost complete recovery is to be expected because there is minimal underlying primary brain injury. Delay in surgical treatment continues to be a major preventable cause of morbidity and mortality.^[4] In this study between the types of treatment, medically and surgical had similar outcomes in terms of mortality, 22% in medical and 21% in surgical candidates. However, good outcome at discharge to treatment was shown more about 47% in medical than 24% in surgically treated patients. This could be due to patients with poor GCS or prognosis were surgically candidates but not necessarily recover better. In a study by Shrestha et al the mortality GOS at discharge was also found to be higher in patients managed conservatively.^[65] It is shown that aggressive management strategy was associated with a decreased mortality rate, but no significant difference in functional outcome at discharge among patients by Eileen Bulger et al.^[20]

5.2 Limitations:

The limitations of this study is that it is a

- Retrospective study.
- Extracranial injured patients not included.
- Some factors not studied like blood pressure, hematocrit, coagulation profile, pupillary size, timing of patients entering emergency department and entry to intensive care unit or operating theatre.
- CT scan findings are recorded from patient files.
- Consecutive patients could not be included and less number of female patients included in the study compared to males.

5.3 Conclusions:

- The gender of the patient has no significant conclusive prognostic effect on outcome of patients with moderate to severe head injury, however the age has a direct effect on mortality and outcome.
- The mechanism of head injury has direct effect on prognosis of severe head injury.
- The important prognostic factors affecting the outcome: age of patients, severity of head injury (GCS), pupillary reactivity to light and the pathology on brain CT scan.
- The unfavorable prognostic factors include: old age, non-reacting pupil to light, severe head injury (low GCS) and raised ICP after head injury.
- Medical and surgical management have similar mortality rate.

5.4 Recommendations:

From my study I would suggest that the authorities should make suitable changes like health education in school, bridging the socioeconomic divide and safer commutable roads with increased public transport systems to prevent this major health and socio-economic problem. I feel it is imperative that the seriousness and complexity of traumatic brain injury in this study must be illustrated to patients, relatives, doctors, society alike. In this study it shows that the mortality and morbidity affect mostly young adults which are the bread winners for an entire family. There should be special focus on this population subset group and a further study to find out why assault 43% is the largest cause of moderate to severe head injury in this study. There needs to be a standardized epidemiological monitoring to form basis of appropriated targeted prevention of head injury. There should be specific focus on trauma organization and specific care for all head injury patients with a multi-disciplinary team. There needs to be a centralization of care from emergency systems to rehabilitation care. We hope there would be more and improved methods of TBI trials in South Africa which require multidisciplinary efforts from researchers and clinicians with appropriate funding.

6. References:

- 1. A. Wani, A. U. Ramzan, A. R. Kirmani, A. Sherwani, N. K. Malik, A. R. Bhatt, S. S. Chibber & M. A. Wani: Functional outcome following severe head injury in decerebrating patients. The Internet Journal of Neurosurgery. 2009 Volume 6 No.1.**
- 2. Adams JH and Graham DI. The relationship between ventricular fluid pressure and the neuropathology of raised intracranial pressure. *Neuropathol Appl Neurobiol* 1976;2:323-32.**
- 3. Adams JH, Graham DI, Murray S, et al.: Diffuse axonal injury due to nonmissile head injury in humans. An analysis of 45 cases. *Annals of Neurology* 12:557-563, 1982.**
- 4. Alexander S, Kerr M, Kim Y, Kamboh M, Beers S, Conley Y. Apoprotein E4 allele presence and functional outcome after severe traumatic brain injury. *J Neurotrauma* 2007; 24: 790–7.**
- 5. Amacher A, Bybee DE: Toleration of head injury by the elderly. *Neurosurg* 20:954, 1987.**
- 6. Bahloul M, Hamida CB, Chelly H, Chaari A, Kallel H, Dammak H, Rekik N, Bahloul K, Mahfoudh KB, Hachicha M, Bouaziz M. Severe head injury among children: Prognostic factors and outcome. *Injury*. 2008 Aug 12.**
- 7. Berger MS, Pitts LH, Lovely M, et al.: Outcome from a severe head injury in children and adolescents. *J Neurosurg* 62:194-199, 1985.**
- 8. Braakman R, Glepke GJ, Habberna JDF, et al.: Systematic selection of prognostic features in patients with severe head injury. *Neurosurg* 6:362-370, 1980.**
- 9. Bruns J, Hauser W. The epidemiology of traumatic brain injury: a review. *Epilepsia* 2003; 44 (Suppl 1): 2–10.**
- 10. Bruns J Jr, Hauser WA. The epidemiology of traumatic brain injury: a review *Epilepsia*. 2003;44 Suppl 10:2-10.**
- 11. Bullock MR, Chestnut R, Ghajar J, et al. Surgical management of acute subdural haematoma. *Neurosurgery*. 2006; 58(Supplement 3): S16-S24.**
- 12. Carlsson CA, Von Essen C, Lofgren J: Factors affecting the clinical course of patients with severe head injuries. 1. Influence of biological factors. 2. Significance of posttraumatic coma. *J Neurosurg* 29:242–251, 1968.**
- 13. Chantal W. P. M. Hukkelhoven et al Patient age and outcome following severe traumatic brain injury: an analysis of 5600 patients *J Neurosurg* 99:666–673, 2003**
- 14. Chantal W. P. M. Hukkelhoven, M.SC., Ewout W. Steyerberg, PH.D., Anneke J. J. Rampen, M.SC., Elana Farace, PH.D., J. Dik F. Habbema, PH.D., Lawrence F. Marshall, M.D., Gordon D. Murray, PH.D., and Andrew I. R. Maas, M.D., PH.D. Patient age and outcome following severe traumatic brain injury.**
- 15. Chesnut RM, Gautille T, Blunt BA, Klauber MR, Marshall LE. The localizing value of asymmetry in pupillary size in severe head injury: relation to lesion type and location. *Neurosurgery* 1994; 34: 840–5.**

16. Chesnut RM, Ghajar J, Maas AR: Guideline for the management and prognosis of severe traumatic brain injury part II: Early indicators for prognosis in severe traumatic brain injury. *J Neurotrauma* 17:556-627, 2000
17. Crash Trial Collaborators. Effect of intravenous corticosteroids on death within 14 days in 10008 adults with clinically severe head injury (MRC CRASH Trial): randomised placebo controlled trial. *Lancet* 2004; 364: 1321–8.)
18. Dennis G. Vollmer , James C.Torner, Howard M.Eisenberg et .al. Age and outcome following traumatic coma: why do older patients fare worse? *J.Neurosurg.* 1991 75s; 37-48.
19. Derek A. Bruce, Eric R. Trumble, and James Steers Pathophysiology and Treatment of Severe Head Injuries in Children.
20. Eileen M Bulgar, MD; Avery B. Nathens, MD, PhD, MPH; Frederick P. Rivara, MD, MPH; Maria Moore, MPH; Ellen J. MacKenzie, PhD; Gregory J. Jurkovich, MD. Management of severe head injury: Institutional variations in care and effect on outcome. *Crit Care Med* 2002 Vol. 30, No 8
21. Evelyn Teasdale, Erico Cardoso* Samuel Galbraith, Graham Teasdale CT scan in severe diffuse head injury: physiological and clinical correlations *Journal of Neurology, Neurosurgery, and Psychiatry* 1984;47:600-603.
22. Farin A, Deutsch R, Biegon A, et al: Sex-related differences in patients with severe head injury: greater susceptibility to brain swelling in female patients 50 years of age and younger. *J Neurosurg* 98:32–36, January, 2003
23. Fearnside MC, Cook RJ et al The Westmead head injury project outcome in severe head injury. *Br.J. Neurosurgery* 7:267-279, 1993.
24. Foulkes M, Eisenberg H, Jane J, Marmarou A, Marshall L et al. The Traumatic Coma Data Bank: design, methods and baseline characteristics. *J Neurosurg* 1991; 75: S8-14.).
25. Galbraith S, Teasdale G. Predictitng the need for operation in the patient with an occult traumatic inter-cerebral haematoma. *J Neurosurg* 1981; 55: 75-81.
26. Gennarelli TA, Spielman GM, Langfitt TW, et al.: Influence of the type of intracranial lesion on outcome from severe head injury. *J Neurosurg* 56:26-32, 1982.
27. Gilchrist J, Thomas K, Wald M, Langlois J. Nonfatal traumatic brain injuries from sports and recreation activities – United States 2001–2005. *Morbidity and Mortality Weekly Report* 2007; 56:733–7.
28. Gómez PA, Lobato RD, Boto GR, et al: Age and outcome after severe head injury. *Acta Neurochir* 142:373–381, 2000
29. Gutman MB, Moulton RJ, Sullivan I, Hotz G, Tucker WS, Muller PJ Risk factors predicting operable intracranial haematomas in head injury *J Neurosurg.* 1992 Jul;77(1):9-14
30. Gutterman P, Shenkin HA. Prognostic features in recovery from traumatic decerebration. *Journal of Neurosurg* 32:330-335,1970.
31. *Head Injury A Multidisciplinary Approach*, Cambridge University Press 2009chapter 1, epidemiology of head injury p. 5.

32. Hernesniemi J. Outcome following head injuries in the aged. *Neurochir* 49:67-79, 1979.
33. James S. Heiden, Richard Small, William Caton, Martin Weiss, and Theodore Kurze. Severe Head Injury, Clinical Assessment and Outcome.
34. Jennett B, Teasdale G, Braakman R, et al.: Prognosis of patients with severe head injury. *Neurosurg* 4:283-289, 1979.
35. Jennett B, Murray L, Adams JH, et al.(1991) Causes of fatal childhood accidents. *BMJ* 302:237.
36. Jennett B. Teasdale G. Early assessment of the head injured patient. In: Jennett B, Teasdale G, eds. *Management of head injuries*, 3rd ed. Philadelphia: FA Davis, 1981;99 Philadelphia: WB Saunders, 1989;23-66. Jennett B, Teasdale G et al.: Predicting outcome in individual patients after severe head injury. *Lancet* 1:1031-1034, 1976.
37. Jess F. Kraus, M.P.H., Ph.D., Corinne Peek-Asa, M.P.H., Ph.D., and David McArthur, M.P.H., Ph.D. The Independent Effect of Gender on Outcomes Following Traumatic Brain Injury.
38. Kaufman. MA, Buchmenn. B et al Severe head injury: should expect outcome influence resuscitation and first day decisions. *Resuscitation* 23:199-206, 1992.
39. Kraus JF, Black MA, Hessol N, et al. (1984) The incidence of acute brain injury and serious impairment in a defined population. *Am J Epidemiol* 119:186–201.
40. Langlois J, Rutland-Brown W, Thomas K, *Brain Injury in the United States: Emergency Department Visits, Hospitalizations, and Deaths*, Centers for Disease Control and Prevention, national Center for Injury Prevention and Control. 2006.
41. Langlois J, Rutland-Brown W, Thomas K. *Traumatic brain injury in the United States: emergency department visits, hospitalizations, and deaths*. Center for Disease Control and Prevention, National Center for Injury Prevention and Control. Atlanta, Georgia, 2004.
42. Lanzino G, Kassell NF, Germanson TP, et al: Age and outcome after aneurysmal subarachnoid hemorrhage: why do older patients fare worse? *J Neurosurg* 85:410–418, 1996
43. Lawrence F. Marshall, M.D., Theresa Gautille, B.S.N., and Melville R. Klauber, M.D. The outcome of severe closed head injury.
44. Lenzlinger PM, Saatman KE, Raghupathi R, et al. Overview of basic mechanisms underlying neuropathological consequences of head trauma. In: *Head Trauma – Basic, Pre-clinical, and Clinical Directions*. New York. 2001: 3-36.
45. Lobato RD, Cordobes F, Rivas JJ, et al.: Outcome from severe head injury related to the type of intracranial lesion: a computerized tomography study. *J Neurosurg* 59:762-774, 1983.
46. Marshall LF, Gautille T, Klauber M, et al.: The outcome of severe closed head injury. *J Neurosurg (Suppl)* 75:28-36, 1991.
47. Marshall LF, Marshall SB, Klauber MR, et al. A new classification of head injury based on computerized tomography. *J Neurosurg* 1991;75(Suppl): S 14-S20.

48. Marshall LF, Marshall SB. Outcome prediction in severe head injury. In, Wilkins RH, Rengachary SS (eds). Neurosurgery 2nd edition. Mc Graw-Hill 1996;2611-21.
49. Marshall LF: Head injury: recent past, present and future. Neurosurgery 47:546–561, 2000.
50. Mendelow A. D. The effect of age on outcome following severe head injury for patients in coma from the outset: A mitochondrial DNA degradation phenomenon? Indian Journal of Neurotrauma (IJN9T5) 2006, Vol. 3, 95-98.
51. Miller JD, Butterworth JF, Gudeman SK, et al.: Further experience in the management of severe head injury. J Neurosurg 54:289-299, 1981.
52. Michaud LJ, Rivara FP, Grandy MPH, Reay DT. Predictors of survival and severity of disability after severe brain injury in children[clinical study]. Neurosurg 31(2):254-264, 1992.
53. Murray G, Teasdale G, Braakman R, Cohadon F, Dearden M et al. The European Brain Injury Consortium Survey of Head Injuries. Acta Neurochir 1999; 141: 223–36.).
54. Narayan RK, Greenberg RP, Miller JD, et al.: Improved confidence of outcome prediction in severe head injury in the clinical examination, multimodality evoked potentials, CT scanning, and intracranial pressure. J Neurosurg 54:751-762, 1981.
55. Nell V, Ormond-Brown S, Epidemiology of traumatic brain injury in Johannesburg– II Morbidity, Mortality and Etiology, Social Science Medicine, Vol 33, No 3, pp 289 – 296, 1991
56. Parslow R, Morris K, Tasker R, Forsyth R, Hawley C. Epidemiology of traumatic brain Chapter 1 Epidemiology injury in children receiving intensive care in the UK. Arch Dis Child 2005; 90: 1182–7.
57. Pentland B, Jones PA, Roy CW, et al.:Head injury in the elderly. Age and Ageing 15:193-202,1986.
58. Peter C. Whitfield, Elfyn O. Thomas, Fiona Summers, Maggie Whyte, Peter J. Hutchinson. Head Injury. A Multidisciplinary Approach. © Cambridge University Press 2009; 1:7.
59. Peterson DA: Stem cells in brain plasticity and repair. Curr Opin Pharmacol 2:34–42, 2002.
60. Randall M. Chesnut. Jamshid Ghajar, Andrew I.R. Maas, Donald W. Marion, Franco Servadei, Graham M. Teasdale, Andreas Unterberg, Hans von Holst, Beverly C. Walters. Early indicators of prognosis in severe traumatic brain injury.
61. S Hanif, O Abodunde, Z Ali, C Pidgeon. Age Related Outcome in Acute Subdural Haematoma Following Traumatic Head Injury Department of Neurosurgery, Beaumont Hospital, Beaumont, Dublin Department of General Surgery, Wexford General Hospital, Wexford
62. Servadei F. Prognostic factors in severely head injured adult patients with epidural haematomas. Acta Neurochir (Wien) 1997; 139:273-8.
63. Servadei F. Prognostic factors in severely head injured adult patients with acute subdural hamatomas. Acta Neurochir (Wien) 1997:139: 279-85.
64. Schneider M, Claassens M, Kimmie Z, Morgan R, Sigamoney N, Roberts A & McLaren P. We also Count. The extent of moderate and severe reported disability and the nature of the

disability experience in South Africa. Community Agency for Social Enquiry Research for the Department of Health,1999.

65. Shrestha A, Joshi R M, Thapa A, Devkota UP, Gongal DN. Outcome of Traumatic Brain Injury in Head Injury Patients Undergoing Surgical Management: A Tertiary Level Neuro-centre Experience. Kathmandu Univ Med J 2011;33(64):283-5.

66. Signorini DF, Andrews PJD, Jones PA, Wardlaw JM, Miller JD. Predicting survival using simple clinical variables: a case study .

67. Sultanah Aminah, Johor Bahru, B S Liew, MS, S A Johari, Dip. Neurosurgery, A W Nasser, MS, J Abdullah, PhD. Severe Traumatic Brain Injury: Outcome in Patients with Diffuse Axonal Injury Managed Conservatively in Hospital.

68. Tagliaferri F, Compagnone C, Korsic M et al. A systematic review of brain injury epidemiology in Europe. Acta Neurochir (Wien) 2006; 148: 255–68.

69. Thornhill S, Teasdale GM, Murray GD, McEwen J, Roy CW, Penny KI. Disability in young people and adults one year after head injury: prospective cohort study. Br Med J 2000; 320: 1631–5.

70. Vollmer DG, Torner JC, Jane JA, et al: Age and outcome following traumatic coma: why do older patients fare worse? J Neurosurg 75 (Suppl):S37–S49, 1991

71. Wang HE, Peitzman AB et al Out of Hospital endotracheal intubation and outcome after traumatic brain injury Ann Emerg Med Nov 2004;44(5):439-50

7. Appendices:

Appendix A – Glasgow Coma Scale

15 is normal, 13-14 is associated with mild head injury, 8-12 is associated with moderate head injury, <8 is associated with severe head injury

	Adult	1-5 years	0-1 year
Eye Opening			
4	Spontaneously	spontaneously	spontaneously
3	to command	to command	to shout
2	to pain	to pain	to pain
1	no response	no response	no response
Best Verbal Response			
5	Oriented	appropriate words,phrases	coos, babbles, smiles
4	confused words	inappropriate words	cries
3	inappropriate words	cries, screams	inappropriate cries
2	Incomprehensible	grunts	grunts
1	no response	no response	no response
Best Motor Response			
6	obeys commands	spontaneous	spontaneous
5	localizes pain	localizes pain	localizes pain
4	withdraws from pain	flexion withdrawal	flexion withdrawal
3	abnormal flexion	abnormal flexion	abnormal flexion
2	Extension	extension	extension
1	no response	no response	no response

Appendix B – Data Sheet

1. No :
2. Hospital Number
3. Age : yrs
4. Sex : M/F
5. Date of Admission :
6. Date of Operation
7. Date of Discharge
8. Mechanism of Injury
9. GCS on admission: [3-12]
10. Pupils on admission:
11. CT scan finding:
12. Raised ICP signs : [01 : Yes; 02 : No]
13. Treatment modality: [01 : Surgery; 02 : Conservative]
14. Outcome -- [GOS 1-5]

Data No. 08
 01: MVA (motor vehicle accident)
 02:PVA (pedestrian vehicle accident)
 03: MBA (motor bike accident)
 04: Assault
 05: Fire arms
 06: Fall from a height
 07: Heavy Objects falling on head
 08: Train accident

Data No 10
 01: Equal and reactive
 02: One side dilated
 03: Both dilated

Data No 11
 01: Linear skull fracture
 02: Depressed skull fracture
 03: EDH (extradural haematoma)
 04: SDH (subdural haematoma)
 05: SAH (subarachnoid haematoma)
 06: ICH (intracerebral haematoma)
 07 : IVH (intraventricular haematoma)

Appendix C – Glasgow Outcome Scale

Score Description

- 1 DEATH
- 2 PERSISTENT VEGETATIVE STATE
Patient exhibits no obvious cortical function.
- 3 SEVERE DISABILITY
(Conscious but disabled). Patient depends upon others for daily support due to mental or physical disability or both.
- 4 MODERATE DISABILITY
(Disabled but independent). Patient is independent as far as daily life is concerned. The disabilities found include varying degrees of dysphasia, hemiparesis, or ataxia, as well as intellectual and memory deficits and personality changes.
- 5 GOOD RECOVERY
Resumption of normal activities even though there may be minor neurological or psychological deficits.

Appendix D – Ethics Clearance Certificate



UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

R14/49 Dr Antony Thomas

CLEARANCE CERTIFICATE

M1211104

PROJECT

Retrospective Study of Moderate to Severe
Acute Head Injury in Chris Hani Baragwanath
Academic Hospital

INVESTIGATORS

Dr Antony Thomas.

DEPARTMENT

Neurological Surgery

DATE CONSIDERED

30/11/2012

DECISION OF THE COMMITTEE*

Approved unconditionally

Unless otherwise specified this ethical clearance is valid for 5 years and may be renewed upon application.

DATE

30/11/2012

CHAIRPERSON


(Professor PE Cleaton-Jones)

*Guidelines for written 'informed consent' attached where applicable

cc: Supervisor : Dr Babu George

DECLARATION OF INVESTIGATOR(S)

To be completed in duplicate and **ONE COPY** returned to the Secretary at Room 10004, 10th Floor, Senate House, University.

I/We fully understand the conditions under which I am/we are authorized to carry out the abovementioned research and I/we guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/we undertake to resubmit the protocol to the Committee. **I agree to a completion of a yearly progress report.**

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES..

8. Abbreviations:

CRASH - Corticosteroid Randomisation After Significant Head Injury

CT - Computed Tomography

DAI - Diffuse Axonal Injury

EBIC - European Brain Injury Consortium

EDH - Extradural Haematoma

GCS - Glasgow Coma Scale

GOS - Glasgow Outcome Score

ICH - Intracranial Hemorrhage

ICP - Intracranial Pressure

IVH - Intraventricular Hemorrhage

MBA - Motor Bike Accident

MVA - Motor Vehicle Accident

PFH - Posterior Fossa Haematoma

PVA - Pedestrian Vehicle Accident

RTA - Road Traffic Accident

SDH - Subdural Haematoma

TBI - Traumatic Brain Injury

TCDB - Traumatic Coma Data Bank

TSAH - Traumatic Subarachnoid Hemorrhage

UK - United Kingdom

U.S.A - United States of America