CHAPTER 1

1.0 **Introduction**

Traumatic brain injuries (TBI) are common condition presenting to the emergency department. They are associated with high morbidity and mortality especially with increasing severity at presentation. [1-4] They are a health burden as they have an impact on health resources, quality of life and finances. TBI is responsible for an estimated 9759 deaths per annum in South Africa (SA).[1] Generally it is estimated that 500 000 traffic accident occurring annually in SA result in severe injuries and death.[5] Firearms, pedestrian motor vehicle accidents and sharp force injuries are the top 3 out of the top ten causes of unnatural causes of death.[2]

Objects such as guns, knives, axes and others are used to cause head injuries either as intentional or unintentional harm. Suicide accounts for 0.2% of death while assault and undetermined intent accounts for 5.7% and 23% respectively.[1] Road traffic accidents cause 8% of TBI deaths.[1] Disasters such as collapse of buildings and fall from heights are part of mechanisms that account for 52% of head injury deaths.[1]

Patient outcome following an injury is dependent on timing and properly delivered definitive care. The teaching by advanced trauma life support advocates for the trauma patient to be transferred to the closest appropriate hospital preferably a verified trauma center. Tertiary hospitals meet the criteria of a trauma centre (advanced trauma life support trained doctors in an emergency department, in-house 24 hours neurosurgical services, available computerized tomography scans and angiograms) and are able to offer optimal care to a severely head injured patient. But unfortunately due to scarcity of resources, there are not

enough tertiary hospitals in South Africa and they are not close to most communities.[6] There is a high load of trauma and very few appropriate institutions are able to manage. Therefore, head injured patients are attended to at regional hospitals.

Regional hospitals do not have neurosurgical expertise. The setup allows stabilization, basic investigation, telephonic neurosurgical consultation and inter-hospital transfer. CT scan availability varies from one facility to the next. Those hospitals without CT scan may need to transfer the patient twice, first to another regional hospital for scanning then referral to a tertiary institution. Patients are triaged prior to transfer and classification of head injury is taken into account.

1.1 **Clinical features of head injury**

The diagnosis of head injury is preceded by the history of actual or suspected trauma (e.g. alcoholic, collapse from arrhythmias or as a result of underlying medical condition). In acute setting the most common symptoms are headache, dizziness, nausea, vomiting, loss of consciousness, hypersomnia, seizures, memory loss, ataxia and so forth. TBI patients may also present with loss of motor function (hemiparesis, monoparesis and hemiplegia) and cranial nerve injuries (anosmia, blurred vision, opthalmoplegia, unilaterally dilated pupil, tinnitus, hearing loss, aphasia, slurred speech, dysphagia and facial palsy). A combination of signs may assist to locate the affected part of the brain. Signs such as combativeness, optic nerve injuries and abnormal pupil reaction may suggest frontal lobe injuries.[4] Furthermore, pupillary signs may be seen in direct eye trauma, third nerve palsy, midbrain and pontine dysfunction.[4]

Identifying clinical features of TBI serves as an initial step towards patient classification and management.

1.2 **Classification of head injury**

Classification of head injuries can assist to predict the likely outcome, guide the decision around appropriateness of investigations and treatments. Mechanism, severity and morphological classification are used concurrently in most patients. None of the classification types are superior to the others and individually contribute vital information that influences treatment options.

1.2.1 *Classification of head injury by mechanism*

Blunt: Head injury associated with road accidents (motor vehicle, motor cyclist, motorbike and pedestrian motor vehicle accidents), falls from height and assault.[7]

Penetrating: Head injuries associated with gunshot wounds and other penetrating objects (blunt/ sharp).[7]

1.2.2 *Classification of head injury by severity (Glasgow coma scale - GCS)*

A. Mild: GCS 13-15. They are further categorized into low, moderate and high risk.[8]

- Low risk – asymptomatic individual without history of loss of consciousness (LOC).[7]
- Moderate risk – GCS 15 with either LOC, amnesia, vomiting or diffuse headache.[7]

- ❖ High risk – GCS of 13-15, with a high risk history (LOC, amnesia, anticoagulation etc) and/or high risk examination findings (skull fracture, neurologic deficit, etc).[7]

B. Moderate: GCS 9-12 the patient may be confused and able to follow commands.[8]

C. Severe: GCS 3-8 the patient remains in a coma following initial resuscitation.[3]

The European Federation of Neurosurgeons has added category on mild TBI with no risk factors and a critical TBI defined by GCS 3-4, non-reactive pupils plus motor scale less than two.[4]

1.2.3 *Classification of head injuries by morphology*

Skull fractures are divided anatomically into cranial vault and basilar. Fractures are further described by shape (linear, stellate etc) and behaviour (depressed, open/ closed).[7,10] In patients with basal skull fractures, specific information around the impact on facial nerve (palsy) and presence of cerebrospinal fluid (CSF) leak need to be included.

Intracranial lesions: are described as focal and diffuse[7]

- ❖ Focal lesions are the haematomas e.g. epidural, subdural and intracerebral.
- ❖ Diffuse lesions are the concussions and diffuse axonal injuries.

1.3 **Overview of management of head injuries**

The blunt and penetrating mechanisms are responsible for primary head injury at the time of impact. Pathophysiological events at cellular level following TBI are progressive and may have delayed presentation.[3] Haemorrhage from scalp or intracranial negatively affects

intravascular pressure and volume. Brain ischaemia develops from decreased cerebral perfusion caused by raised intracranial pressure. Other factors contributing to hypoxia are decreased level of consciousness, aspiration of gastric contents, neurogenic pulmonary oedema, disseminated intravascular coagulopathy, systemic hypotension or a combination of factors.[3] Syndrome of inappropriate antidiuretic hormone secretion and diabetes insipidus can occur in moderate to severe head injuries causing electrolyte disturbances.[11] Hypothermia, hyperglyceamia and hypocapnia are also risk factors to poor prognosis.[4] Therefore resuscitative measures such as oxygenation, ventilation techniques, temperature control and choice of fluids are aimed at reducing secondary injury. Prophylactic antibiotics reduce rates of pneumonia, local wound sepsis, brain abscess and septicaemia. An episode of hypotension at any point of treatment increases morbidity and mortality. Therefore early correction of hypotension and achieving haemostasis is vital.

A superior fluid of resuscitation has not been identified yet. Different types of intravenous fluid used for resuscitation have their advantages and disadvantages. Normal saline, Ringers lactate, dextrose water, hypertonic saline and colloids are commonly available intravenous fluids. In the absence of hypoglycaemia in a TBI patient, dextrose containing fluids should be avoided as they may cause anaerobic glycolysis and lactic acidosis.[12] Normal saline has a higher osmolality as compared to Ringers lactate and therefore is the preferred crystalloid. Normal saline and Ringers lactate apart from being good volume expanders; they also distribute themselves to extracellular space and risk worsening the cerebral oedema in a patient with a hypo-osmolar state.[12] Hypertonic saline is associated with reduction in acquired respiratory distress and some protection against bacterial illness.[13] The disadvantage of using hypertonic

saline is its severe adverse events such as renal failure, coagulopathy, hyperkalaemia, pulmonary oedema and central positive myelinolysis.[13] Colloids (e.g. albumin) are associated with a higher mortality. A follow-up double blind comparison SAFE (Saline versus albumin fluid evaluation) study on patients with traumatic brain injuries was done and found that patients who received albumin (33.2%, 71/214) had higher mortality when compared to saline (20.4%, 42/206) group.[14]

Secondary survey involves full neurological assessment using Glasgow coma scale (Table 1.1) and identifying concomitant injuries. GCS score is the most widely used parameter for grading the clinical severity. Its full application is disputable in patients with severe facial trauma, sedation, aphasia and those intubated at the scene of accident. The lowest score for eye, verbal or best motor is one not zero but under those scenarios where one parameter cannot be evaluated modification of the total score is done. The findings of the European brain injury consortium (EBIC) survey confirms that full GCS is not testable in all patients, only 56% of their patients with initial GCS<12 on admission to neurological units could be tested.[4] Nevertheless, efforts should be made to calculate GCS as management strategies (timing of initial/ repeat CT scan, surgical evacuation of heamatoma) depend on it.

Table 1.1 Glasgow Coma Scale

Behaviour	Response	Score
Eyes open	Spontaneous	4
	To speech	3
	To pain	2
	No response	1
Best verbal response	Alert and Orientated	5
	Disorientated conversation	4
	Speaking but nonsensical	3
	Moans or unintelligible sounds	2
	No response	1
Best motor response	Obeys commands	6
	Localizes pain	5
	Withdrawal to pain	4
	Flexion (abnormal) to pain	3
	Extension to pain	2
	No response	1

Source: Moppet IK. Traumatic brain injury: assessment, resuscitation and early management. Br J Anaesth 2007;99:18-21

Resuscitation of TBI patients with Glascow Coma score less than five is controversial[16-19]. Some authors categorize TBI patients with GCS < 5 as unsalvageable and scarce resources should be reserved for those with a chance of survival. In absence of shock, patients with little central nervous system (CNS) function are unlikely to benefit from craniotomy. However, other specialist in the field of neurosurgery advocate that severe TBI require aggressive medical and surgical management including emergency craniotomy for traumatic intracranial lesions regardless of GCS score.[17,19]

Alcohol intoxication influences the level of consciousness and may create a dilemma in triaging patients for aggressive resuscitation. A study of 412 severe TBI patients demonstrated

that a number of patients with a GCS of 8 or less did not have a head injury but rather intoxication as the cause of their altered mental status .[20] In contrast, a study of 108 929 head injured patients found that alcohol intoxication did not result in significant changes in GCS in patients with moderate or severe TBI.[21] Therefore decisions to intubate, ventilate and assess the patient for their need for craniotomy should not be delayed by waiting for alcohol to wear off. CT scan guidelines[22-24] stipulates that alcohol intoxication is a risk factor and an appropriate indication for radiological investigation. CT scan is the preferred diagnostic tool for identifying intracranial pathology. There are numerous guidelines on when to perform CT scan of the head (Table 1.2). In SA the most popular and applied guidelines are Canadian CT Head Rule (CCHR).

The New Orleans criteria (NOC) and Canadian CT Head Rule (CCHR) are designed to guide the use of CT scan in a minor head injured patient who could potentially have a life threatening intracranial lesion. Smit et al[22] found that both decisions rules were 100% sensitive in identifying patients likely to undergo neurosurgical interventions and both have minimum of 83% sensitivity in identifying patients with intracranial traumatic findings on CT scan. CCHR had higher specificity for intracranial traumatic CT findings when compared with NOC.[22] GCS < 12, presence of skull fracture, signs of basal skull fracture and age above 65 years are strong predictors of intracranial pathology (extrapolated from CCHR) and this easily accommodates patients with moderate to severe TBI. The same risk factors are applicable in paediatric population.[25]

Table 1.2 CT scan guidelines

National Institute for Clinical Excellence (Nice)[16]	Canadian CT Head Rule (CCHR) [22,23]	New Orleans Criteria (NOC)[22]	Neurotraumatology[24]
1. GCS <13 at any time since injury. 2. GCS =13 or 14 two hours post injury. 3. Suspected open or depressed skull fracture. 4. Any sign of basal skull fracture 5. Post traumatic seizures. 6. Focal neurological deficit. 7. More than 1 episode of vomiting. 8. Amnesia of >30 minutes before the event. 9. Coagulopathy(history of bleeding, clotting disorder 10. *Dangerous mechanism of injury 11. Age ≥ 65 years (Where evidence of LOC or amnesia experienced)	1. GCS score <15 at 2 hours after injury. 2. Suspected open or depressed skull fracture. 3. Any sign of basal skull fracture. 4. Vomiting equal to or more than 2 episodes. 5. Age ≥ 65 years (Medium risk) 6. Amnesia before impact ≥30 minutes. 7. *Dangerous mechanism.	1. Headache 2. Vomiting 3. Seizures 4. Intoxication 5. Short-term memory loss 6. Age more than 60 years 7. Injury above the clavicles	1. GCS core more than 14. 2. Suspected skull fracture. 3. Neurologic deficit. 4. Vomiting 5. Amnesia 6. LOC. 7. Headache 8. History of seizures. 9. Previous neurosurgery. 10. Alcohol or substance abuse. 11. Coagulopathy.

*Dangerous mechanism of injury- pedestrian struck by motor vehicle, occupant ejected from a motor vehicle and/or fall from elevation ≥3 feet or 5 stairs.

Raised intracranial pressure (ICP) is a predictor of intracranial pathology and is associated with mortality. Normal ICP is 6-15 mmHg. Those above 20 mmHg are associated with poor outcome thus need for therapeutic interventions, while an ICP of 40-45 mmHg are associated with brain herniation.[26,27] Iatrogenic rise in ICP may occur during laryngoscopy and airway manipulation in a poorly sedated patient. As a result use of rapid intubation with administration of

pretreatment such as lidocaine and fentanyl aims to curb the above effects.[15,28] Sedatives and analgesia reduce pain and restlessness thus indirectly reduces intracranial pressure.[27] Consequently, seizures can lead to secondary brain insult by increasing ICP and exposing patient to hypoxia. Anticonvulsant prophylaxis reduces development of early seizures in patients with penetrating TBI, traumatic intradural pathology (subdural and epidural) and severe TBI.[15,27,29] Intracranial pressure is monitored by insertion of intracranial pressure monitoring device. Clinical findings suggestive of raised ICP are the presence of dilated pupils, hemiparesis in a comatose patient GCS <8 and progressive neurological deterioration. Combination of methods are used to decrease ICP namely nursing TBI patient at 30^0 bed elevation, intravenous fluid restriction with regard to mean arterial pressure and adequate oxygenation using blood gases analysis as an indicator. Hyperventilation reduces intracranial pressure by causing vasoconstriction. It is only necessary when PCO2 levels are above 30 mmHg in patients with raised intracranial pressure.[7] It is never advised that it be used blindly. It reduces or compromises cerebral blood flow thereby might worsen cerebral ischaemia.[26] Mannitol is the preferred osmotic diuretic to reduce raised ICP. It has been reported that it reduces blood viscosity, the diameter of cerebral arterioles and cerebral blood volume.[27]

Surgical management for raised ICP include ventriculostomy catheter or fiberoptic monitors are used to assess intracranial pressure and may also be used to decrease the high pressures by draining cerebrospinal fluid.[26,28] Emergency burr holes (trephination) are indicated for drainage of a life threatening rapidly expanding epidural haematoma in situations where rapid transfer to neurosurgeon is not possible.[7,15,29] Although it is considered a life saving procedure, when done in this settings rarely achieve the desired result (control the source of

bleeding). Precision and competency in performing the procedure is vital. Other indications for burr holes are presence of signs of brain herniation and progressive neurological deterioration.

Definitive management of a TBI patient with haematoma causing midline shift, compression of underlying structure and neurological deterioration is craniotomy. Regarding Marshall CT classification[30] (Table 1.3) class III, IV, haematomas with mass effect and presence of subarachnoid haemorrhage will require invasive surgical treatments. However type, size and position of the heamatoma are taken into account when considering surgical drainage.

Table 1.3 Marshall Classification of CT scans, modified by the European Brain Injury Consortium

Level	Classification
I	No visible intracranial pathology
II	Diffuse injury with cisterns present, 0-5mm shift and high-medium lesions <25 cc (including bones and extraneous bodies) a) A single lesion b) Two or more unilateral lesion c) Bilateral lesions
III	Diffuse injury with cisterns compressed or absent, 0-5mm shift and high-medium lesions <25 cc
IV	Diffuse injury with midline shift >5 mm and high-medium lesions <25 cc
Lesion with mass effect	Lesion with high-medium density volume >25 cc a) Extradural haematoma b) Subdural haematoma c) Intraparenchymal haematoma d) Multiple lesion
Subarachnoid haemorrhage	Present/ absent

Source: Rusticalli B, Villani R et al. Treatment of minor and severe traumatic brain injury: National reference guidelines. Minerva Anaestesiol 2008;74(10):583-616

Otherwise, asymptomatic TBI patient with an intracranial haematoma and no midline shift may require conservative management. In addition TBI patient with CT scan findings of an acute subdural haematoma, GCS score >13 since injury, absence of associated intraparenchymal haematoma, absence of basal cistern effacement, contusion or edema may be managed conservatively. Nevertheless, these decisions should be made or supervised by neurosurgeon.

Other surgical treatments include local wound care, thorough debridement and irrigation of exposed brain tissue and bony structures which reduces incidence of local infection. This can either take place in emergency department (ED) or theatre especially in patients with open or penetrating head injuries. Less urgent interventions like elevation of a depressed skull fracture may be planned for a later stage in a stable asymptomatic TBI patient.

1.4 **Literature review**

Leratong is the only regional hospital in West Rand district in Gauteng province. It has a comprehensive ED that attends to medical, paediatric, surgical and gynaecological patients. In 2009, about 70 000 patients were treated in the ED. The hospital receives mostly uninsured patients and transfers from a district hospital. Patients requiring craniotomy are transferred to Chris Hani Baragwanath Hospital after telephonic consultation with the neurosurgeon. The incidence of traumatic brain injury in Leratong hospital is unknown. Previous records of studies performed in South African (SA) regional hospital regarding the epidemiology and outcomes of treatment are not available. Studies performed in SA tertiary hospital either focus on

subsets[18,32] of TBI patients, assessing specific resuscitation procedure[33] or indirect analysis of outcomes of treatment[34] .

In a Johannesburg tertiary hospital, Ouma[32] conducted a 5 year (1992-1999) retrospective study on prognostic factors following civilian gunshot wounds to the head. The study population had 70% direct admissions and 30% transfers. Eighty six percent of males were affected and the commonest age was 21-40 years. Cause of injury was mostly assault (64%) and suicide accounted for the remaining 36%. Sixty one percent of patients arrived within the first 2 hours of injury and had GCS less than 5 on presentation. The commonest CT scan pathology was contusion and brain oedema (66%). Craniotomy was performed on 22% of patients. Highest mortality (60-100%) was observed in patients with GCS less than 7, bilaterally dilated pupils, penetrating gunshot wound to the head with multiple lobe involvement and presence of a subarachnoid haemorrhage. Age, sex, cause of shooting (intentional/ unintentional), time lapse from injury to admission and referrals from regional hospitals did not negatively influence the outcome when treatment protocols are applied appropriately. This implies that regional hospitals can manage TBI patients appropriately.

Semple et al[18] in retrospective analysis of 181 gunshots head had similar findings; GCS less than or equal to 5 was associated with 98% mortality, ventricular injury with 100%, mortality bihemispheric injury 90% and diffuse cerebral swelling 81%.

Hardcastle et al[33] did a one month review of mechanism of TBI and found that 52.6% were due to motor vehicle accident, 28.1% due to penetrating injuries and 10.5% were due to blunt trauma. They also assessed the Glasgow Coma Scale (GCS) score as a triage tool on TBI and

indications for definitive airway management; found that Tygerberg academic hospital's practices are in-keeping with evidence based medicine.

In Natal, 22 years ago Bullock et al[34] concluded that referring hospitals do not manage TBI patients appropriately. They reviewed 100 consecutive head injuries (using autopsy results) and found that 88% of patients had hypoxic neural damage which could have been minimized by early appropriate resuscitation, early identification of intracranial pathologies and timeous transfer to neurosurgical unit.

There are numerous studies conducted outside SA addressing TBI as a broader topic. Yattoo et al[10] assessed the trends of head injury in Kashmir. Road traffic accidents and falls from height were the most common mechanism of injury. Majority of patients (80%) had GCS 15 and scalp lacerations. The ages mostly affected were 0-10 and 21-30 years. The month of July had high incidence of head injury. CT scan was done on 14.1 % of patients and no abnormal findings on only 12.9%. TBI deaths were high in 31-40 and low in 71-80 year olds. In spite of the best management, 15-20% of TBI prove to be fatal.[10]

Petroni et al[35] in an Argentinean trauma centre verified that age, GCS, hypotension, computed tomography (CT) findings and pupil response are still strong predictors of outcomes in severe TBI when tested in a developing world.

In Ryder Trauma Center United states of America (USA) MacLeod et al[36] assessed trauma deaths in the first hour of injury and found that majority of deaths occurred in patients whose vitals were critical, who had severe multiple injuries or head injuries.

Esposito et al[31] examined the profile of head injured patient who actually required neurosurgical expertise and found that over 95% required non-operative management alone. They found that the average GCS for both craniotomy and survival was 9. Craniotomy was performed in 3.6% of all head injured patients which represents 1% of all trauma patients studied. Subdural haematoma occurred in 18% of head injured patients (5% of all trauma patients), with 13% of those undergoing craniotomy.[31] Epidural haematoma occurred in 10% of the head injured patients (3% of trauma patients) with 17% of those undergoing craniotomy.[31]

Yattoo et al[10] in prospective study also showed a low frequency for craniotomy; 9.5% of TBI patients required surgical treatment versus 90.5% who received conservative management. Most of their patients had scalp lacerations (40.4%), commuted skull fracture (5.9%), extra-dermal haemorrhage (3.2%), depressed fracture (3%), brain oedema (1.3%), subarachnoid bleeds (0.3%) and intracerebral haemorrhage (0.3%).

Most of the studies on TBI were undertaken under almost ideal setting, either taking place in tertiary hospital in SA [18,32-34] or in first world emergency department.[10,31,35-36] The shortcomings of the reports are their lack of relevance in a regional hospital setting. Furthermore, ATLS® guidelines on inter-hospital transfer states that the patient with depressed or open skull fracture, decreased Glasgow coma scale and presence of lateralizing signs need to be sent to a hospital with neurosurgical facilities.[7] If the indications are applied literally, tertiary hospitals will be overwhelmed with patients who solely require conservative management. The results of the study will assist to clarify criteria of transfer to neurosurgical team.

1.5 **Aims of this research**

- To determine the characteristics of TBI patients in Leratong regional hospital.
- To determine the criteria applied in requesting brain computerized tomography (CT scan).
- To determine the relationship between the outcome and clinical presentation.
- To compare the outcome of patients whose brain CT scans were done locally versus those who travelled to another facility.
- To determine the criteria used to transfer head injury patients to neurosurgeons.

CHAPTER 2

2.0 **Materials and methods**

2.1 **Study design**

This retrospective descriptive study was undertaken in Leratong regional hospital which is situated in the West Rand district of Gauteng province (South Africa). The following registers were evaluated to identify any TBI entry treated for the period of 1 January to 31 December 2009:

- Emergency department registers for priority 1 and 2 patients,
- Surgical admission ward registers for children and adults.

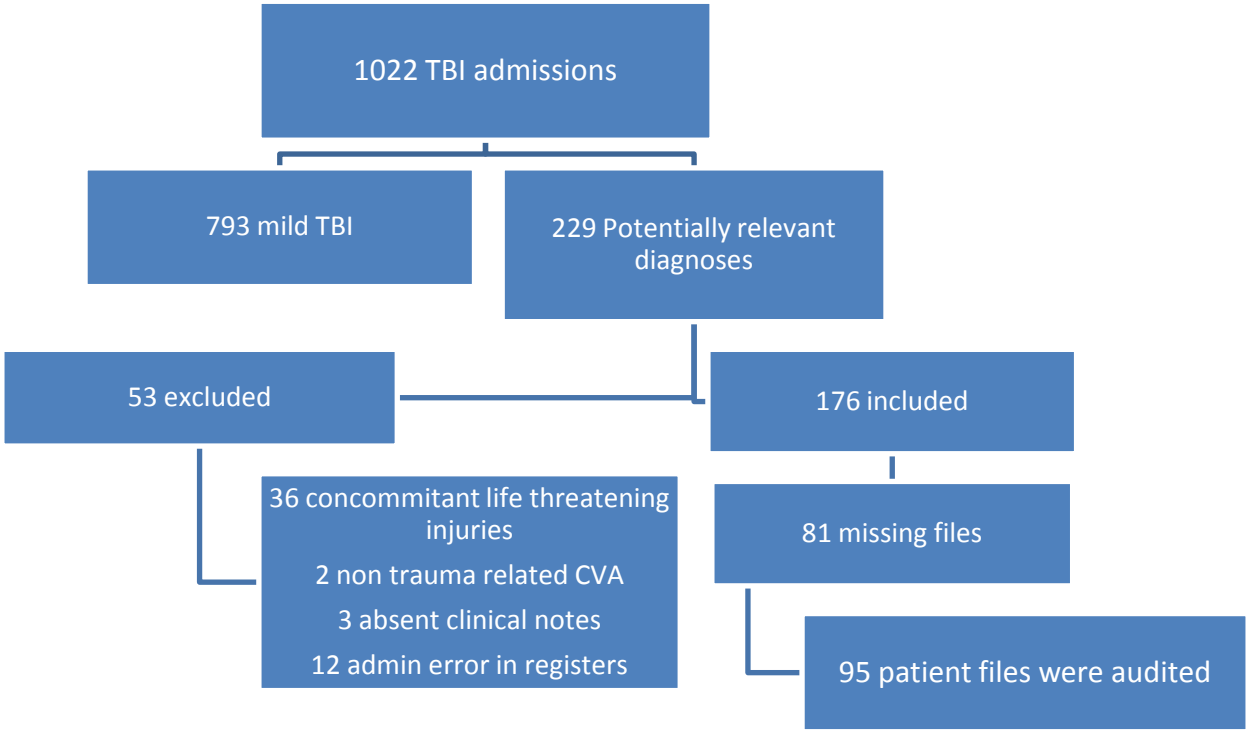
Patients with no age restriction admitted to hospital for moderate to severe TBI were included in the study excluding patients with GCS 13-15, degloving injuries, eye trauma, plastic surgery candidates and maxilla-facial injuries. Patients with concomitant life threatening injuries e.g. ruptured spleen in additional to the head injury were also excluded (Figure1.1).

2.2 **Study sample**

The total number of TBI patients admitted to hospital in the year 2009 was 1022 (figure 1.1). Seven hundred and fourteen had mild TBI. The remaining 308 records were patients with neurological deficit and cranial pathology. Seventy nine patients who had diagnosis of skull

fracture: intracranial bleeds, craniotomy and cranial nerve palsy post TBI were excluded from the study as they maintained GCS 13-15 from initial presentation to beyond 24 hours of admission.

Figure 2.1 Flow chart of study sample



Mild TBI: Glasgow coma score 13-15; TBI: Traumatic brain injury; CVA: cerebrovascular accident

Further, 38 patients were excluded as they had concurrent intra-abdominal injuries, pure maxillofacial case, multiple gunshot to other systems, cerebrovascular accidents and crush syndrome.

Absence of electronic clinical records posed challenges in tracking most patient files. Three files had absent clinical notes and 12 files were administrative errors in registers (misidentification of file number to name or name to diagnosis). Files of patients who died were easy to access as they are stored separately and there are strict control measures on their movement. Unlike other files (81 missing), here are the possible explanation why they could not be found; used by the patient in outpatient department, patient admitted, left at the pharmacy/ emergency department after recent use, finance department, departmental clinical audit, preparation for case presentation or patient left the hospital premises with own file. Therefore, 134 files excluded from the study.

Analysis was carried out on the remaining 95 patient files who met inclusion and exclusion criteria. In 10% of patients, age and mechanism of injury remained unknown for those individuals picked up on the road without personal identification or escorting relatives. While in 10.5-20.0% TBI patient the blood pressure and pulse recording was missing or not recordable due to the severity of hypovolaemic shock. Only 68% of the sample had documented pupil size and reactivity.

From the files the following information was retrieved:

- Incidence of TBI by gender
- Monthly distribution
- Mechanism of injury
- Clinical history

- Time of arrival to emergency department, time of transfer, death and admission to the ward.
- Radiological tests done for each individual patient were also recorded.
- Findings on the CT scans were reviewed based on radiologist report. In absence of copy of radiologist report, interpretation by treating senior medical practitioner was included.
- The data collection form (Appendix A) comprised information on sex, age, mechanism of injury, GCS, CT scan, Blood pressure.
- Airway management and fluid resuscitation were recorded.
- At hospital discharge outcomes were recorded as death, transfer to neurosurgeon and complications.

Subdural hygroma (CT scan finding) was defined as collection of cerebrospinal fluid in a subdural space.[37] Acute metabolic, physiologic alterations and loss of function were observed complications. Good recovery was defined by no functional loss. Patients with isolated neurological disability (apraxia, aphasia, facial palsy and confusion) were grouped together as moderate disability. Upper, lower limb palsy, prolonged coma of more than 72 hours and hemiplegia also grouped as severe disability. Patients in a vegetative state were identified to be; any neurological disabilities, feeding problems and their inability to participate in rehabilitation activities (physiotherapy, speech or occupational therapy). Consequently this information was transferred to a spread sheet (Appendix B) to ease the process of data analysis.

2.3 **Data analysis**

Statistica version 9.1 software was used to analyze the data. Incidence of TBI and patient profiles was described in terms of:

- Age and gender
- Severity (GCS)
- Day and time of arrival to the emergency department
- Monthly distribution
- Mechanism of injury
- Indications for CT scan
- CT scan pathology
- Transfers, time lapse before transfer and outcomes
- Craniotomy
- Loss of function (disability) and death

Categorical data were expressed as frequency tables, histogram, charts and/or percentages. Glasgow Coma Scale was used as a measure of severity and expressed as mean and standard deviation. Continuous variables such as age, blood pressure and pulse were also summarized as mean ± SD and tested for normality. A non-parametric test, Mann- Whitney U test was used to analyze the relationship between outcomes and clinical presentation (GCS, blood pressure, age and pulse). Pearson Chi-square was used to compare CT scan pathologies to outcomes (alive, dead, presence or absence of complications). P-values of 0.05 were accepted as significant for all measures.

2.4 **Medical treatment**

Patients with suspected TBI were initially assessed by medical practitioner in the ED and referred to the surgical team (intern, medical practitioner and specialist general surgeon). Depending on clinical condition, cardiopulmonary resuscitation, endotracheal tube placement and mechanical ventilation, as well as volume infusion for haemodynamic stabilization were performed. After the neurologic assessment was performed using Glasgow coma scale score & pupil assessment (size and reaction), 80% of patients received plain radiographs of the skull and were evaluated by CT scan within the hospital. While other 14.7 % patients were referred to neighbouring regional hospital for CT scan as this service was not available outside office hours including weekends and holidays.

Basic blood investigation included full blood count, urea, electrolytes, acid- base gas analysis, clotting profile and blood for cross matching.

Moderate and severe TBI patients were nursed in 30^0 bed elevation. Intravenous antibiotic (Rocephine®/ Augmentin®/Kefzol®) administered to patients with open skull fracture and epanutin loading was given to all severe TBI, open skull fracture and those with witnessed seizures. Analgesia was given to all patients.

Management of open wounds included dressings with antiseptic solution and primary closure of simple laceration/ incised wound under local anaesthesia. Wider, deeper or degloved head injuries were sent to theatre for debridement and repair. Tetanus toxoid was administered according to ATLS® recommendations. Patients who required craniotomy were transferred to Chris Hani Baragwanath Hospital after a telephonic consultation. After patient

has been diagnosed and treated by neurosurgical team, patients were returned to Leratong for continuing care and rehabilitation.

Those requiring conservative management were retained in Leratong hospital intensive care unit or surgical ward. Treatment recommendations such as adding mannitol, lasix, epanutin or repeating of CT scan following telephonic discussion with the neurosurgeon were adhered to.

Once stabilized, patients identified to have motor, speech deficit and chest problems received bed side rehabilitation until it was safe to be moved to physiotherapy department.

2.5 **Ethics**

Approval from Committee for Research on Human Subject (Medical) of the University of the Witwatersrand was sought prior to conduction of the study and protocol number M10512 received (Appendix C).

2.6 **Limitations**

Due to the retrospective nature of this study the following challenges were experienced during gathering of information;

- Missing registers. The impact of missing registers was minimized by utilizing multiple registers from ED, surgical wards and intensive care unit following patients care pathways.
- Small errors were observed in information written in the registers where the diagnosis in the registers did not correspond to the patients file.
- Eighty one files were missing. This is partly due to absence of electronic clinical records and storage issues experienced by most facilities still keeping manual records. As a result of 46% of missing files, data will be skewed.
- The degree of incomplete clinical notes was minimized by availability of two sets of clinical notes per patient (general practitioner in ED and another doctor from general surgery department).

CHAPTER 3

3.0 **Results**

3.1 **Characteristics of TBI patients**

Mean age of the 95 TBI patients was 28 ±13.9 years and 88.4% were male. The youngest patient was 6 days old and oldest 75 years (Table 3.1).

Table 3.1 Frequency of moderate to severe TBI by age and gender

	Gender		
Age (years)	Female n (%)	Male n (%)	Total n (%)
0-10	3 (3.2)	6 (6.3)	9 (9.5)
11-20	2 (2.1)	10 (10.5)	12 (12.6)
21-30	2 (2.1)	**36 (37.9)**	**38 (40)**
31-40	0 (0)	11 (11.6)	11 (11.6)
41-50	0 (0)	9 (9.5)	9 (9.5)
51-60	2 (2.1)	3 (3.2)	5 (5.3)
>61	1 (1.05)	1 (1.05)	2 (2.1)
Unknown adult	1 (1.05)	8 (8.4)	9 (9.5)
Total No.	11 (11.6)	**84 (88.4)**	95 (100)

The mechanism of injury was predominantly road traffic accident 47% (45/95) and assault 32.6% (31/95) (Table 3.2). The most common mechanisms of injury in children and adolescents (6 days to 21 years) were pedestrian vehicle accident 10.5% (10/95), motor vehicle accident 2% (2/95), falls 3.2% (3/95) and assault 1% (1/95). Seventy eight percent of patients with moderate to severe TBI presented to the emergency department weekday after office hours 44.2% and weekends 33.7% (Table 3.3). The highest number of patients was seen in August and November while the least was seen in April (Table 3.4).

Table 3.2 Mechanism of injury of moderate and severe TBI

Mechanism	Number of cases n (%)
Road traffic accident	**45 (47.4)**
-Motor vehicle accident	13 (13.7)
-Pedestrian vehicle accident	**31 (32.6)**
-Motor cyclist accident	1 (1.05)
Fall from height	2 (2.1)
Falling objects	2 (2.1)
Assault	**31 (32.6)**
Gunshot	10 (10.5)
Unknown	5 (5.3)
Totals	95 (100)

Table 3.3 Arrival time to the emergency department according to weekdays

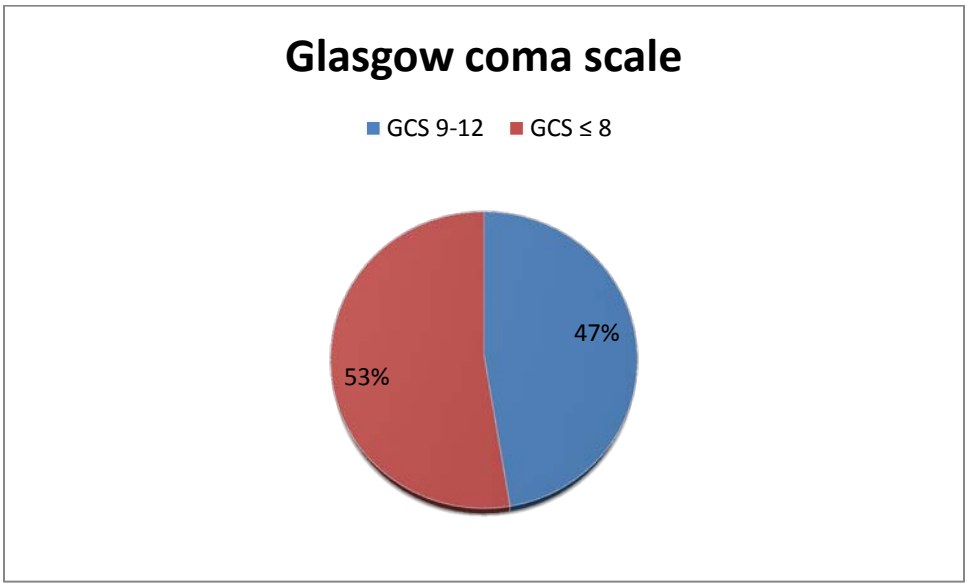
Time	Monday- Friday (day) n (%)	Monday- Friday (night) n (%)	Saturday & Sunday n (%)
08h00 - 16h00	16 (16.8%)	-	-
16h00 – 08h00	-	**42 (44.2%)**	-
24 hours	-	-	**32 (33.7%)**
Unknown time	5 (5.3%)	-	-

Table 3.4 Monthly distribution of moderate to severe TBI

Season	Number of cases n (%)
January	8 (8.4)
February	7 (7.4)
March	6 (6.3)
April	**2 (2.1)**
May	12 (12.6)
June	5 (5.3)
July	7 (7.4)
August	**14 (14.7)**
September	5 (5.3)
October	10 (10.5)
November	**14 (14.7**)
December	5 (5.3)
Total	95 (100)

Musculoskeletal injuries were seen in 55% of TBI patients. Fifty three percent of patients (50/95) had GCS ≤ 8 and 47% (45/95) had GCS of 9-12 with mean 8±3.1 (Figure 3.1).

Figure 3.1 Glasgow coma scale

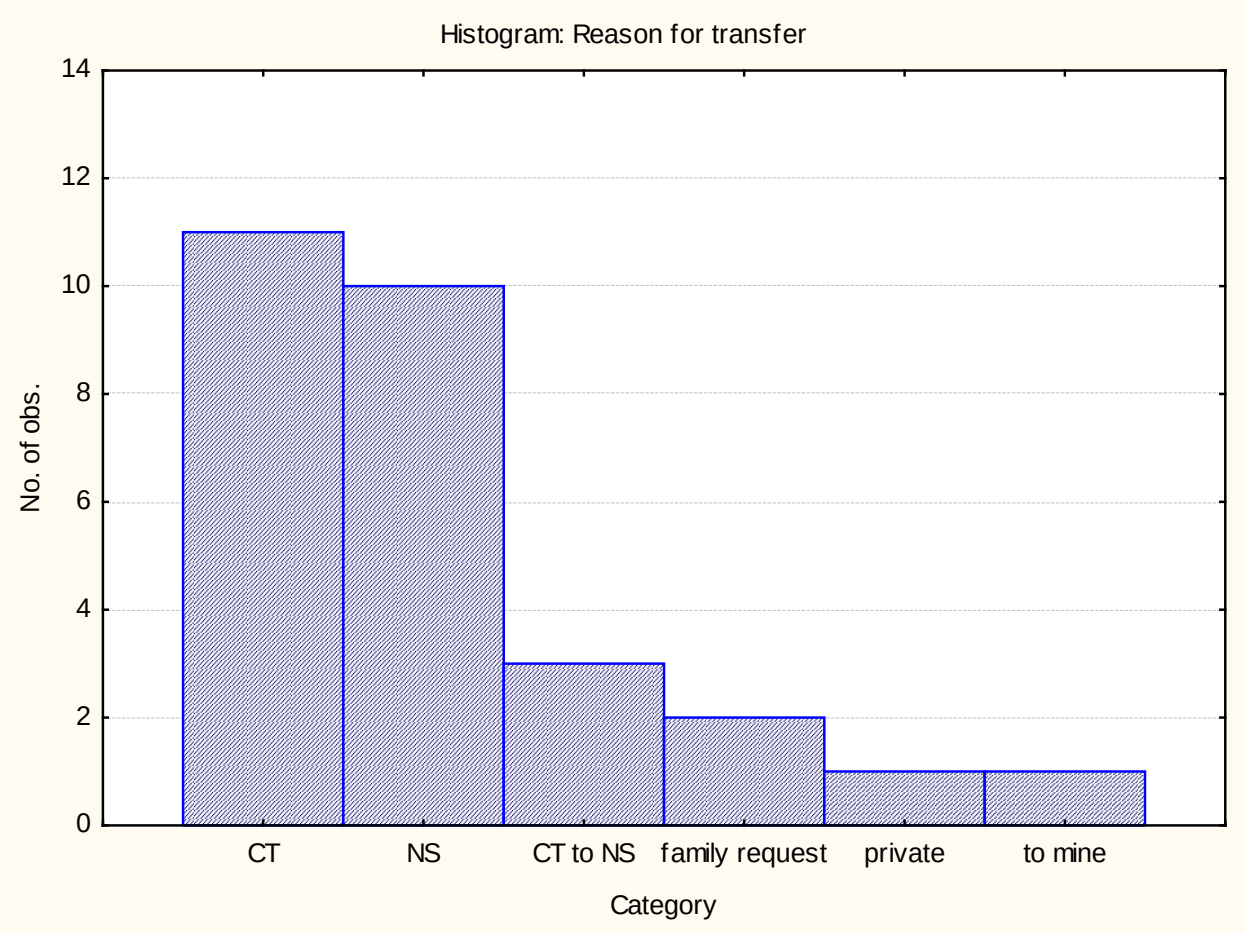


Five patients were resuscitated and intubated at the scene. Their GCS on arrival was 2/10 but verbal scores were not allocated. Of the 12(12.6%) patients with systemic hypotension (SBP <90 mmHg), one was a child and 11 (11.7%) showed systemic hypertension (SBP >140 mmHg).

3.2 **Transfers**

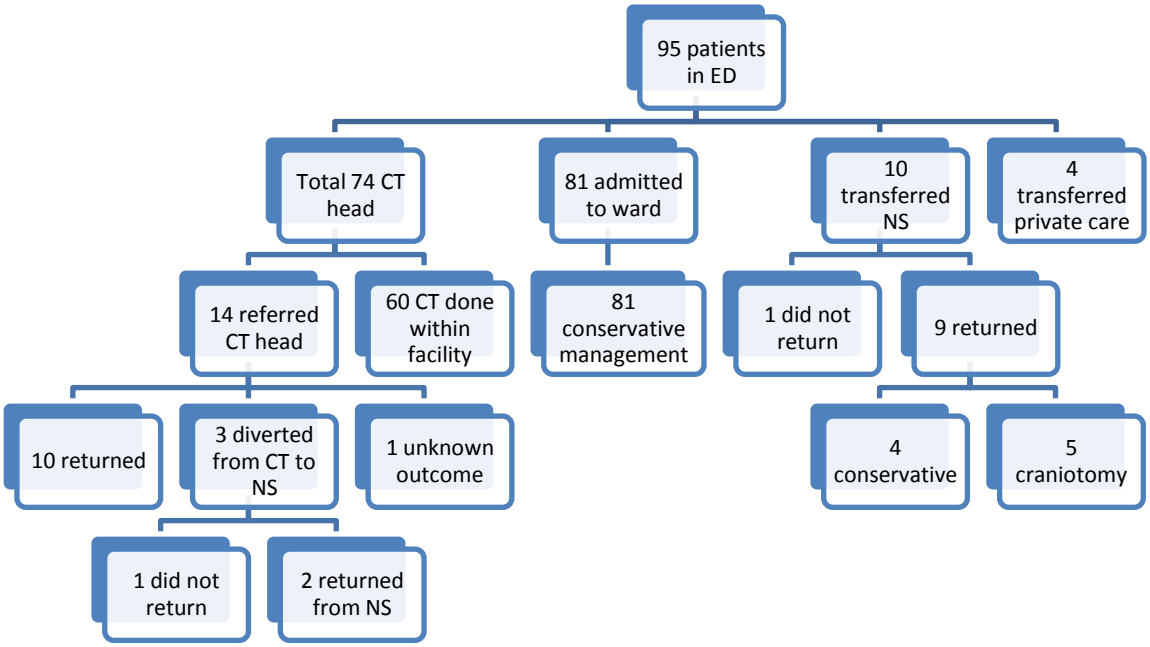
TBI patients were transferred to neighbouring hospitals for various reasons; unavailability of resources, neurosurgical consultation, second opinion with regard to interpreting CT scan film, patient and relatives preferences (choice of private care facility). Figure 3.2-to- 3.3 show the reasons and flow of patients within the system.

Figure 3.2



CT: computerized tomography scan; NS: neurosurgeon; to mine: mine hospital; private: private hospital

Figure 3.3 Pathways of care for moderate to severe TBI.



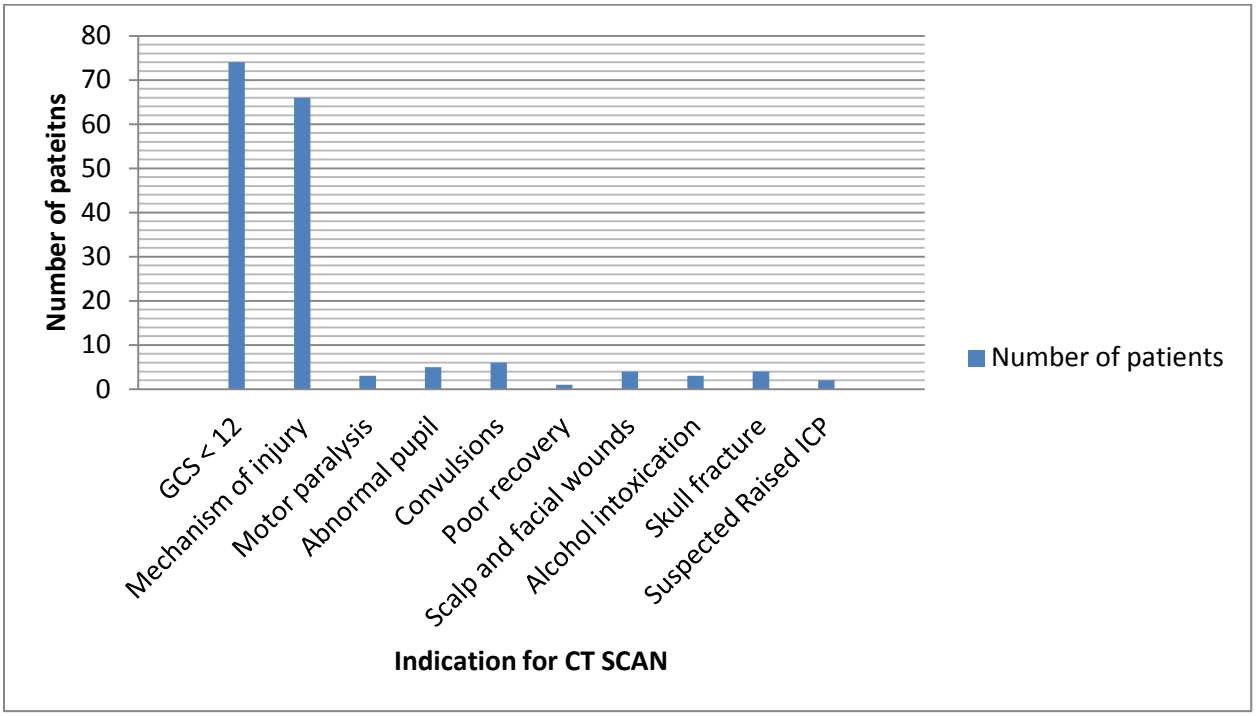
ED: emergency department; CT: computerized tomography scan; NS: neurosurgeon

3.2.1 **CT scan findings**

A total of 78% (74/95) of patients had CT scan of the head performed: 63% (60/95) of CT scans were performed within the hospital and 15% (14/95) were CT referrals to the neighbouring regional hospital (Figure 3.3). Of the 63% performed locally 7% CT scans were performed more than 24 hours after injury. In 21% (20/95) of the 95 patients with TBI, 16% (15/95) CT scan could not be obtained due to unstable status of the patient and 5% (5/95) were those with GCS 2/10.

Most of the CT scan requests were based on GCS < 12 (100%) and mechanism of injury (70%) (Figure 3.4).

Figure 3.4 Indications for CT scan



Other indications for CT scan were abnormal pupil size, presence of seizures, skull fracture, loss of consciousness, visible injuries on head, cranial nerve palsy, alcohol intoxication, suspected raised intracranial pressure and poor recovery.

Intracranial traumatic lesions on the CT scan were found in 96% of patients. Contusions (34%), skull fractures (32%) and subdural heamatomas (29%) were the commonest CT scan finding (Table 3.5). Multiple CT scans pathologies were observed in 86.5% (64/74) of patients. Patients with intraventricular haemorrhage (6%) had with associated pathologies, most importantly brainstem haemorrhage and haemorrhagic contusions in the basal ganglia. Traumatic subdural hygroma usually occur following head injury. Only 4% of patients who had CT scan after 72 hours of admission had hygromas.

Table 3.5 CT scan findings

Lesions	Number of cases n (%*)
Normal	3 (4)
Abnormal;	
Skull fracture	**24 (32)**
Facial fracture	13 (18)
Pneumocephalus	2 (3)
Subdural haematoma	**22 (29)**
Epidural haematoma	6 (6)
Subarachnoid haemorrhage	19 (26)
Intraparenchymal haemorrhage	12 (13)
Cerebral oedema	14 (15)
Contusion	**25 (34)**
Hygroma	3 (4)
Intraventricular haemorrhage	6 (6)
Raised intracranial pressure	7 (7)

*Percentages are more than 100% as patients can have multiple pathologies on CT scan.

3.2.2 **Outcomes of transferred patients**

The process of initially receiving the patient in the emergency department, resuscitation, investigation, telephonic arrangements with the hospitals and emergency medical services took an average of 6 hours (Table 3.6) with the earliest transfer at 1 hour 40 minutes and latest 10 hours.

Table 3.6 Time lapse from Emergency department arrival to departure to next hospital for CT scan and/or neurosurgery

Time between arrival to ED and departure to CT/neurosurgeon	Number of cases n (%)
< 3 hours	4 (16.7)
3-5 hours	5 (20.8)
5-8 hours	10 (41.7)
≥8 hours	5 (20.8)
Total	24 (100)

The following factors were responsible for more than 8 hours of delay: communication breakdown between facilities, unavailability of transport, ventilator in trauma unit and vacant bed in an intensive care unit (ICU) in the receiving hospital with neurosurgical expertise. The above factors influenced the decision to postpone CT scan to the next day. One patient deteriorated and died before the CT scan could be performed. Of the 5 patients (4 CT referrals and one neurosurgical) with more than 8 hours of delay to transfer; 4 patients had severe disabilities and one never returned from Chris Hani Baragwanath Hospital with an unknown outcome.

Despite that some of the transferred patients had a potentially reasonable/fair prognosis (normal blood pressure, pulse and reactive pupils); poor outcome was still experienced by CT inter-hospital referrals (Table 3.9).

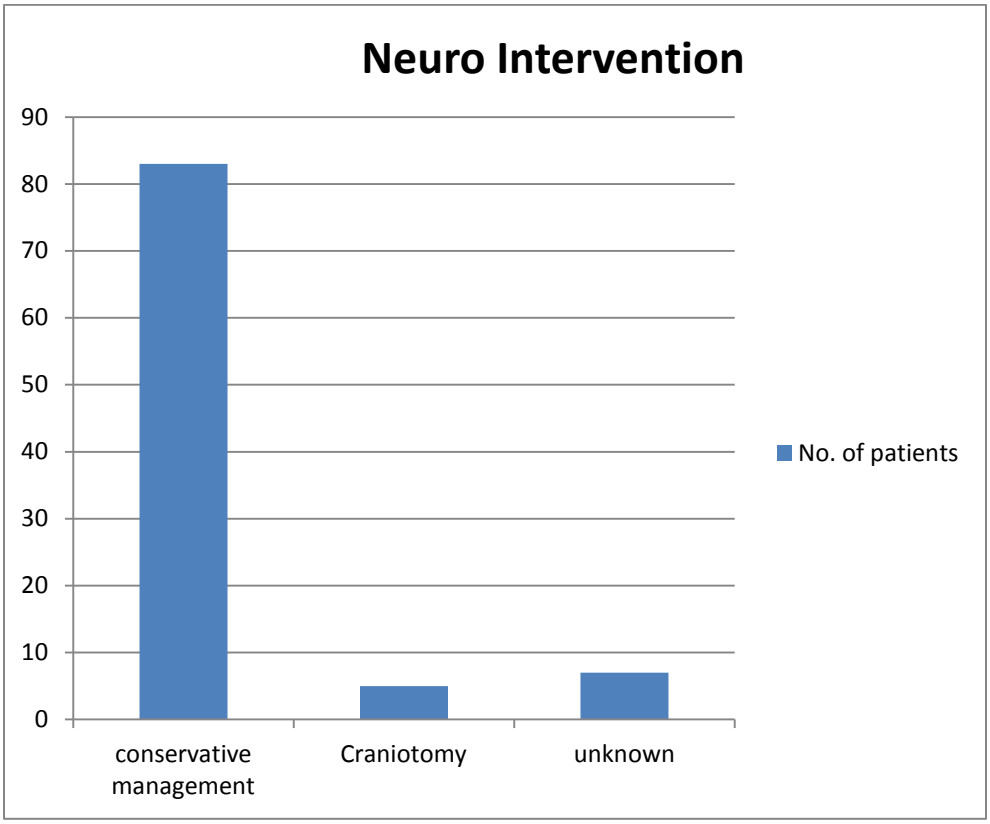
Table 3.7 Clinical presentation and outcomes of inter-hospital transfers

Clinical presentation	CT referral	Neurosurgical transfers	Totals n (%)
GCS 8-12	6	3	9(37.5)
6-7	5	6	11(45.8)
≤5	3	1	4(16.7)
Systolic BP: ≥140	6	1	7(29)
>90<140 (normal)	7	8	15(62.5)
≤90	1	1	2(8)
Pulse: ≥90	7	4	11(45.8)
>60<90 (normal)	4	5	9(37.5)
≤60	3	1	4(16.7)
Pupils:			
Unilateral dilated	5	1	6(25)
Bilateral dilated	0	1	1(4)
Constricted	1	0	1(4)
Normal	8	8	16(67)
CT finding:			*
NAD	2	1	3(12.5)
SDH	3	4	7(29)
SAH	9	1	10(42)
EDH	1	3	4(16.7)
IVH	2	0	2(8)
Cerebral oedema	2	2	4(16.7)
Contusion	1	5	6(25)
IPH	2	3	5(20.8)
ICP	0	3	3(12.5)
Facial fractures	4	1	5(20.8)
Pneumocephalus	2	1	3(12.5)
Skull fracture	5	3	8(33)
Hygroma	0	0	0(0)
Outcomes:			
Alive	3	6	9(37.5)
Dead	5	1	6(25)
Disability	4	3	7(29)
Unknown	2	0	2(8)
Craniotomy	0	5	5(20.8)

SDH: subdural haematoma; SAH: subarachnoid haematoma; EDH: extradural haematoma; IVH: intraventricular haemorrhage; IPH: intraparenchymal hemorrhage; ICP: intracranial pressure; GCS: Glasgow coma scale; BP: Blood pressure. *Percentages are more than 100% as patients can have multiple pathologies on CT scan.

Furthermore 10 patients with localized lesions transferred to tertiary hospital, 9 returned to Leratong hospital, 5 had craniotomy and 4 were managed conservatively. Indication for surgery varied according to the patient status and CT findings. Patients with GCS above 6/15 with space occupying lesion (4 subdural haematoma and 1 epidural haematoma) and significant midline shift had craniotomy for drainage of the haematoma (Figure 3.5).

Figure 3.5



3.3 **General outcomes of the study sample**

3.3.1 **Complications**

Complications such as lung aspiration and respiratory acidosis were related to airway management and ventilatory interventions. Infections occurred in a small number of patients: 3% (3/95) patients had pneumonia, 1% (1/95) bone sepsis post craniotomy, 1% (1/95) urinary tract infection and 2% (2/95) had septicaemia. One of the patients that suffered septicaemia was severely immunocompromised (retroviral disease with CD4 count = 95) and contracted pneumonia 7 days following craniotomy for drainage of a bilateral subdural haematoma with local wound and bone sepsis. Then attended rehabilitation for left hemiplegia, speech and swallowing problems. The other patient did not have underlying chronic disease that would have contributed to development of septicaemia and both patients had craniotomy done for an acute subdural haematoma.

3.3.2 **Disability**

Cognitive impairment, cranial nerve palsy and motor dysfunction were most common disabilities at presentation. During recovery phase severe TBI patients experienced difficulty in producing fluent, intelligible language, comprehending simple instruction, feeding problems and multiple functional losses when compared to moderate TBI. Post concussion symptoms were only seen in one patient with moderate TBI. Of the 95 patients, only one developed epilepsy. Four patients remained in persistent vegetative state, 8 patients with severe disability, 15 patients with moderate disability and 30 patients had good recovery (Table 3.8). Therefore 28.4% (27/95) required rehabilitation.

Table 3.8 Outcomes of patients with moderate and severe TBI

Age groups	Good recovery n (%)	Moderate disability n (%)	Severe disability n (%)	Vegetative n (%)	Dead n (%)	Unknown outcome n (%)	Total n (%)
0-10	3 (3.2)	1 (1)	0 (0)	1 (1.1)	4 (4.2)	0 (0)	9 (9.5)
11-20	6 (6.3)	3 (3.2)	1 (1.1)	0 (0)	2 (2)	0 (0)	12 (12.6)
21-30	11 (12)	9 (9.5)	1 (1.1)	2 (2.1)	12 (12.6)	3 (3.2)	38 (40)
31-40	4 (4.2)	1 (1.1)	1 (1.1)	0 (0)	5 (5.3)	0 (0)	11 (11.7)
>41	3 (3.2)	1 (1.1)	4 (4.2)	0 (0)	6 (6.3)	2 (2.1)	16 (16.8)
unknown	3 (3.2)	1 (1.1)	1 (1.1)	1 (1.1)	2 (2.1)	1 (1.1)	9 (9.5)
Total	30 (32.1)	16 (18)	8 (8.4)	4 (4.2)	31 (32.6)	6 (6.3)	95 (100)

Good recovery: no functional loss; Moderate disability: isolated neurological disability (e.g. cranial nerve palsy); Severe disability: prolonged coma and loss of limb function (hemiplegic, upper or lower limb monoplegia); Vegetative state: multiple neurological impairments not compatible with return of independent functioning.

3.3.3 **Relationship between the outcome and clinical presentation**

Hypotension (p=0.009), GCS (7.3±3.3, p=0.001) and presence of contusion (p=0.035) were associated with morbidity. TBI patients with hygroma (p=0.02) already had obvious neurological deficit at the time CT scan was requested. In contrast facial fractures (p =0.009) were not associated with development of complications, disability or death (Table 3.9).

The following clinical presentation showed a statistically significant association with death; subdural haematoma (10/22, p=0.015), GCS (5.8±2.7, p<0.01), pulse (76.4±29.7 beats/min, p=0.006) and abnormal pupil characteristics (22/41; p=0.003) (Table 3.10). However, the majority of the patients with brain contusion 88% (22/25) survived (p=0.04).

Table 3.9 Relationship between clinical presentation and complications

Variables	With complications or dead	Without complications	P value
No. of patients	59	36	
Age	32.2 ± 21.3	36.3 ± 27.5	0.42
SBP	117.4 ± 44.9	134.8 ± 33.7	0.06
DBP	69.1 ± 25.7	83.5 ± 19.6	**0.009**
Pulse	89.2 ± 32.5	88.8 ± 21.8	0.95
GCS	7.3 ± 3.3	9.5 ± 2.3	**0.001**
Pupil (abnormal)	30	11	0.059
SDH	17	5	0.13
SAH	11	8	0.75
EDH	3	3	0.06
IVH	5	1	1.00
Cerebral oedema	6	8	0.33
Contusion	12	13	**0.035**
IPH	8	4	0.50
ICP	1	6	1.00
Facial fractures	4	9	**0.01**
Pneumocephalus	1	1	1.00
Skull fracture	15	9	0.82
Hygroma	3	0	**0.29**

Values represent number of patients or mean ±SD. SDH: subdural haematoma; SAH: subarachnoid haematoma; EDH: extradural haematoma; IVH: intraventricular haemorrhage; IPH: intraparenchymal hemorrhage; ICP: intracranial pressure; GCS: Glasgow coma score. Number of patients does not add up to 95 as they have multiple pathologies on CT scan.

Table 3.10 Relationship between clinical presentation, abnormal CT findings and mortality

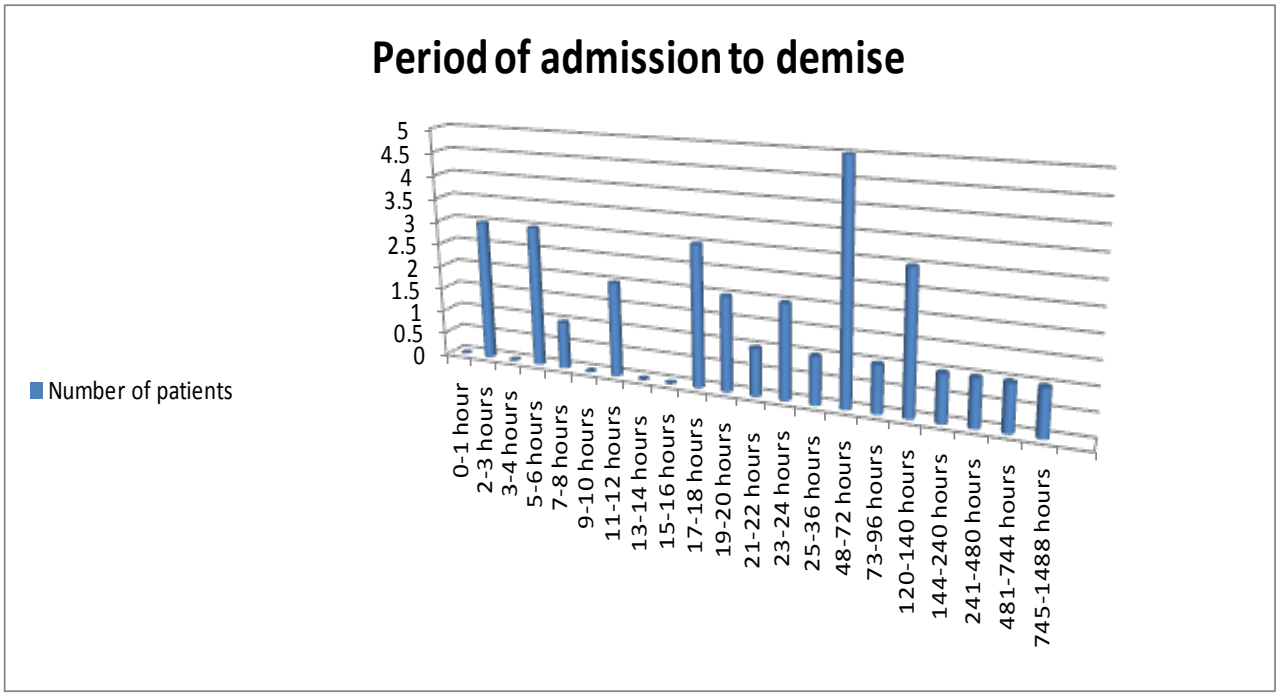
Variables	Dead	Alive	P value
No. of patients	31	58	
Age (in years)	27.8 ± 16.6	27.4 ± 12.8	0.88
SBP (mmHg)	120.0 ± 44.0	126.5 ± 37.4	0.93
DBP (mmHg)	77.3± 17.5	75.9 ± 21.7	0.63
Pulse (beats/min)	76.4 ± 29.7	93.6 ± 25.7	**0.006**
GCS	5.8 ± 2.7	9.2 ± 2.6	**0.000**
Pupil (abnormal)	22	19	**0.003**
SDH	10	12	**0.015**
SAH	5	14	0.99
EDH	2	3	0.56
IVH	3	3	0.17
Cerebral oedema	5	9	0.40
Contusion	3	22	**0.04**
IPH	4	8	0.60
ICP	2	5	0.89
Facial fractures	3	10	0.77
Pneumocephalus	0	2	0.39
Skull fracture	6	18	0.97
Hygroma	0	3	0.28

Values represent number of patients or mean ±SD. SDH: subdural haematoma; SAH: subarachnoid haematoma; EDH: extradural haematoma; IVH: intraventricular haemorrhage; IPH: intraparenchymal hemorrhage; ICP: intracranial pressure; GCS: Glasgow coma scale

3.3.4 **Time-to-death analysis**

A total of 31/95 patients died. Out of them 54% (27/50) presented with severe TBI and 8% (4/45) had moderate TBI. Most patients 35.6% (11/31) who died were pedestrians hit by motorists then followed by gunshot 19% (6/31), assault 19% (6/31), motor vehicle accident 9.7% (3/31), falls 9.7% (3/31) and 7% (2/31) the cause of injury was unknown. All patients 60% (6/10) presenting with both entrance and exit gunshot wounds died, while all patients 20% (2/10) with tangential gunshot wounds survived. The remaining 20% (2/10) of penetrating gunshot wounded patients had mixed outcomes.

Figure 3.6 Period of admission to death



Flat blue dot= 0 patients; raised bars= number of patients on x-axis; 96 hours= 4 days ; 120-140 hours≈ 5 days; 144-240≈ 6-10 days ; 241-480 hours≈ 10-20 days; 481-744≈20-31 days ; 745-1488 hours≈ 1-2 months.

Seventy four percent (23/31) of TBI patients who died were unstable and progressed to brainstem death within 72 hours of admission to the hospital. Among them two children (4 year olds) fell from the height and a 6 days old neonate who was hit on the head by a falling pole died within 24 hours of the injury. The remaining eight patients died after 6 days.

CHAPTER 4

4.0 **Discussion**

The results of this study suggest that regional hospitals might be appropriate facilities for triaging and conservative management of TBI. About 77.5% patients with mild head injury were admitted in hospital and managed conservatively. The remaining 22.5 % had moderate and severe (10.6% and 11.9% respectively) TBI. A similar distribution was seen in the literature where 80% of patients had mild TBI, 10% moderate and 10% severe.[4,9]

According to most of the literature the major cause of TBI is road traffic accidents.[1,2,10,33] In this study, pedestrian vehicle accident was the most common mechanism of injury followed by motor vehicle collision, assault, gunshot, falls from a height and falling objects on the head. Motorbike and cyclist were uncommon mechanisms of injury. Gunshot injuries were seen in those involved in fire exchange with police during a criminal act or self inflicted. This was possibly related to firearms being readily available to the community and with the high numbers of armed robberies taking place in West rand region of Gauteng Province, more victims of interpersonal violence were expected.

Similarly two studies[16,38] conducted in United Kingdom found that road traffic accidents were a dominant mechanism of injury for moderate and severe head injuries. Falls were the second commonest cause of TBI in their area. In contrast to our results in Leratong hospital, assault was the second commonest cause of injury.

It would be assumed that festivities will be associated with a higher incidence of TBI. However November (15%), August (15%) and May 2009 (13%) had the highest numbers of TBI. This was probably due to visible policing 'arrive alive' campaigns and other interventions by law enforcement during school holidays.

The highest incidence of TBI was mostly males of age 21-30 years and was similar to the findings in previous studies. Most authors[2,10,38] apply different age group ranges in their studies but males 20-40 years are most affected by trauma. Possible explanation could be that men elicit a high risk behavior (aggression, violence, car racing in public roads etc) especially under influence of alcohol when compared to females under the same circumstances.[39]

At Leratong Hospital CT scan was used for diagnosing intracranial traumatic lesions. In 78% of TBI patients in this study CT scan of the head was performed. The major indication was GCS 3-12 and mechanism of injury. Other indications mentioned were abnormal pupil size, presence of seizures, skull fracture, loss of consciousness, visible injuries on head, cranial nerve palsy, alcohol intoxication, suspected raised intracranial pressure and poor recovery. Significant proportions (96%) of CT head were abnormal and these results confirmed that moderate to severe TBI carry high probability for intracranial lesions. The majority of patients admitted at night or weekends in Leratong hospital had CT scans done the next day in order to avoid inter-hospital transfers. Consequently one patient deteriorated and died. Delaying CT scan to the next day is not encouraged by trauma system of care. NICE (National institute for clinical excellence) head injury guidelines stipulate that CT scan should be performed within one hour of an acute indication.[40] MacNamara et al suggest that rapid access to CT image transfer link to

the neurosurgical center is vital in the initial assessment of moderate and severe head injury in hospitals that do not have neurosurgery on site.[41]

Patient transfers for CT purposes could be dangerous and need to be avoided by having an on-site CT services. Patients who travelled from Leratong hospital to neighbouring regional hospital for CT scan had poor outcomes (of the 14 patients 5 died, 4 had moderate disability and two had unknown outcome). It was mostly due to severity of their clinical condition and delays in inter-hospital transfers due to unavailability of transport.

Other constrains reported were unavailability of a vacant bed in an intensive care unit (ICU) in the receiving hospital with neurosurgical expertise. Unavailability of ICU beds in the admitting hospital leads to delay in sending patients for definitive care and this is a widespread problem in South Africa. A study conducted in SA in assessing critical care resources found that 77% (304/396) public sector hospitals do not have ICU/high care facilities when compared to 16% (40/256) in private hospital.[42] They also identified problems with inter-hospital transportation namely: insufficient number of ICU ambulance, shortage of skilled staff, poor working condition of vehicles and road conditions especially in rural areas which impacted negatively on patient outcome. In addition several publications have reported an occurrence of adverse events during inter-hospital transfers ranging from loss of intravenous access, cardiopulmonary collapse to neurological deterioration.[43-46] There were also concerns regarding skill level of medical escorts (incapable of performing basic resuscitation), their inexperience in care of critically ill and tendencies to ignore medical advice for en route management of patients.[44] Technical problems like equipment failure or shortage of oxygen on road

contributed to clinical deterioration of patients; yet some are preventable.[45] Therefore en-route condition could have also contributed to the poor outcome of transferred patients from Leratong hospital.

Transfer of patients always depends on availability of resources and has cost implications. Risk of transfer needs to be weighed against benefit. Reports[43-49] suggests that acute inter-hospital transfer increase both morbidity and mortality but there is many cofounding variables that influence outcomes (e.g. severity of illness, extent of resuscitation, the expertise available before and during transfer).

Delay to neurosurgical care is a cumulative event. It starts at the regional hospital during clinical evaluation, resuscitation and CT scan. At the referred hospital difficulties were experienced in finding the receiving doctor and the distance travelled were time consuming thereby increase delay to neurosurgical care. A Providence study found that transfer time varied: TBI patients with GCS <3 were transferred expeditiously within 1 hour 50 minutes post stabilization when compared to GCS 7-9 transferred at an average of 3 hours 15 minutes.[50] However the mean time from arrival to transfer was 3 hours 47 minutes, from initial assessment to CT scan approximately 30 minutes and CT scan to disposal one hour 44 minutes.[51] The greatest delay prior to transfer of TBI patients with GCS <13 was experienced after CT scan. But in this study it took an average of 6 hours before departure to the next hospital with the earliest transfer at 1 hour 40 minutes and latest 10 hours. The transfer did not depend on the GCS score but availability of ICU bed and the arrival of the emergency medical services.

Transfer criteria had always been a debatable issue. The main aim of inter-hospital transfer was to maximize patient's benefit by allowing access to resource such as CT scan and neurosurgical expertise.[48] Since 1978 to 1981 in Glasgow CT scan was only available to tertiary hospitals therefore peripheral hospitals transferred TBI patients to neurosurgeon for diagnosis and management. The criteria applied for transfer were coma in a breathing spontaneously TBI patient, at least one reacting pupil, history of prolonged confusion of 6 hours post injury and presence of skull fracture.[52] This transfer criteria included mild, moderate and severe head injuries excluding brain dead patients. ATLS® guidelines have broad transfer criteria as well, which is intended to identify TBI patients with acute intracranial traumatic lesions and does not consider prognosis of severe TBI. Furthermore, they accommodate non-tertiary hospital without CT scan facilities to transfer high risks TBI for a dual purpose of diagnostic and optimal care. In this study ATLS® guidelines were adjusted, CT scan referrals were sent to the nearest hospital instead of tertiary hospital (in accordance to the Provincial health policy) and only those identified for neurosurgery were re-routed to the tertiary hospital. Alternatively other studies[48,53,54] could identify injured patients requiring high level of care by just assessing clinical presentation in the ED. Hence injured patients with GCS <13, hypothermia <36.6 ^{0}C, tachycardia >98 beats/min and respiration <16 were likely to require high therapeutic intensity interventions (such as neurosurgical procedures, exploratory laparotomy, vasopressor support, massive blood transfusion). In this study patients were triaged prior to transfer and clinical presentation was prioritized. From the 10 transferred patients 87.5% had GCS >6, 62.5% had a normal systolic blood pressure, 45.8% had tachycardia, pupil normal in 50%, skull fracture in 42% and 87.5% had acute intracranial traumatic lesions. Transfers to neurosurgery were based

on eligibility for craniotomy in a haemodynamically stabilized TBI patient and a probable good prognosis. Only 5% of moderate to severe TBI patients had craniotomy performed for space occupying lesions (4 craniotomies done for subdural haematoma and one done for extradural haematoma). Similarly to our findings recent publications[10,31] have showed that a small number of TBI patients require craniotomy, at least 6-10% required craniotomy and more than 90% conservative management. Therefore patients requiring non-operative management and non invasive monitors were suitable candidates to be managed in regional hospital setting.

Furthermore, the size of haematoma was recorded in millimeters when performing craniotomy however there were inconsistencies in describing the size of hematomas by different radiologists and practitioners. Some doctors measured the exact size of haematoma in millimeters while other practitioners used ordinal scale to describe the size of the haematoma. Marshall's classification could be used to standardize CT scan reporting. This classification (especially level 2 to 4) described the intracranial lesion by size, multiplicity and behavior towards other brain structures.

TBI patients with profound structural brain damage, persistent vegetative state and brain death did not benefit from neurosurgical transfers. A diagnosis of vegetative state was made if a patient showed no evidence of awareness of self or environment, no evidence of sustained, reproducible, purposeful or voluntary behavioral response to sensory stimuli and critically no evidence of comprehensive language and usually those patients died.[10] Therefore aggressive management should be reserved for patients who are likely to survive.[18] In this study TBI

patients with GCS <5 were not accepted for transfer because poor outcome was associated with a GCS 3-5.

A study by Chamoun et al showed that GCS 3 is not the only criteria to be taken into account when judging salvageability of severely injured TBI.[55] They found that GCS 3 was associated with mortality rate of 49.5% (93/189), poor prognosis in elderly, patients presenting with raised ICP, bilateral fixed and dilated pupils. Pupil characteristics have diverse outcomes. Bilateral reactive pupil had 30%, unilateral non-reactive (>30%) and bilaterally fixed pupils 70-90% mortality. [4] Therefore all forms of abnormal pupil carry some degree of poor prognosis.

In this study, patients with severe TBI experienced a statistically significant higher mortality 54% (27/50) when compared to moderate TBI 8% (4/45). Bradycardia and abnormal pupil characteristics (dilated, constricted or fixed) was associated with mortality while there was no association with hypotension. Presence of bradycardia signifies a disturbance of the vital functions of the brainstem; this may result from raised intracranial pressure. The combination of systemic hypertension and bradycardia is regarded as a Cushing reflex, hence associated with mortality. The presence of abnormal pupil suggests compression of oculomotor nerve, optic nerve or pontine dysfunction by a localized mass lesion or in form of brain herniation. However ICP above 40 mmHg was associated with brain herniation and poor outcomes. Therefore abnormal pupil might be used as an indirect measure of a likelihood of raised intracranial pressure.

In this study skull fractures, contusions and subdural haematomas were among the most common CT scan finding in TBI. The pathogenesis of contusions and subdural haematoma

explains why they were the commonest presenting brain lesions. The acceleration and deceleration forces produced by the impact e.g. road traffic accident, leads to injury of the brain against the skull and tearing of bridging veins. Primary brain injury occurred at impact and producing instant damage of the neurons. Skull fractures were produced by direct force to the skull, might present with or without brain injury and hence they were not associated with mortality. Subdural haematoma was associated with mortality while contusion had better prognosis.

A post morterm conducted on victims of traffic accidents proved that diffuse axonal injury was associated with acute subdural haemorrhage and that subsequent mortality was due to primary underlying brain injury rather than the presence of subdural haematoma alone.[56]

Generally mortality rates have been estimated at less than 30% in moderate TBI and 40-50% in severe TBI.[3,4,9] However, patients with gunshot wound to the head and with GCS less than 5 had 100% mortality.[9] In a retrospective study[57] (85 patients with craniocerebral gunshot wounds) reported a high mortality of 86 % in penetrating gunshot wound and lower mortality (4%) in a non-penetrating gunshot wound to the head. Further, penetrating gunshot wound patients were mostly compromised with average GCS 3.3 while non penetrating group had GCS 11. In gunshot wounds to the head low GCS score at admission, presence of multilobar or skull base and involvement of ventricle was associated with high mortality.[17,31,57-59] Similarly, TBI patients in Leratong hospital displayed similar pattern of outcomes with regard to severity of their condition and the most severe mechanism of injury was perforating (across the brain matter) gunshot wound to the head which were fatal. This is explained by the fact that lateral

trajectory enables easy access to vital and vulnerable structures of the brain such as ventricular system. It is also one of the causes for bihemispheric cerebral injury.

Risk factors to poor outcome have been identified in various studies[35,59] age, gender, genetics, pupil characteristics, Glasgow coma scale, abnormal CT findings and mechanism of injury were categorized as recognized factors of poor outcome because they could not be modified by therapy.[4] A prospective study[38] with 1554 patients with head injury showed that children and adolescent had highest death rate in comparison to elderly patients. In contrast another study[10] of 3861 patients with head injury reported higher mortality in age groups 31-40 and elderly (71-80 years) had lowest mortality. However, in Leratong hospital it was shown that mortality was not associated to age. Furthermore, approximately 33% of patients studied died, 20% suffered various grades of disability especially those with intracranial pathology, facial fractures and abnormal pupil. This could be explained by the injury of white matter during primary impact of the head. In addition, haemodynamic instability (hypotension) showed an association with morbidity, meaning that systemic hypotension leads hypoxia (secondary brain insult) by decreasing cerebral perfusion.

The clinical presentation of TBI associated with poor outcome has been identified in this study, however these characteristics (perforating gunshot wounds, intracranial pathology, a lower GCS, haemodynamic instability, bradycardia and abnormal pupil) do not present alone but in combination. Therefore moderate and severe TBI patients are identified as high risk groups.

CHAPTER 5

5.1 **Conclusion and recommendations**

This audit contributed to our knowledge of moderate and severe traumatic brain injury in a regional hospital (Leratong) in the following way:

Firstly the results indicate that road accidents are responsible for the majority of TBI and perforating gunshot injuries to the skull are still lethal. These events are mostly occurring during weekends and at night. Another factor is the vulnerability of men in their 3rd and 4th decade of life. The highest number of TBI was experienced in August and November instead of holiday period. These suggest that if policing were to be reinforced throughout the year the plane may be leveled. Another intervention is primary prevention of head injuries. Embracing preexisting head injury awareness day (20th March yearly) by advertising and hosting active workshop may improve social responsibility.

Secondly, CT scan remains an important tool of diagnosis in patients with moderate to severe TBI and should be used responsibly. This study showed that most patients where CT scan was performed had intracranial traumatic lesions and patients with GCS 2/10 were excluded. However no exclusion criterion in CT scan guidelines for patients with severe TBI was indicated. Performing CT scan indiscriminately in this group of patients (especially GCS 2) considering resource constrains at hand, it is not cost effective and sustainable. For that reason we need to create our own criteria. Delay in diagnosing an intracranial lesion in a critical patient is

hazardous. Similarly inter-hospital referrals for CT scan endanger the lives of our TBI patients. A 24 hour CT scan access with radiologist support is vital.

Thirdly, patients requiring non-operative management were suitable candidates to be managed in regional hospital setting. A small percentage of TBI patients need neurosurgical expertise and this finding was consistent with previous studies. In addition some patients required placement of ICP monitors, control of status epilepticus and ongoing resuscitation for cerebral perfusion as part of conservative management of TBI (NB this are specialist level interventions). Therefore, this highlights the need to review triage criteria to neurosurgeon while being specific to our South African hospital settings. The present study showed that: resuscitated TBI patients with a GCS score above 5 and presenting with an intracranial haematoma causing neurological deterioration were successfully transferred to a neurosurgical unit. No clinically significant association to outcomes was seen in TBI patients with skull fractures. Therefore, referring back to ATLS® guidelines using skull fracture as a guideline for inter-hospital transfer was not applicable in our setting as these patients are not critical.

Finally, a third of TBI patients had a full functional recovery, another 3rd disabled and a 3rd also died. In accordance to the percentage of patients who died, was similar to the mortality ranges of moderate TBI in the literature. However those with severe TBI had 4% higher mortality. Subdural haematoma, a lower GCS score, bradycardia and abnormal pupil characteristics showed a strong association with mortality. Patients with cerebral contusion and hypotension had acute complications and residual disabilities. Facial fractures were less likely to have complications or die. This study could not exhibit the relationship between poor outcome

and CT scan transfers due to insufficient sample size of the transferred group. As a result a need arises for a follow up study on outcomes of transferred patients, with information gathered from initial referring hospital, data in-transit and data from the receiving hospital. Lastly, conduction of multicenter prospective study in regional hospitals will enable a more comprehensive understanding of head trauma at this level of care.

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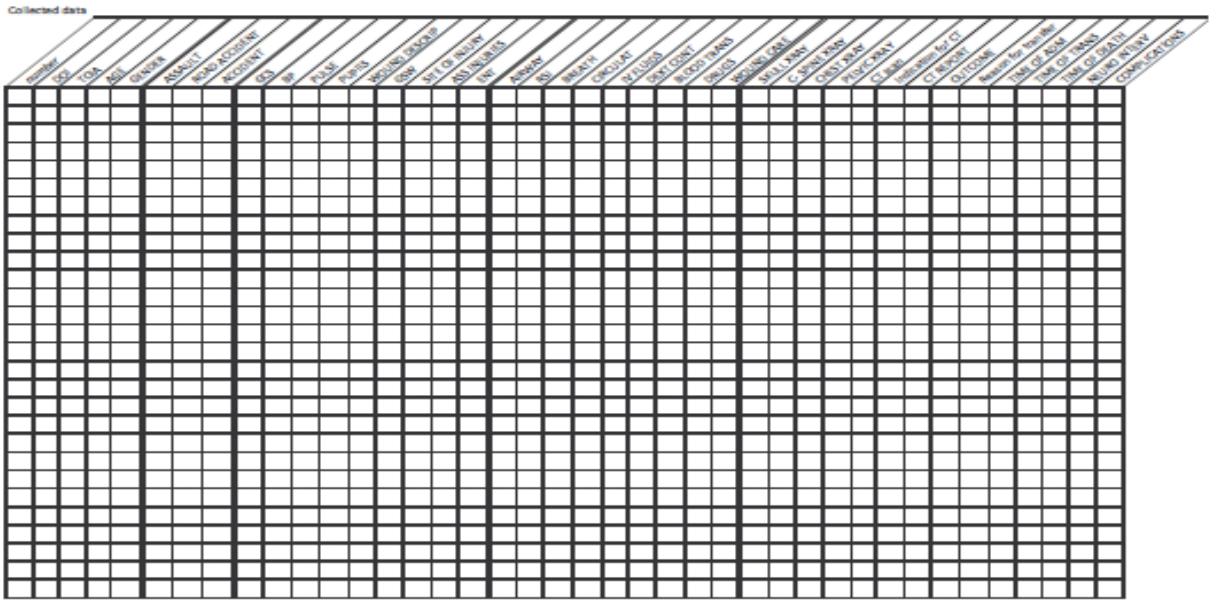
ANNEXURE A: Data collection sheet

Code number:		DOI:	TOIA:	Age:	
Gender=		1. male	2. female		
History: (tick)					
Assault=	1. stab	2. blunt force	3. suicide attempt		4. gunshot
Road accident=		1. MVA	2. PVA	3. MBA	4. MCA
Accident=	1. falling objects		2. falling from height		3. unknown
Examination findings					
GCS:		BP:		Pulse:	
Pupils=		1. dilated R	2. dilated L	3. fixed	4. unequal
wound description=		1. laceration	2. abrasion	3. bruises	4. swelling
		5. incised	6. gunshot wound		
gunshot wound=		1. entrance	2. exit	3. tangential	
Site of injury					
1. temporal	2. parietal	3. occipital	4. frontal	5. facial	
Associated injuries:					
ENT=	1.bleeding	2. fluid	3. normal		
Management					
Airway=	1. O2 supp	2. OP tube	3. ET tube	4. tracheostomy	RSI=1.yes/0.no
Breathing=	1. spontaneous		2. manual bagging		3.ventilator
Circulation=	1. active bleeding		2. controlled bleeding		
	IV fluids= 1.yes/0.no		Dextrose containing= 1.yes/0.no		
	Blood transfusion= 1.yes/0.no				
Drugs=	1.tetanus toxoid		2. BZN	3.Antibiotic	4. epanutin
	5. Mannitol				
Wound care= 1.yes/0.no					
INVESTIGATIONS					
Radiological=					
Skull= 1.yes/0. no		C-spine= 1.yes/0. no		CT scan= 1.yes/0. no	
Chest= 1.yes/0. no		pelvis= 1.yes/0. no			
Indication for CT scan:					
OUTCOME=		1. admission	2. transfer	3. deceased	4. complications
Reason for transfer:					
Time of admission:			Time of transfer/ death:		
neurological intervention= 1.yes/0.no					

ANNEXURE B


Collected data
number
DOI
TOA
AGE
GENDER
ASSAULT
ROAD ACCIDENT
ACCIDENT
GCS
BP
PULSE
PUPILS
WOUND DESCRIP
GSW
SITE OF INJURY
ASS INJURIES
EMT
AIRWAY
RR
BREATH
CIRCULAT
IV FLUIDS
DIAY CONT
BLOOD TRANS
DRUGS
WOUND CARE
SKULL XRAY
C-SPINE XRAY
CHEST XRAY
PELVIC XRAY
CT scan
Indications for CT
CT REPORT
OUTCOME
Reasons for transfer
TIME OF ADM
TIME OF TRANS
TIME OF DEATH
WOUND INTERV
COMPLICATIONS

ANNEXURE C

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG
Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)
R14/49 Dr Jane Sikundla

CLEARANCE CERTIFICATE	**M10512**
PROJECT	An Audit of Patients with Moderate to Severe Head Injries in Leratong Regional Hospital
INVESTIGATORS	Dr Jane Sikundla.
DEPARTMENT	Department of Emergency Medicine
DATE CONSIDERED	28/05/2010
DECISION OF THE COMMITTEE*	Approved unconditionally

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

DATE 15/12/2010 **CHAIRPERSON** .....
(Professor PE Cleaton-Jones)

*Guidelines for written 'informed consent' attached where applicable
cc: Supervisor : Dr J Ouma

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...