Prehospital airway management in severe closed traumatic brain injury: An analysis of its impact on outcome

By

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DECLARATION

I, **Simpiwe Sobuwa**, hereby declare that the work on which this dissertation/thesis is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

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GLOSSARY

ALS	Advanced life support
BLS	Basic life support
BP	Blood Pressure
CT	Computed tomography
DAI	Diffuse axonal injury
EC	Emergency Centre
ECP	Emergency Care Practitioner
EDH	Extradural haematoma
GCS	Glasgow Coma Scale
GOS	Glasgow Outcome Score
GSH	Groote Schuur Hospital
HPCSA	Health Professions Council of South Africa
ICP	Intracranial pressure
ILS	Intermediate life support
MAP	Mean arterial pressure
MVA	Motor vehicle accident
RTA	Road Traffic Accidents
SAH	Subarachnoid haemorrhage
SDH	Subdural haemorrhage
SBI	Secondary brain injury
SpO_2	Pulse oximetry reading
TBH	Tygerberg Hospital
TBI	Traumatic brain injury
TCDB	Trauma Coma Databank

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ABSTRACT

Purpose

The purpose of this study was to describe the outcomes of patients with severe traumatic brain injury managed by emergency care providers in the Cape Town Metropole.

Methodology

A descriptive observational analysis of consecutively injured adult patients who sustained severe traumatic brain injury in the Cape Town Metropole. Patients were categorised by their method of airway management: rapid sequence intubation, sedation-assisted intubation, failed intubation, basic airway management and intubated without drugs. Severe traumatic brain injury was defined by GCS of 8 or less. 124 patients were included for analysis. Prehospital transport times were determined for each group.

Findings

There was a statistical significance linking airway management and outcome (p= 0.013). Patients who underwent basic airway management had the highest proportion of the good outcome group (72.9%) compared to patients who were intubated in the prehospital setting. A good outcome was the result in 61.8% and 38.4% of patients who experienced sedation-assisted intubation and RSI respectively. Secondary to patients intubated without drugs, RSI had the highest proportion of the poor outcome group (61.5%) followed by the sedation-assisted group (38.2%).

The group that underwent basic airway management had the shortest mean total transport time of 51 minutes followed by the failed intubation group with 58 minutes. Patients who underwent RSI had a mean total transport time of 61 minutes and the group who had an LMA in-situ due to a failed intubation had a mean total time of 74 minutes. Lastly, the group that encountered sedation-assisted intubation had a mean total time of 70 minutes.

There appears to be no association between the time taken in getting the patient to hospital and outcome in severe traumatic brain injury (p= 0.647). 42.9% (n= 24) of the patients that were in hospital within an hour from dispatch time had a poor outcome and 57.1% (n= 32) had a good outcome. In the group that missed the 60 minute cut-off, 38.7% (n= 24) had a poor outcome and 61.2% (n= 38) had a good outcome.

Conclusion

Pre-hospital intubation did not demonstrate improved outcomes over basic airway management in patients with severe traumatic brain injury. A large prospective, randomized trial is warranted to yield some insight into how these airway interventions influence outcome in severe traumatic brain injury. While it appears intuitive that less pre-hospital time is better for trauma patients, this study failed to demonstrate a relationship between response times, total out-of hospital time and outcome.

CHAPTER 01

INTRODUCTION

1.1. Background to this Research

Traumatic Brain Injury (TBI) is the leading cause of death in young adults in the United States [1] although case fatalities are higher in low-middle income countries where resources are limited [2].

According to data from the forensic pathology services in Cape Town for the period 2006- 2010, on average 343 people have died from TBI in the Cape Town Metropole area each year. Road traffic accidents (RTA) is the most common cause of TBI (49%). This number is thought to be higher as the search for a cause of death is limited to head injuries on the database. Therefore patients diagnosed with multiple injuries might have been missed. Prevalence of TBI is higher amongst young adults, with the average age being 34 years. Furthermore, the mortality rate for males is almost 5 times higher than that of females. This can be attributed to alcohol and other substance abuse [3]. These findings coincide with the CRASH (corticosteroid randomisation after significant head injury) trial with 10 008 patients of which 81% were male [4].

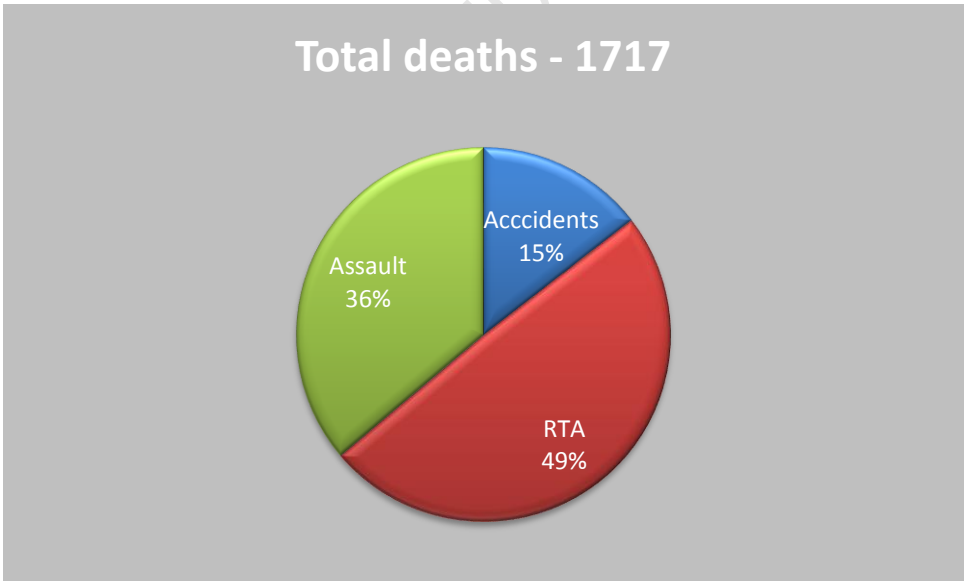

Figure 1: Mortality because of TBI in the Cape Metropole (2006-2010)

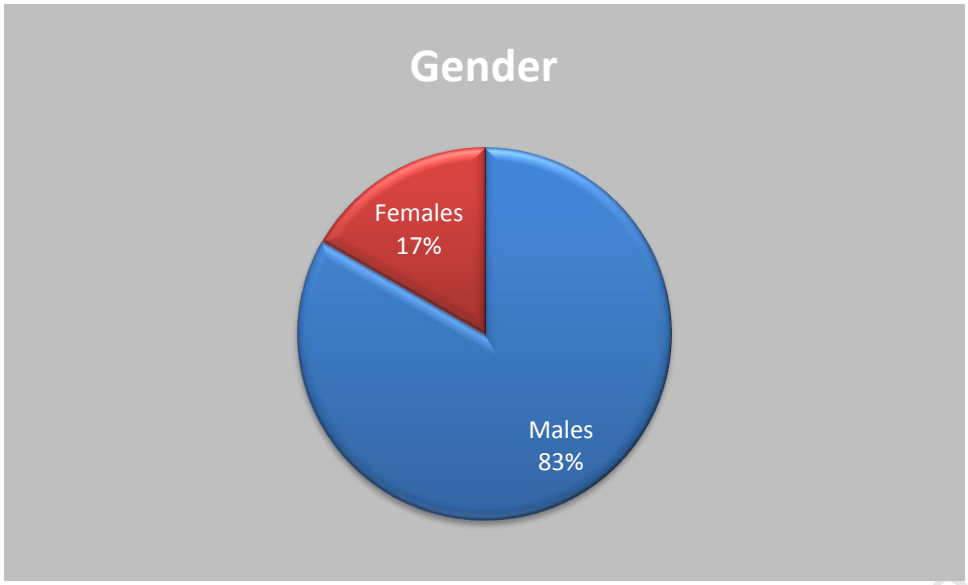

Figure 2: Gender distribution among deceased patients because of TBI in the Cape Metropole (2006-2010)

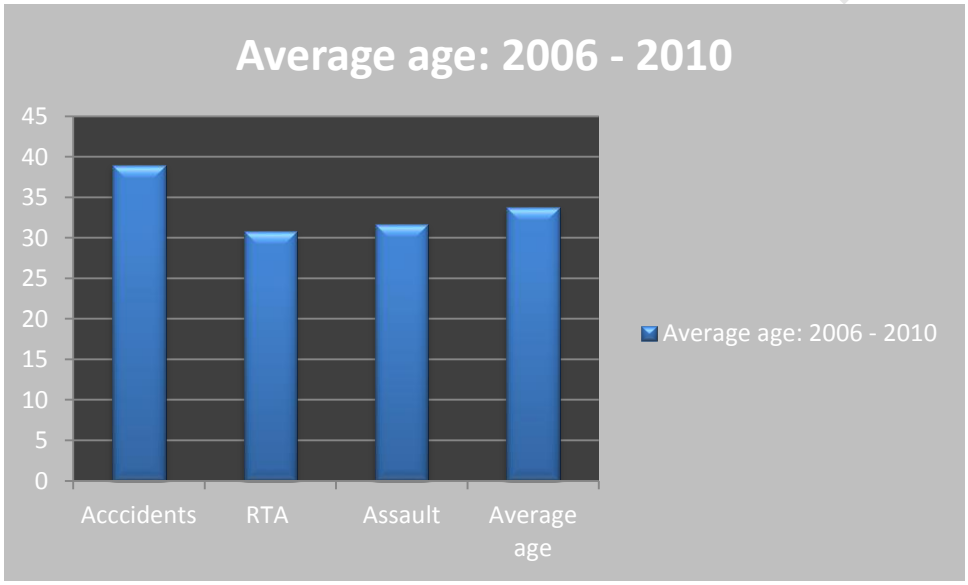

Figure 3: Average age for deceased patients resulting from TBI in the Cape Metropole

TBI survivors may experience psychological, physical and cognitive changes which may interfere with their daily living. This has a significant socio-economic impact on families as the injured person may potentially be unemployed. Management of TBI patients is rapidly evolving because of a greater understanding in the physiologic derangements resulting in secondary brain injury (SBI).

Secondary brain injury is also the leading cause of in-hospital deaths following TBI in the United States [5]. Recent evidence suggests that a single episode of hypotension

is associated with increased morbidity and doubling of mortality when compared to the patients without hypotensive episodes [6]. Patients with a combination of hypoxia and hypotension have even worse outcomes. The understanding of secondary brain injury resulting in worse outcomes can result in improved pre-hospital resuscitation of the patient in an effort to minimise the effects of secondary brain injury.

Pre-hospital assessment & appropriate management of the patient with traumatic brain injury is crucial to their survival. However, the role of pre-hospital airway management methods in severe TBI has been an area of controversy [7]. There interventions have been called into question as advanced life support practitioners tend to spend more time at the scene instead of rapid transport to the nearest appropriate facility [7].

The value of Emergency Medical Services (EMS) in the management of TBI was suggested by Colohan et al [8] who compared the risks of death in India to a United States cohort. The risk of death in India was two times higher due to the lack of an EMS system. They found that patients took longer to arrive at the Emergency Department (ED) in India which led to a difference in outcome [8].

Up until recently, all severe TBI patients in the field would be intubated by paramedics using midazolam and morphine. However, there is no evidence to motivate usage of these drugs for intubation of patients with severe TBI. Both drugs have the potential to cause hypotension and respiratory depression. The drugs have a synergistic effect when administered together thus hypotension and respiratory depression are more likely to occur when administered rapidly and in large doses [9]. Airway reflexes may still be present after administration of the above medication, thus increasing intracranial pressure during laryngoscopy.

In conducting this study, we hypothesize that this research may help in contributing to a better understanding of how pre-hospital airway management methods can help improve secondary brain injury. This study may also contribute to the quality provision of pre-hospital care for patients with TBI. The research may also identify possible problem areas in the pre-hospital management of closed severe TBI and form a platform for positive change and intervention.

1.2. Purpose of Study

The purpose of this study was to describe the outcomes of patients with severe traumatic brain injury as managed by various airway management methods by prehospital emergency care practitioners in the Western Cape, Cape Town Metropole.

1.3. Research Objectives

- The first objective was to assess the airway management methods currently practiced in the Cape Town Metropole in TBI
- The second objective was to assess the outcome of TBI in the study population
- The third objective was to describe the outcome of TBI with each airway management method employed in the study
- The fourth objective was to describe the association of airway management methods and prehospital transport times
- The fifth objective was to determine the association of prehospital transportation times on outcome following severe traumatic brain injury

1.4. Delineation of the Study

The study only recruited patients in South Africa, Western Cape, Cape Town, Metropole region who sustained a severe traumatic brain injury (GCS ≤8); by mechanism of injury or physical examination treated by prehospital emergency care practitioners and transported to Tygerberg and/or Groote Schuur hospital.

1.5. Definition of Terms

Advanced Life Support (ALS) Practitioner:

A health care practitioner that is on the paramedic register with the Health Professions Council of South Africa (HPCSA) Professional Board of Emergency Care after completing the Critical Care Assistant course or the 3 year National Diploma in Emergency Medical Care

Basic Airway Management:

Basic airway interventions aimed at clearing the airway and administration of oxygen via a face mask or bag-valve mask device

Basic Life Support Practitioner:

A health care professional who has successfully completed the basic ambulance assistant course and registered with the Health Professions Council of South Africa as a basic life support practitioner

Extradural Haematoma:

A collection of blood between the dura mater and the skull

Failed Intubation:

Failure to successfully insert an endotracheal tube past the vocal cords during attempted intubation

Glasgow Coma Scale:

A scale aimed at assessing level of consciousness of a patient following traumatic brain injury with the score ranging between 3 and 15

Glasgow Outcome Score:

A five-point scale given to victims of traumatic brain injury at some point during their recovery

Intermediate Life Support Practitioner:

A health care professional who has successfully completed the ambulance emergency assistant course and registered with the Health Professions Council of South Africa as an intermediate life support practitioner

Intracranial Pressure:

The pressure inside the cranial vault exerted on the brain tissue, cerebrospinal fluid and blood volume

Response Time:

The period from dispatch to scene arrival

Road Traffic Accident:

An incident where a motor vehicle collides with another vehicle, pedestrian or other stationery objects such as trees or poles

Sedation-assisted Intubation:

Sedation-assisted intubation is the administration of a sedative/induction agent prior to intubation thereby increasing the likelihood of endotracheal intubation by suppressing the gag reflex

Scene Time:

The amount of time spent on scene by emergency personnel calculated from arrival on scene till scene departure

Subdural Haematoma:

A collection of blood between the dura mater and the arachnoid mater

Total Time:

The period from dispatch to hospital arrival

Traumatic Brain Injury:

Traumatic brain injury is physical injury to brain tissue that temporarily or permanently impairs brain function

1.6. Overview of the Dissertation

The study is introduced in chapter 1 which encompasses the background of this study, purpose, objectives, limitations and definitions. The review of the literature is found in chapter 2. The methodology employed in this study is discussed in chapter 3 with regards to the design of the study, study setting, data collection, data analysis, inclusion/exclusion, sampling and ethical considerations of the study. The results of the study are described in chapter 4. These results are then discussed in chapter 5. The conclusion and recommendations will be found in chapter 6.

CHAPTER 02

LITERATURE REVIEW

Introduction

The review of the literature is found in this chapter which discusses the pathophysiology of a traumatic brain injury, both primary and secondary. The literature review then discusses the prehospital assessment of a patient with a traumatic brain injury and how outcome is predicted for this group of patients. Lastly, the pre-hospital airway management and in-hospital management of patients with severe traumatic brain injury will be discussed in this chapter.

A literature search was conducted on Pubmed using the following key words: [prehospital airway management] AND [severe traumatic brain injury]. The search was limited to studies on humans, publications in English and published up to 2011. The search yielded 108 articles which most were excluded as they were not relevant to the main research question. Letters, comments, case studies and editorials were excluded. Studies involving patients with severe TBI receiving prehospital airway management and reported on mortality or functional outcome were eligible for review.

10 articles were eventually included published between 1997 and 2010. Two of the articles were systematic reviews [93, 94] and one randomised controlled trial [104]. There were four retrospective reviews [89, 91, 92, 100] of trauma registries and three prospective cohort studies [90, 99, 101].

The one review [93] included three randomised controlled trials but none of them looked at patients with traumatic brain injury. They predominantly focused on patients with cardiac arrest in the prehospital setting. The other review [94] was unable to make any recommendations regarding prehospital intubation in patients with severe traumatic brain injury due to poor methodologies and inconsistent results. The randomised controlled trial reported an increase in favourable neurologic outcome at 6 months in patients undergoing prehospital rapid sequence intubation by paramedics.

Three studies [99-101] reported the prehospital airway management method as rapid sequence intubation and two of them [99, 100] demonstrated improvements in outcome at discharge and a decrease in hospital mortality for patients with severe traumatic

brain injury. The other study [101] showed an increased morbidity and mortality in traumatic brain injured patients undergoing prehospital rapid sequence intubation. The four outstanding reports [89-92] did not mention if any pharmacological agent/s was used during the intubation attempt in the prehospital milieu. Three of the reports [90-92] reported an increased morbidity and mortality with prehospital intubation in patients with a traumatic brain injury. One study [89] demonstrated an improvement in survival in patients with scene Glasgow coma score of 8 or less who underwent prehospital intubation.

2.1. Pathophysiology of Traumatic Brain Injury

2.1.1. Primary Brain Injury

Traumatic brain injury is physical injury to brain tissue that temporarily or permanently impairs brain function. Traumatic brain injury is incorrectly used synonymously with the term head injury but these are actually two separate entities. A head injury is defined as an injury to the head that is clinically evident upon physical examination and is recognised by the presence of lacerations, deformities, ecchymosis or cerebral spinal fluid otorrhea or rhinorrhea [10]. TBI on the other hand is traditionally divided into two subcategories; primary brain injury and secondary brain injury. Primary brain injury is a result of external forces and/or rapid acceleration/deceleration forces which occur at the moment of impact. The size and pattern of the damage is determined by the intensity, path and duration of these forces. Primary brain injury is often characterised into focal and diffuse lesions. Secondary brain injury is the physiological changes and the biochemical events that occur minutes to days following the primary insult [11].These changes are complex and are not clearly understood. The common types of primary and secondary brain injury are discussed below.

PATHOLOGY OF TBI

PRIMARY

Diffuse

Concussion

Axonal injury

Petechial haemorrhages

Focal

Focal contusion

Focal haematoma

Extradural

Subdural

Intracerebral haematoma

SECONDARY

Systemic

Hypoxia

Hypotension

Pyrexia

Anaemia

Hyper/hyponatremia

Hyper/hypocapnea

Hyper/hypoglucosemia

Cranial Events

Mass lesion

Intracranial hypertension

Cerebral edema

Vasospasm

Hydrocephalus

Infection

Seizures

Biochemical cascades

2.1.2. Diffuse Brain Injury

Traumatic concussion is a temporary and reversible altered mental state, lasting from seconds to minutes, usually less than six hours and often accompanied by retrograde amnesia. Concussions are regarded as mild when there is no loss of memory suffered with a temporary physiologic disturbance resulting from the sudden blow to the head. Concussions usually occur in contact sports such as rugby and/or boxing as a result of direct impact to the head. Patients who have suffered a concussion are advised to rest for a couple of weeks following the incident. The latter is due to the fact that concussed patients have been found to have abnormal cerebral autoregulation for quite a few days following the injury [12]. This makes them more susceptible to additional head trauma which can lead to cerebral oedema, a phenomena known as the 'second impact syndrome'.

Diffuse axonal injury (DAI) occurs when there is extensive, diffuse damage to the axonal fibres and myelin sheaths of the brain. It is sometimes clinically defined as a loss of consciousness for more than six hours in the absence of a precise focal lesion. DAI results from rapid acceleration/deceleration forces which are thought to result in shearing and tearing of the white mater tracts. DAI was first reported by Strich in 1956 who discovered diffuse degeneration of axons in patients with severe post traumatic dementia [13]. DAI is a histologic finding which consists of petechial haemorrhages, swelling and disruption of axons. The axonal injury can also occur due to the biochemical cascades that occur following the initial insult and might not arise from the primary insult.

It is common following motor vehicle accidents but can also arise with falls from a height [14] and is evident in the majority of patients with a lethal traumatic brain injury [15]. It is graded as mild, moderate and severe. Gennarelli et al graded patients with diffuse axonal injury as:

- Grade 1 - Axonal injury mainly in parasagittal white matter of the cerebral hemispheres;
- Grade 2 - As in Grade 1, plus lesions in the corpus callosum;
- Grade 3 - As in Grade 2, plus a focal lesion in the cerebral peduncle [16].

Patients with severe diffuse axonal injury are usually in a prolonged coma for weeks to months and often with an unfortunate outcome. Diffuse axonal injury is associated

with significant morbidity and mortality rate [17]. The Traumatic Coma Data Bank (TCDB) noted that 56% of patients with a closed severe head injury had a diffuse injury in comparison to 42% who had a focal mass lesion [18]. Nonetheless, patients with a focal mass lesion had a higher mortality compared to those with a diffuse injury 39% versus 24%.

2.1.3. Focal Brain Injury

Cerebral contusions are bruises along the brain tissue resulting from the brain being jarred around within the skull, causing vascular and tissue disturbance in the brain. Cerebral contusions occurring at the site of impact of the skull are called coup whilst those occurring on the opposite point of direct impact are called contra-coup. The latter occurs as a result of rapid deceleration usually occurring at places with an irregular internal surface in the skull. Some contusions are benign with no symptoms present and need no medical intervention. Others may cause cerebral edema and increased intracranial pressure. Small, hard objects tend to result in coup contusions due to the energy that tends to dissipate at the site of impact. Larger objects in contrast tend to result in contra-coup contusions as the energy dissipates at the end of the head motion causing less injury at the site of impact.

An extradural haematoma consists of a collection of blood between the skull and dura mater. It is most commonly associated with torn dural vessels, typically arterial as a result of a fracture to the temporal bone (91% of the time in adults and 75% in children) [19]. The incidence of an extradural haematoma in adults is lower in comparison to a subdural and intracerebral haematoma. The TCDB series observed that extradural haematomas were present in 6% of patients with severe closed traumatic brain injuries [18]. Extradural haematomas that result in rapid neurological deterioration following a lucid period of wakefulness and immediate loss of consciousness are usually caused by arterial bleeding as blood accumulates rapidly between the dura and the bone. Rivas et al showed that patients with an extradural collection of more than 150 ml of blood often have a poor prognosis demonstrating that the volume of the haematoma is a predictor of outcome [20].

The classic presentation of an extradural haematoma is a lucid interval, dilation of the ipsilateral pupil and contralateral hemiparesis. However these findings are not always present as the lucid interval only appears in 14% to 21% of cases [21]. The

contralateral hemiparesis results from the compression of the corticospinal tracts in the cerebral peduncle. This occurs in 70% to 80% of epidural haematomas in the frontotemporal region [22, 23]. These patients require prompt diagnosis and neurosurgical intervention to ensure survival as they rapidly deteriorate and die.

A subdural haematoma is an accumulation of blood between the dura mater and the pia-arachnoid mater. These usually arise due to an injury to the cortical veins that bridge the subdural space and drain into the dural sinuses. Subdural haematomas are more common than epidural haematomas as venous blood spreads freely throughout the subdural space, thereby covering the entire cerebral hemisphere [17]. The majority of focal intracranial lesions in the TCDB series were as a result of a subdural haematoma occurring in 24% of patients with a severe closed head injury. Compression of the brain by the haematoma leads to an increase in intracranial pressure, leading to an increase in morbidity and mortality.

Subdural haematomas are classified by their clinical presentation of symptoms. They are classified as acute when there is a collection of blood up to 48 hours post-insult, subacute when there is blood and fluid 2 to 14 days post insult and chronic typically > 14 days after injury. A chronic subdural haematoma may emerge and generate symptoms late weeks to months following the traumatic event. The elderly patients are more susceptible to these haematomas as their brain atrophies with aging resulting in an extra intracranial physiologic space. They may consider the traumatic event as insignificant or may have even forgotten it.

Maloney and Whatmore found subdural haematomas to occur between 26% and 63% for patients with traumatic brain injury [24]. Signs and symptoms of an acute subdural haematoma include agitation, drowsiness, confusion, slow cerebration, headaches, ipsilateral dilated and fixed pupil and contralateral hemiparesis. Subdural haematomas are associated with greater morbidity and mortality than epidural haematomas as most subdural haematomas occur with significant brain damage [25]. There majority of subdural haematomas occur following acceleration/deceleration injuries; up to 72% are caused by falls, and road traffic accidents result in 24% of subdural haematomas [26]. Therefore there is often concomitant brain injury i.e. DAI associated with the subdural bleed leading to a poor outcome. The most important management for a significant subdural haematoma is rapid surgical evacuation.

An intra-cerebral haematoma is a haemorrhage within the brain tissue itself, accounting for nearly 20% of all intracranial haematomas [27]. It is often caused by a lacerated blood vessel/s or from coalescence of contusions. It is classified as an intracerebral haematoma if the blood constitutes at least two thirds of the lesion otherwise it will be called a contusion. Signs and symptoms of an intra-cerebral haematoma may include hemiplegia on the contralateral side, decreasing level of consciousness to deep coma, headache and dilated pupil on the same side of the haemorrhage.

Subarachnoid haemorrhage may well transpire in cases of TBI if superficial micro-vessels are lacerated and blood collects in the subarachnoid space. It is also an 'independent' prognostic variable for poor outcome in patients following TBI. A large European study found a two-fold increase in poor outcome in patients sustaining a traumatic subarachnoid haemorrhage [28]. In one study, 39% of 753 patients with severe TBI were found to have a subarachnoid haemorrhage [29].

2.1.4. Skull Fractures

Skull fractures occur when the impact exceeds the skull's tolerance levels to damage. Linear skull fractures are the most common type of fractures [30], resulting from low-velocity blunt trauma and it increases the likelihood of an intracranial haematoma.

A depressed skull fracture occurs due to blunt trauma resulting in the bone being displaced inwards. Blunt trauma that causes a skull fracture needs to be of significant force and can therefore result in underlying injury to the brain. The major problem relates to open skull fractures as this increases the possibility of intracranial sepsis that poses a major risk for additional secondary damage to the brain. The septic complication can be prevented and treated. Therefore emergency debridement is required in significant depressed skull fractures.

Skull base fractures may manifest as periorbital ecchymosis (raccoon eyes) and leakage of CSF (cerebrospinal fluid) through the nose (rhinorrhea) or ears (otorrhea). Middle and posterior fossae fractures that injure the temporal petrous bone can result in bleeding over the mastoid region (Battle's sign). It can also cause CSF leakage with possible infective complications.

2.2. Secondary Brain Injury

Secondary injury is non-mechanical damage caused by imbalances suitably significant to further damage cerebral cellular function via a range of ischaemic, neurochemical and ionic insults. It is a common feature in traumatic brain injury due to altered delivery of oxygen and glucose to the brain, or parts of it, due to hypoxaemia, hypotension [31]. The brain is a highly multifaceted organised system that requires a considerable amount of glucose and oxygen to survive and secondary injuries interfere with the supply of glucose and oxygen. It is housed in the skull (closed, rigid casing) which confines its' ability to compensate for expanding mass lesions, edema, swelling and haemorrhage. Although the brain is approximately 2% of the total body weight, it receives 13-15% of the cardiac output and has a 20% rate of the total body energy expenditure at rest.

Unlike other organs in the body, the brain has no oxygen reserves, has minimal supplies of high energy phosphate compounds, mainly glucose and glycogen. The brain is a well perfused organ with a blood flow of 50-60ml/100g/min. Oxygen delivery to the brain can be estimated by the product of cerebral blood flow, percentage oxygen concentration of haemoglobin and haemoglobin concentration. Arterial blood supplies an average oxygen content of 19.6mL/dL, while the internal jugular venous oxygen content is 12.9mL/dL.

The amount of oxygen extracted by the brain or the arteriovenous oxygen difference ($AVDO_2$) is 6,7mL/dL [32]. The cerebral blood flow and the total oxygen demand remain constant depending on the functional state of the brain.

Cerebral autoregulation is the ability of the brain to maintain constant perfusion over a wide range of systemic mean blood pressures, 50 to 150 mm Hg for non-hypertensive and to as high as 200 mm Hg for hypertensive people [33]. It is dependent on an intact and functional blood-brain barrier.

Autoregulation is a compensatory mechanism that maintains cerebral blood flow by causing vasodilation or vasoconstriction. The vasodilation/vasoconstriction occurs due to changes in $PaCO_2$ that are mediated by changes in pH. Carbon dioxide is a potent chemical in determining cerebral blood flow (CBF). Hypercarbia or hypercapnia leads to cerebral vasodilation which results in increased CBF. Hypocarbia or hypocapnia leads to cerebral vasoconstriction resulting in decreased

cerebral blood flow. Oxygen has a converse effect resulting in cerebral vasodilation when the PaO_2 is low and vasoconstriction when the PaO_2 is high. Acidosis results in vasodilation thus increasing CBF and alkalosis results in vasoconstriction thereby decreasing CBF.

Cerebral perfusion pressure (CPP) is defined as the blood pressure gradient across the brain which is 70 to 100 mmHg in an adult. CPP is also characterised as the difference between the mean arterial pressure (MAP) and the intracranial pressure (CPP= MAP-ICP). Significant decreases in the cerebral perfusion pressure result in brain tissue ischemia and failure in autoregulation. The brain then becomes dependent on the mean arterial pressure for perfusion. Maintenance of cerebral perfusion and oxygenation is crucial in the setting of TBI as the brain is vulnerable to ischemic injury. Chesnut et al [6] noted hypoxia and hypotension (blood pressure <90 mmHg) in more than a third of TBI patients and found that they were two of the five most compelling predictors of mortality. The study also established pre-hospital hypotension as an independent predictor of poor outcome. During the first 24 hours following TBI, CBF is often half of that of normal individuals. However, a paradoxical increase in CBF might occur in some patients due to a failure in autoregulation. Low CBF is linked with poor outcome and mortality. It tends to be the lowest with diffuse injuries accompanied by significant oedema, with large subdural haematomas and when the patient has experienced hypotension [34].

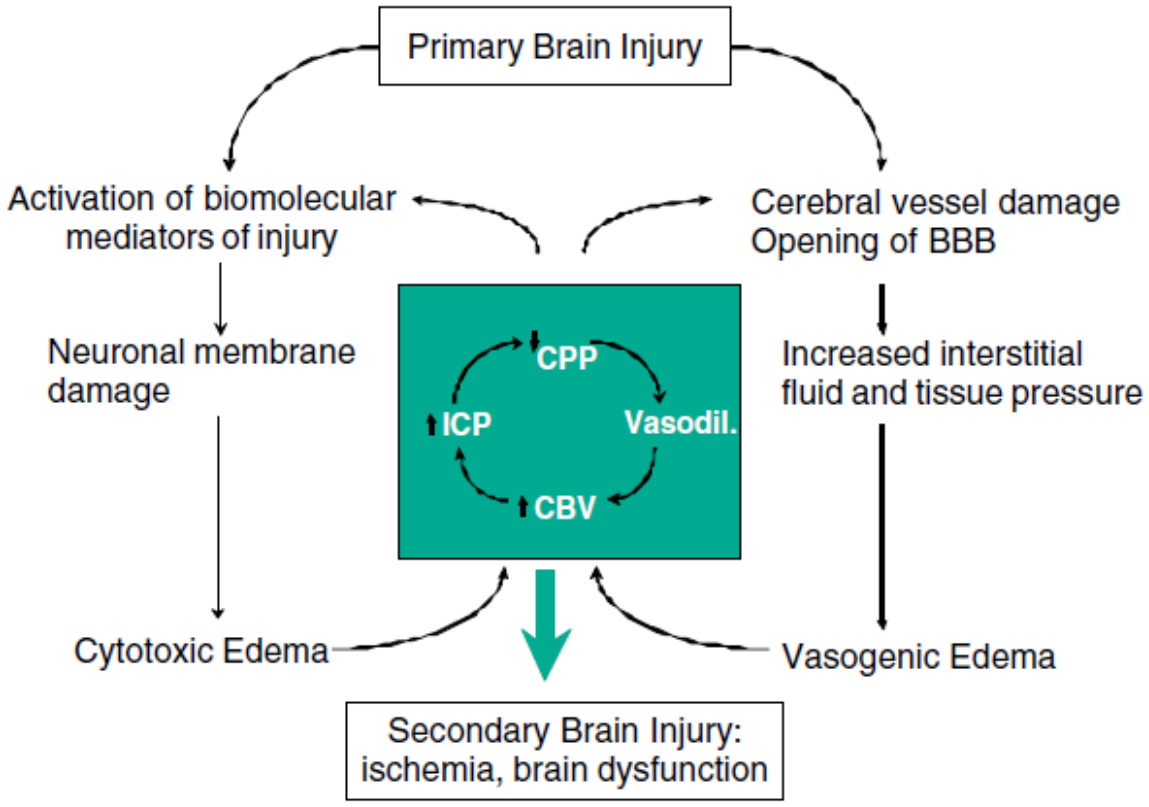

Figure 4: The cycle of primary and secondary injuries [34]

Intracranial pressure (ICP) is the pressure within the cranium. It ranges from 0-15mmHg and is kept stable by homeostatic mechanisms. Abrupt changes such as coughing or sudden volume change might influence ICP [35]. These homeostatic mechanisms controlling ICP may be altered following TBI. This can lead to a sustained, elevated ICP, resulting in neural injury and death. Brain tissue, blood and cerebrospinal fluid (CSF) were listed by Monroe and Kellie as the three components in the intracranial compartment. Because the cranium is a closed box, they further stated that the cranial compartment is incompressible with a fixed volume in the adult. The Monro-Kellie doctrine/hypothesis states that an increase in one of the constituents of the intracranial compartment must be compensated by a decrease in the other compartment/s [36].

Following TBI; the volume within the cranium increases largely due to brain tissue edema. A dramatic increase in ICP does not occur provided the compensatory mechanisms are still functional. These compensatory mechanisms include but not limited to: displacement of CSF and blood out of the cranium and decreased CSF production. However, further increases in intracranial volume during the

compensatory phase will result in an increase in ICP as the compensatory mechanisms provide a finite solution. Further increase in one of the intracranial contents beyond the compensatory ability will result in a steep rise in ICP. This is portrayed in Figure 5.

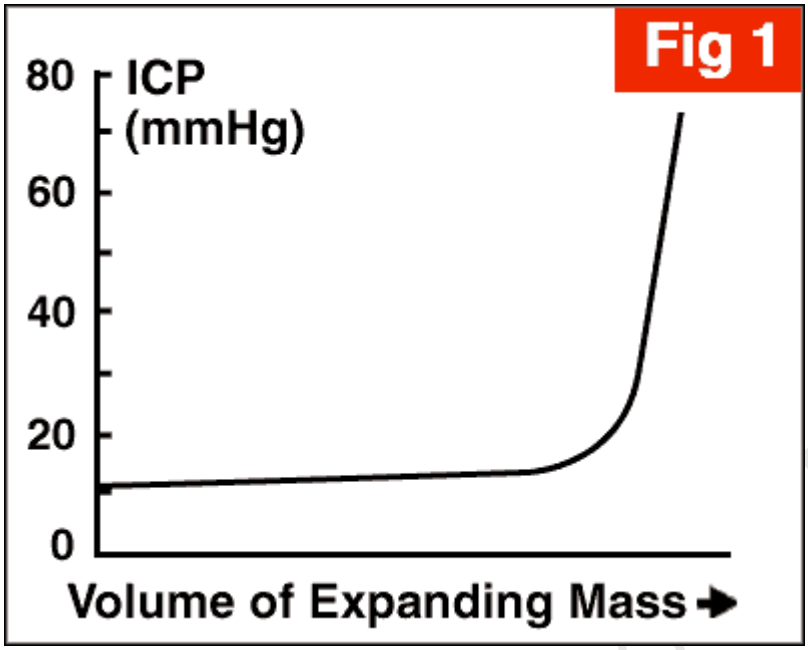

Figure 5: Intracranial pressure-volume curve [37]

Displacement of the brain from one intracranial compartment to another e.g. from supratentorial to infratentorial or vice versa is known as brain herniation, usually uncal or tonsillar. The lateral transtentorial herniation occurs when the uncus herniates between the midbrain and tentorial edge. The compression of the corticospinal tracts in the cerebral peduncle results in contralateral hemiparesis whilst compression of cranial nerve III by the herniated uncus (usually on the same side of the mass lesion) results in an ipsilateral dilated pupil. Patients also present with an altered level of consciousness and respiratory depression which is often fatal if not aggressively managed.

One of the risks associated with increased ICP is the potential to cause ischaemia by reducing CPP. The CPP falls when the ICP approaches the level of the mean systemic pressure. The response to this phenomenon would be to increase the blood pressure and dilate the cerebral blood vessels. This causes a vicious cycle as it results in increased blood volume to the brain; thus increasing ICP; further lowering CPP. Recent evidence suggests that patients with severe TBI need to maintain CPP

of 60-70 mmHg in order to reduce/prevent cerebral ischemia [38]. Studies that measured CBF in the early period of injury (4-12 hours post insult) found that patients who had a CBF of less than 16-18mL/100g brain tissue/min would ultimately die. However other studies suggest that with sustained CBF of 17mL/100g/min, irreversible ischaemia and infarction will occur [17].

Cerebral ischaemia results in interruption and damage to cell membranes and eventually to cell death. With a period of hypermetabolism and decreased perfusion that lasts for days following severe TBI, there is a change from aerobic to anaerobic metabolism in the neurons. Production of ATP is reduced, there is failure of the sodium/potassium pump; calcium, water and sodium shift into the cell and potassium flows out. Lactate is thus produced as a by-product of anaerobic metabolism which further jeopardises ionic homeostasis. Cytotoxic (intracellular) edema and acidosis occur along with the ionic imbalance [39]. Vasogenic oedema can also result following failure of the blood-brain barrier and increased capillary permeability.

2.3. Assessment

The Brain Trauma Foundation recognised blood pressure, oxygenation, pupillary exam and GCS as the four significant areas to the pre-hospital assessment of patients with severe TBI. They emphasise on monitoring and evaluation of the blood pressure, pulse oximetry, GCS score and pupil exam as these have the most impact on the management and outcome of patients with TBI.

2.3.1. Oxygenation

Pulse oximetry should be assessed continuously in the pre-hospital arena because hypoxia is a common feature of patients with severe TBI. Cooke and colleagues [40] demonstrated that 27% of 131 patients with severe TBI were hypoxic (SpO_2 <90%) on hospital admission. In Italy, Stocchetti and colleagues [41] discovered that 55% of patients with TBI were hypoxic (SpO_2 <90%) at the scene of the accident. 46% of the patients with hypoxaemia did not suffer from hypotension. In the non-hypoxic group, the mortality rate was 14.3% with 4.8% suffering from severe disability. On the other hand, the mortality rate was 50% among patients with SpO_2 of <60% and all of the survivors were found to be severely disabled. Oxygen desaturation resulted in poor outcome in both these studies and thus emphasises the importance of airway maintenance, oxygenation and ventilation in the pre-hospital setting to optimise

outcome for the patient with TBI. The Traumatic Coma Data Bank (TCDB) study prospectively enrolled 717 patients with severe TBI and investigated the detrimental influence of hypotension (defined as a single observation of systolic BP <90 mm Hg in the field) and hypoxaemia (defined as cyanosis, apnea, SpO_2 <90% or PaO_2< 60 mm Hg by arterial blood gas analysis) on these patients [6]. The TCDB study determined that hypoxaemia occurred in 22.4% of patients with severe TBI and was associated with increased mortality and morbidity.

2.3.2. Blood Pressure

The blood pressure should also be regularly assessed in the pre-hospital setting and evaluated on a frequent basis with the most precise means accessible. Independent of hypoxia, hypotension at any point during resuscitation has been shown to have a detrimental effect on outcome and increase mortality from severe TBI [42]. The TCDB discovered that pre-hospital hypotension occurred in 34.6% of the patients and was associated with a 150% increase in mortality. Patients with a single episode of hypotension had increased morbidity and twice the mortality when compared to a matched group of patients without hypotension [6]. It was also among the most influential predictors of poor outcome and was statistically independent of the others i.e. age, pupillary status, admission GCS score and intracranial diagnosis. Hypotension is generally defined as a systolic blood pressure of <90 mm Hg in adults. This value came about haphazardly and is more of a statistical than a physiologic criterion [38].

2.3.3. Glasgow Coma Score

The Glasgow Coma Score (GCS) was devised by Teasdale and Jennett [(43)] in 1974 as an assessment tool to measure the level of consciousness in acute cerebral disorders (see Table 1). Since then, the GCS has been used universally to classify severity in traumatic brain injury as mild (GCS 13-15); moderate (GCS 9-12); or severe (GCS 3-8) based on the level of consciousness. It makes use of three markers i.e. motor response, eye opening and verbal response. It is valuable to assess the initial GCS score and then measure it frequently as a decrease in the GCS score can suggest deterioration and a rise in intracranial pressure. However, the Brain Trauma Foundation [38] suggests measurement of the GCS after a clear airway has been obtained and after necessary ventilation or circulatory resuscitation

has been performed. The rationale to the afore-mentioned suggestion is the reason that the GCS is affected by anything that reduces cerebral function i.e. hypoglycaemia, hypoxia, hypotension and use of pharmacological substances. The Brain Trauma Foundation also recommends the measurement of the GCS to be done preferably prior to the administration of sedatives; paralytic agents or after the drugs have been metabolized. For these reasons, the interrater reliability of the GCS score has been called into question. Then again, Gill, Reiley and Green [44] found only moderate degrees of interrater agreement for the GCS and its components after measuring interrater reliability of the GCS in 116 patients evaluated by 2 independent blinded observers in the ED setting.

Despite its widespread acceptance and use in TBI, the GCS does not assess the precise neurological responsiveness but rather gives a gross estimate. In fact Teasdale and Jennett [45] stated that "we have never recommended using the GCS alone, either as a means of monitoring coma, or to assess the severity of brain damage or predict outcome." There is no criterion model to quantify altered mental status and it is therefore impossible to assess the accuracy of the GCS (criterion-related validity). The GCS has in actual fact been the primary criterion standard by default [46]. However one would deem it to have construct validity as it has shown statistical associations with outcomes such as mortality [47], Glasgow Outcome Score [48] and the need for neurosurgical interventions [49]. In the face of this evidence, it is still not comprehensible that the GCS can accurately predict these precise outcomes in any given patient [47].

	Eye response	Verbal response	Motor response
1.	No eye opening	No verbal response	No motor response
2.	Eye opening to pain	Incomprehensible sounds	Spastic extension to pain (decerebrate posturing)
3.	Opening to speech	Inappropriate words	Spastic flexion in response to pain (decorticate posturing)
4.	Eyes opening spontaneously	Confused	Withdraws from pain
5.		Oriented	Localizes to pain
6.			Obeys commands

Table 1: The Glasgow Coma Scale

2.3.4. Pupil Examination

The pupils should be assessed in the pre-hospital setting to gain valuable information pertaining to diagnosis, treatment and prognosis. The Brain Trauma Foundation [38] recommends measurement of the pupils after the patient has been resuscitated and stabilised. Both pupils should be assessed separately for symmetry, size and reaction to light and to specify if it is unilateral or bilateral fixed, dilated pupil(s). Symmetry in normal pupils should be < 1-mm difference and should act in response to bright-light stimulus. A fixed pupil is characterized as < 1mm response to bright light [38]. The normal light reflex depends on properly functioning cranial nerves II and III, brainstem, retina and lens.

Increased intracranial pressure may result in uncal herniation which can result in compression of cranial nerve III. This can lead to a decrease in parasympathetic tone to the pupillary constrictor fibres, resulting in a dilated pupil with diminished reactivity. Complete paralysis of the third cranial nerve leads to a fixed and dilated or blown pupil. Non-reactive and bilaterally dilated pupils may be caused by direct brain stem injury; distinct elevations of ICP which are predictive of increased mortality or bilateral compression cranial nerve III as seen in central transtentorial

herniation [50]. The normal reflex can also be altered by metabolic effects such as hypothermia, hypotension, hypoxia and use of alcohol which can also lead to abnormal reactivity and/or dilated pupil. It is for these reasons that the resuscitation and stabilisation of the patient is advocated prior to the assessment of pupillary function [51].

2.4. Predicting Outcome after Traumatic Brain Injury

2.4.1. Glasgow Coma Score

The GCS has been used as a tool to predict outcome in TBI, though Teasdale and Jennet [48] contended that the GCS alone is not a good predictor of outcome as it only measures one aspect of the physiological dysfunction occurring in patients with coma. They combined the GCS with age and abnormalities of the brainstem reflexes to predict outcome in traumatic brain injury. Baxt and Moody [52] compared the mortality of 128 consecutive severely brain-injured patients (Glasgow Coma Score less than or equal to 8) treated by a land advanced life support system and transported to a trauma center against the mortality of 104 consecutive severely brain-injured patients treated during the same time period by an advanced care rotorcraft aeromedical emergency service and transported to the same trauma center. The prognostic value for mortality for patients with a GCS of 3-5 was 50% and 61% for the rotorcraft and land advanced life support respectively. The prognostic value for patients presenting with a GCS of 6- 8 was 34.5% and 40.7% for the rotorcraft and land advanced life support respectively. However the GCS for patients treated by the land advanced life support system was only calculated upon arrival in the Emergency Department and not on the field by the paramedics. This was a serious confounder as the patients might have received sedation, analgesia, or paralysis prior to their arrival, which could significantly obscure the GCS.

Calculating the GCS may prove somewhat problematic when it comes to patients that have immobilised limbs due to fractures, intubated or present with swelling of the eyelid/s making eye opening impossible. The TCDB study randomly decided to allocate a GCS verbal score of 1.1 (strategy of pseudoscoring) to patients that were intubated prior to the assessment of the GCS. This practise however may significantly overrate the severity of the injury and questions the reliability and validity of the scale. When a verbal score of 1 was applied due to endotracheal intubation,

Gale and colleagues [53] established a mortality rate of 65% for patients with a GCS score of 3-5, while it was 88% for those with a true (testable) GCS score of 3-5. In their study, Starmark et al [54] discovered that the verbal response was the most untestable facet accounting for 58% of their patients while the eye response was not testable in 7%. The motor response was testable in 100% of their patients.

Despite the growing concerns with the GCS, it has been shown to have a substantial correlation with patient outcome following severe TBI, both as a sum score [55] or just as the motor component score [56]. In their prospective study, Narayan et al [57] found that patients with a GCS score of 3-5 had a 77% positive predictive value for a poor outcome which was characterised by a Glasgow Outcome Score of 1-3. Patients with a GCS score of 6-8 were found to have a positive predictive value of 26%.

Then again, in a prospective study of 315 consecutive patients with a severe head injury, Fearnside and colleagues [58] established a significant inverse correlation between the initial GCS score (obtained 6-48 hours post-injury) and mortality.

Gennarelli et al [59] examined the association between GCS scores of 3-15 and mortality in their large work of 46, 977 head-injured patients. They noted that patients who were admitted to the emergency room with a GCS score of 3-8 had a sharp progressive increase in mortality. Kuday, Uzan and Hanci [60] discovered that the initial GCS score was the single most important factor affecting outcome in their study of 115 patients with epidural haematomas. Notwithstanding the conflicting evidence, internationally the GCS score remains the assessment of choice for standardising measurement of the level of consciousness of patients.

2.4.2. Age

Several studies have highlighted the significance of age in the prediction of outcome in patients with traumatic brain injury [61, 62]. The outcome in these investigations varies widely with the mortality ranging from 9% to 75% in severe traumatic brain injury [63]. Most of these studies have emphasised that younger patients have a better prognosis than adults following severe TBI. This was noted as early as 1973 by Hernesniemi [64] in his analysis of 420 patients. The immature brain appears to tolerate anoxia and hypoxia better than adults [63]. Children have a lower cerebral perfusion pressure due to the low blood pressure. They also form less oedema and can rapidly clear any oedema that develops better than adults.

With this in mind, there appears to be no defining point where prognosis significantly worsens. Some authors report that children under five years have a higher mortality rate [65, 66, 67] than children under ten years of age as discovered by Zuccarello et al [68] and Mazza et al [69] who found a better prognosis in patients under ten years of age in their study of 62 children with traumatic extradural haematomas. With advancing age, the elderly are more prone to systemic diseases. The TCDB study discovered an increased proportion of poor outcomes (death and vegetative) in adult patients with pre-existing systemic diseases in those above age 56 (86% vs 50%). In their retrospective review of 672 head-injured patients, Mamelak et al [70] found age to be the most important predictor of outcome, followed by best motor score and pupillary reactivity. Current literature points to age >40 having poorer outcomes [71].

2.4.3. Pupillary Diameter and Light Reflex

There is no published literature in the pre-hospital setting investigating the association between pupillary findings and outcome following severe TBI. However, there are some authors that have suggested a strong relationship between pupillary findings and outcome in the in-hospital setting [20, 57, 72].The pupillary light reflex is an indirect measure of brain stem injury and herniation as compression of the third cranial nerve results in a fixed and dilated pupil. Jiang et al [50] and Signorini et al [73] discovered a strong relationship between fixed, dilated pupils and ultimate mortality.

Normally, an ipsilateral fixed and dilated pupil suggests lateral transtentorial herniation, while bilaterally fixed and dilated pupils are consistent with central transtentorial herniation in a fully resuscitated patient [74]. A pupil that is dilated and nonreactive secondary to a direct blow to the orbit presents a confounder in relation to prognosis in patients without the compression of the intracranial third nerve or the brainstem. Of the patients with severe TBI, 65% usually have responsive pupils following resuscitation, 12% have one abnormal pupil and 28% are found to have bilateral non-reactive pupils [74]. Braakman et al [72] investigated prognostic features on 305 consecutive head-injured patients and discovered a 90% mortality on patients with bilateral absent pupillary light reflex. Comparably, in a prospective study of 133 severe head-injured patients, Narayan et al [57] found that 35% of the patients had bilateral absent pupillary light reflex and 70% of these patients were found to have a poor outcome (dead, vegetative or severely disabled).

The timing of surgical intervention and the underlying pathology play a key role on outcome from bilateral non-reactive pupils. Phonprasert et al [75] found the mortality with bilateral fixed pupils in only 56% stuporous patients from epidural haematoma contrast to an average of 88% in patients with subdural haematomas [76.]

2.4.4. Computed Tomography Scan Features

The computed tomography (CT) scan remains the assessment of choice in patients following TBI. Findings at baseline can provide useful prognostic information post-insult. The CRASH trial found the existence of obliteration of the third ventricle or basal cisterns on the CT to be correlated with a poor prognosis at 14 days. Maas et al [77] found similar findings when they found that inexistent basal cisterns are the most compelling predictors of mortality at six months. On the other hand, Wardlaw et al [78] found a subarachnoid bleed to be the strongest predictor of mortality after CT.

In 1991, Marshall classified CT scan features of TBI patients in six groups based on the morphological anomaly of the CT scan. This CT classification distinguishes between patients with and without a mass lesion and further categorises patients with a diffuse brain injury into four groups mainly: Diffuse I, II, III and IV.

Patients with type I diffuse injuries had the lowest mortality rate at 10% followed by type II, III and IV who had mortality rates of 14%, 34%, 56% respectively.

According to the TCDB series, patients with a subdural haematoma have a 50% mortality which is related to the GCS on admission, age, presence of pupillary abnormality, period from injury to treatment [79]. Patients with subdural haematomas approximately 18mm thick and with a midline shift of 20mm have a survival rate of 50% which decreases to 0% when the midline shift is greater than 25mm [80].

The neurological status at the time of surgery is indicative of outcome from patients with epidural haematomas. This ranges from 0% for non-comatosed patients, 9% for comatosed patients and 20% for patients in deep coma. GCS or motor score on admission, age, pupillary abnormalities, immediate coma or lucid interval, period from injury to treatment and the degree of midline shift are all essential parameters that are related to outcome.

2.5. South African Pre-hospital Environment

The South African pre-hospital milieu has evolved rapidly over the last two decades from “scoop and go” to rapid response units and aeromedical transportation of the patient with severe TBI. The emergency medical training has also seen the introduction of the National Diploma and Baccalaureus Technologiae in Emergency Care being offered at the four universities of technology located in Cape Town, Bloemfontein, Durban and Johannesburg.

There are mainly three levels of pre-hospital care in South Africa .i.e. basic life support (BLS), intermediate life support (ILS) and advanced life support (ALS). The majority of pre-hospital care providers registered with the HPCSA Professional Board of Emergency (PBEC) are graded as BLS [81]. It is speculated that there are more non-registered BLS providers in the country due to a lack of job prospects. The BLS practitioner is referred to as the Basic Ambulance Assistant (BAA), having completed one month training at an accredited institution. Their scope of practice is very limited to basic airway management, cardiopulmonary resuscitation, use of an automated external defibrillator (AED) and basic trauma management. Activated charcoal, oral glucose powder/gel, medical oxygen and nitrous oxide are the only drugs in their scope of practice.

The BAA can qualify for the ILS course which is a minimum of 12 weeks after completion of a 1000 experiential hours. The ILS is also called an Ambulance Emergency Assistant (AEA). In addition to the BLS practitioner, the AEA can insert an intravenous line, perform needle cricothyroidotomy, chest decompression, administer dextrose 50%, oral aspirin, B_2 stimulants for asthmatic patients and use a manual defibrillator.

The AEA is then eligible to enter the 9/10 month Critical Care Assistant course (CCA) after a further 1000 hours of clinical experience which qualifies the provider as an advanced life support practitioner (ALS). Following which they are registered as independent practitioners with the HPCSA and function within their designated scope of practice. However some services have a medical officer/s on standby for medical advice and support to the paramedics. Endotracheal intubation and the use of supraglottic devices are some of the skills in the advanced life support protocols from the HPCSA; giving them the option of being able to secure the airway and optimizing

oxygenation and ventilation in the patient with severe TBI. However there is no published data in the South African pre-hospital setting determining the outcome of these critically ill patients following advanced airway management by South African advanced life support practitioners. A survey conducted by the World Health Organisation (WHO) concluded that there was insufficient evidence for several of the common pre-hospital interventions [82].

In the Cape Town Metropole, Midazolam and Morphine are the most commonly used pharmacological agents utilised by paramedics to intubate patients with severe TBI. Morphine is usually administered for its synergism with benzodiazepines. On the other hand, airway reflexes may still be present following the administration of these agents thus increasing the risk of aspiration. They also have the potential to cause hypotension and respiratory depression when administered rapidly and in large doses. Chesnut et al [6] have demonstrated the detrimental influence of hypotension and hypoxia in severe traumatic brain injury; even a single episode of hypotension was found to increase morbidity and double the mortality when compared to a matched group of patients without hypotension. The paramedics that have completed the Baccalaureus Technologiae: Emergency Medical Care and hold registration with the HPCSA Professional Board of Emergency Care as an Emergency Care Practitioner (ECP) can perform rapid sequence intubation (RSI) in the patient with severe TBI. There is no published data in the South African pre-hospital setting to determine the efficacy of pre-hospital RSI in the management of TBI.

Advanced life support for the TBI patient in the Western Cape, Metropole is provided largely by practitioners working in both the public and the private sector. There majority of these ALS practitioners work in the public sector. Paramedics are dispatched by the control centre to patients with severe TBI. More often than not the incidents come to the control centre as a motor vehicle accident or assault. The incidents are priority one cases which means the nearest available ambulance needs to be dispatched without delay. Upon diagnosis of TBI, the ALS provider should transport the patient directly to a facility with neuro-imaging capabilities should the following be present; any loss of consciousness, amnesia of events, seizure since the injury, persisting headache, any focal neurological deficit, GCS < 15 at any time since the injury and/or any suspicion of a penetrating head injury or skull fracture

[83].The two public tertiary hospitals in Cape Town are Tygerberg (TBH) and Groote Schuur Hospital (GSH).

2.6. Emergency Services intervals and TBI outcome

The first hour after the onset of a traumatic incident is referred to as the "golden hour". This term means that definitive trauma care must be rendered within an hour after a traumatic injury. The concept has been taught for many decades with the credence that the quicker the patient gets to definitive care, the better the outcome. However there is little or no evidence to support this hypothesis. Newgard et al [84] evaluated the association of EMS intervals and mortality among 3,656 trauma patients. They found no association between EMS intervals and mortality. The Ontario Prehospital Advanced Life Support (OPALS) major trauma study enrolled 2,867 patients to determine the impact of advanced life support on survival and morbidity of trauma patients [85]. The study did not find a decrease in mortality or morbidity for patients with major trauma. On the other hand, they found mortality to be higher for patients with GCS scores less than 9 in the advanced life support phase in comparison to the basic life support phase. This may be attributed to the 'stay and play' approach by the ALS practitioners who may delay life-saving transport to the nearest appropriate facility.

According to data from the Western Cape Metro Ambulance Service control centre, the service received an average of 25, 627 calls a month in the first six months of 2011. They also completed an average of 5, 925 priority one calls a month with the response time under 15 minutes at an average of 62%.Out of the total priority one calls done between January and June 2011, 1, 935 trauma patients were transported to GSH and TBH which are the only two public hospitals with 24-hour neuroimaging facilities in the Western Cape, Cape Town. However, the amount of patients treated for a severe traumatic brain injury in the Cape Town Metropolitan area is unknown.

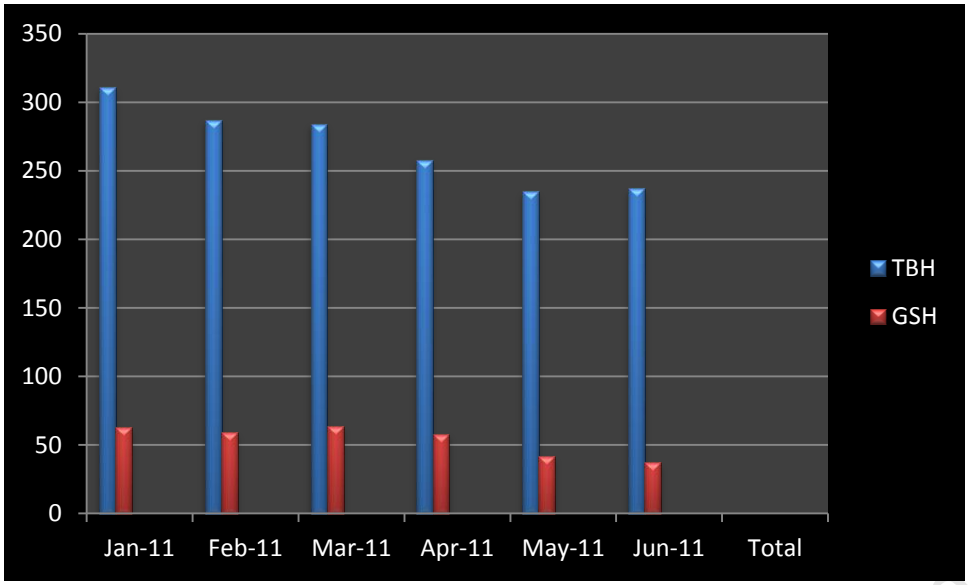

Figure 6: Number of trauma patients admitted to GSH and TBH after priority one calls

2.7. Pre-hospital Airway Management in TBI

The pre-hospital phase is arguably the most vital period in determining outcome following TBI. As noted earlier, brief episodes of hypoxia in severe TBI are strongly associated with increased morbidity and mortality. In their prospective observational study, Stocchetti and colleagues [41] discovered that 55% of patients with TBI were hypoxic (SpO_2 <90%) at the scene of the accident. There are various methods to improve oxygenation and ventilation for the head-injured patient in the field that range through basic airway management, supraglottic airway devices, endotracheal intubation and surgical airways. For the purposes of this study, basic airway interventions, sedation-assisted intubation and rapid sequence intubation will be discussed.

2.7.1. Basic Airway Interventions

Basic airway interventions aimed at clearing the airway and administering supplemental oxygen via a face mask are easily applicable in the pre-hospital setting and are economical. There is a concern for the patient with severe TBI that oropharyngeal airway insertion might not be achievable due to a possible gag reflex or clenching jaw of the patient. However the patient can be turned to the lateral position to avoid the risk of aspiration or a nasopharyngeal airway can be inserted. Penetration of the anterior skull base has been noted by Muzzi et al [86] for patients

with a skull base fracture upon insertion of the nasopharyngeal airway thus this airway adjunct should be used with extreme caution in TBI patients. Davis et al [87] observed mean initial SpO2 values of 90% or less in patients undergoing pre-hospital intubation attempts. Nonetheless, they also found that non-invasive airway management was nearly as effective as endotracheal intubation in reversing the hypoxaemia.

2.7.2. Endotracheal Intubation

There has been a major controversy regarding the role of endotracheal intubation in the pre-hospital management of TBI. The argument for early intubation in severe TBI is to optimise oxygenation and ventilation. There is also the concern of these patients losing their ability to maintain their own airway, resulting in aspiration hence the insertion of a cuffed endotracheal tube to protect the airway. These concerns seem to have emerged from a series of investigations documenting high aspiration rates in comatosed women suffering post-partum haemorrhage, with ensuing studies documenting evidence of aspiration in fatal traumatic brain injury [88]. However, the impact of aspiration on outcome was never documented by these studies nor did they investigate the ability of intubation to prevent aspiration.

In a retrospective case control study, Winchell and Hoyt [89] found that patients with blunt injury and scene GCS of 8 or less had better survival when intubated in the out-of- hospital milieu, particularly those with severe head injury by anatomic criteria. Conversely, a few studies have found an increased mortality in patients with severe TBI undergoing pre-hospital endotracheal intubation [90 - 92]. In the largest study of pre-hospital intubation in patients with severe TBI, Wang et al [92] compared the effects of out-of-hospital endotracheal intubation versus emergency department endotracheal intubation on mortality and neurological outcome. They discovered approximately fourfold increase in odds of death in patients who were intubated out of hospital in comparison to those who received emergency department endotracheal intubation. They also discovered worse neurological outcomes associated with out-of-hospital endotracheal intubation.

Despite the various theories supporting early intubation in the patient with severe TBI, recent evidence suggests that it has got the potential to cause more harm than good. Reasons for the above are not clear whether it is complications associated

with the skill or selection bias as patients warranting intubation are inherently more severely injured. Nonetheless, the intubated group in the large study by Wang et al [92] was intubated without the use of pharmacological agents. This could shed some more light on their findings as there is overwhelming evidence that endotracheal intubation without the use of pharmacological agents results in worse outcomes compared to patients that are intubated on hospital admission. In their retrospective review of 852 patients, Murray et al [91] found that patients intubated in the pre-hospital setting or in whom intubation was attempted had an increased mortality (81% and 77%, respectively) in comparison to 43% who were not intubated on scene. Bochicchio et al [90] also found a significant increase in morbidity and mortality in patients with severe traumatic brain injury who were intubated in the pre-hospital field without the use of pharmacological agents. Patients who received pre-hospital intubation had longer ventilator days, hospital stays and intensive care unit days in comparison to those intubated on immediate arrival at the hospital. They also had 1.5 times greater risk of nosocomial pneumonia compared with patients intubated at hospital. However; these findings do not show causality.

Von Elm et al [93] conducted a systematic review on the benefit and harm of prehospital intubation in patients with TBI. They included seventeen studies with patient data collected between 1985 and 2004. Their review did not provide any support for prehospital intubation for the patient with TBI. A Cochrane systematic review by Lecky et al [94] contained three randomised controlled trials that investigated the effectiveness of prehospital intubation compared to other alternative airway management methods. The trials however were mainly on patients suffering from out-of-hospital cardiac arrest and none of them contained patients with TBI. None of the studies showed a significant difference in outcome between the treatment groups.

2.7.3. Sedation-Assisted Intubation

Sedation-assisted intubation is the administration of a sedative/induction agent prior to intubation. In theory, this technique should increase the success rate of endotracheal intubation by suppressing the gag reflex; nonetheless, this is not always possible. In a retrospective review of patients receiving sedation intubation protocol using diazepam and morphine; Sams and Kelly [95] discovered that in 45.9%

of patients, a gag reflex was still present during or after the sedation-assisted intubation. They also had a success rate of 94.5% with 73.6% of these intubations being successful at the first attempt with a median dose of 20 mg Morphine and 20 mg Diazepam.

Dickenson et al [96] embarked on a study to determine the efficacy of midazolam as a drug to facilitate intubation. Successful endotracheal intubation was found in 17 of 20 cases (85%). However, only nine patients had trauma complaints whilst the rest had medical complaints. They found a single dose of midazolam to be satisfactory to facilitate ETI in 15 of the 17 cases with a mean dose of 3.6mg. In a larger study, Wang et al [97] found a success rate in 45 of 72 cases (62.5%) with less success in trauma patients (41.2%) than for medical patients (69.1%).

In theory, sedation-assisted intubation could lead to or worsen outcomes due to secondary brain injury as a result of blood pressure changes as noted by Sams and Kelly [95] where 22% of patients had a change in blood pressure of more than 20 mmHg. There is also the fear of increasing the intracranial pressure during laryngoscopy should the patient not be fully relaxed during intubation thus further reducing cerebral perfusion. With high failure rates associated with this procedure (37.5%) as noted by Wang et al [97], there is an increased risk of aspiration as the patient might have a depressed cough reflex following the administration of the pharmacological agent/s. On the other hand, sedation-assisted intubation evades the complications of RSI should the practitioner fail to secure the airway.

2.7.4. Rapid Sequence Intubation

Rapid sequence intubation is defined as the rapid administration of an induction/sedative agent followed by the administration of a rapid-onset neuromuscular blocking agent; usually suxamethonium. Currently, the Brain Trauma Foundation [38] does not recommend intubation for ground-transported patients in urban settings whom are spontaneously breathing and maintaining pulse oximetry greater than 90%. There has been considerable debate regarding the role of intubation and the use of rapid sequence intubation in the pre-hospital management of TBI. There is a concern of the disastrous implications that failed intubation would have on the pharmacologically paralysed patient.

Of 114 patients enrolled in a study by Ochs et al [98] to evaluate the ability of paramedic rapid sequence intubation (RSI) to facilitate intubation of patients with severe head injuries; 84.2% underwent successful endotracheal intubation, 14.9% underwent Combitube insertion with only 0.9% airway failure. Klemen and Grmec [99] found that there was a decrease in the number of deaths on hospital admission, a reduction in hospital mortality in the GCS group 6–8 and an improvement in functional neurological outcome and shortened hospitalization time in patients receiving pre-hospital advanced life support versus the group of patients not receiving pre-hospital advanced life support with RSI. Bulger et al [100] established a similar improvement in patient outcomes following the use of neuromuscular blockers in the pre-hospital setting. Patients intubated using neuromuscular blocking agents had an unadjusted mortality of 25% in comparison to 37% for those not receiving RSI. When adjusted for confounders, patients intubated using RSI had a better survival rate compared to the non-RSI group.

On the other hand, a study by Davis et al [101] showed a significant increase in mortality and a decrease in good outcomes in the RSI group compared to the non-intubated patients. It is unknown what caused the increased mortality in the RSI patients but transient hypoxia, inadvertent hyperventilation as measured on arrival at the ED and longer on scene times associated with RSI might have led to a negative outcome. There is also an indication of increased morbidity associated with pre-hospital RSI. Sloan et al [102] found an increase in pulmonary complications; mainly pneumonia following pre-hospital RSI, with 28% of pre-hospital RSI patients developing pneumonia in comparison with 6% of patients intubated in the ED. Li et al [103] compared the complications of sedation-assisted intubation in 67 patients with RSI in 166 patients in an ED study. They found airway trauma in 28%, failure to intubate in 18%, aspiration in 15% and fatality in 3% in the sedation-assisted intubation patients. These complications were however not found in the RSI group.

A prospective, randomised controlled trial by Bernard et al [104] investigated whether prehospital RSI improves outcome at six months in patients with severe TBI. 312 patients were allocated randomly to either prehospital RSI or emergency department intubation. Patients who received prehospital RSI had a favourable neurological outcome of 51% in comparison to patients who were intubated in the hospital setting (39%).

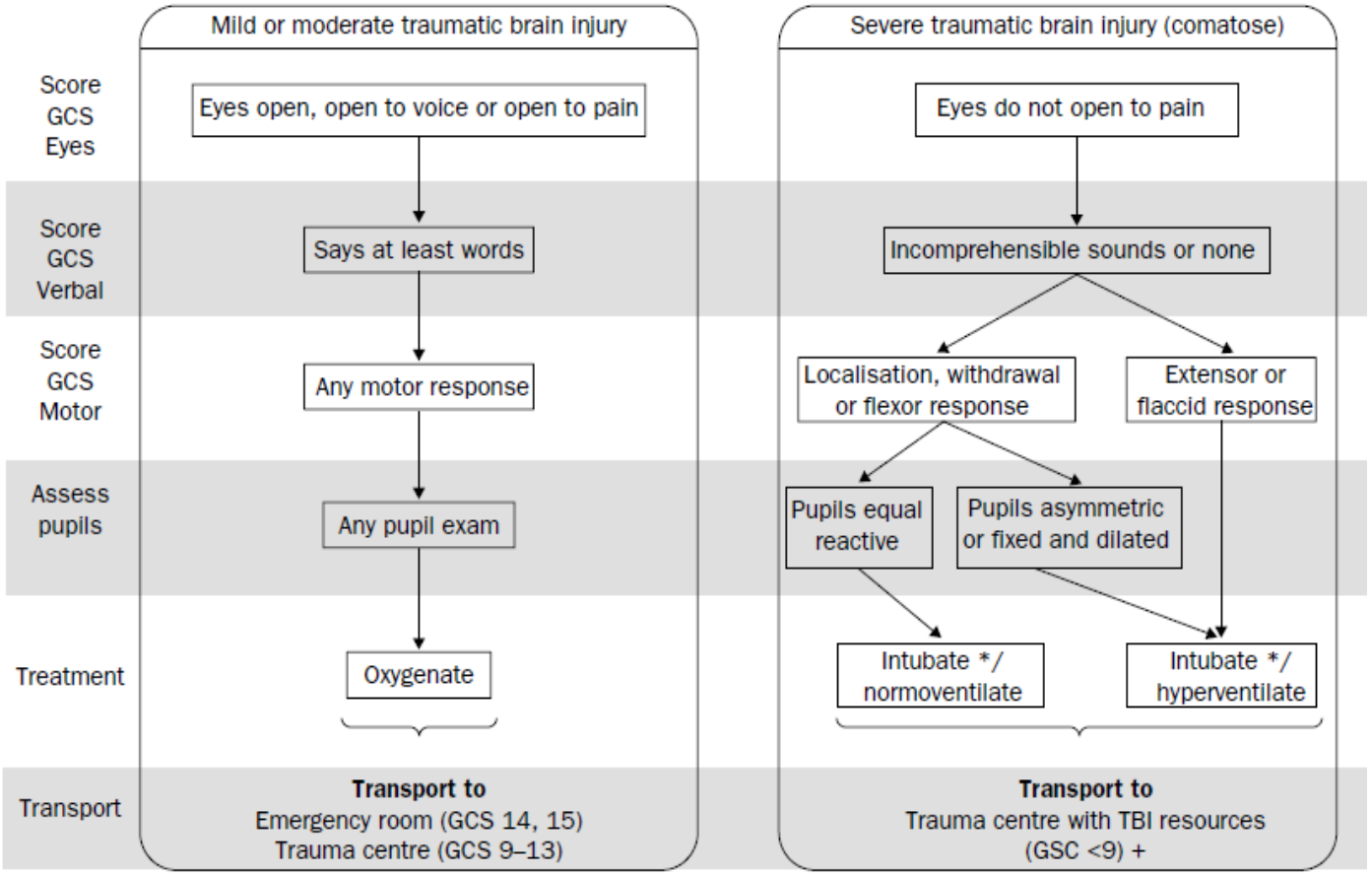

Figure 7: Pre-hospital triage of patients with traumatic brain injury [1]

2.8. In-hospital Management of TBI

The goal of in-hospital management of severe TBI is to stop and prevent the catastrophic events associated with secondary brain injury. Upon arrival of the critically-ill injured trauma patient at the tertiary hospital, the patient is admitted at the resuscitation unit where the emergency medicine registrar and trauma unit staff attends to the patient. During this crucial period, the A (airway) B (breathing) C (circulation) D (disability) and E (exposure) of trauma are dealt with.

The principles of emergency in-hospital management of the patient with TBI involves a rapid stepwise assessment of the ABCs, identifying and managing associated injuries, identifying and managing factors related to secondary brain injury and investigations to determine extent and prognosis of TBI.

These may include but are not limited to:

1. Airway assessment and placement of ETT if not previously done

2. Assess ventilation and maintain adequate oxygenation
3. Circulation: placement of an electrocardiograph to monitor for any arrhythmias; insertion of two-large bore intravenous lines if not previously done

The patient is then sent for trauma diagnostic testing to rule out any other life-threatening injuries. These are the chest and cervical spine x-ray, abdominal ultrasound followed by the CT brain. Uncontrasted CT scan is the diagnostic investigation of choice for patients with severe TBI in central hospitals in Cape Town. These are available 24 hours with radiological and neurosurgical support for interpretation. Access to MRI in public sector hospitals is limited. The trauma unit staff than await the diagnosis of the radiologist upon which the neurosurgeon is than called upon diagnosis of TBI on the CT scan to determine the definitive care.

The advanced trauma life support guidelines (ATLS) [105] advocate a target of 30 minutes from injury to CT scan. However, the investigator found a mean time of 4.8 hours to CT scan from arrival at the trauma unit after a review of 12 patients at Tygerberg Hospital. It is unknown whether this time-delay has an influence on outcome though Seelig and colleagues [106] observed a 30% mortality for patients operated within four hours in comparison with 90% mortality in individuals operated after four hours after an acute subdural haematoma. After the radiologist gives a report of the CT, the neurosurgeon is then called to decide on the course of action for the patient.

CHAPTER 03

METHODOLOGY

3.1. Introduction

This chapter presents the methodology utilised in this study with reference to the study design, study setting, data collection, data analysis, sampling and ethical considerations

3.2. Study Design

The study was a cohort descriptive observational analysis of consecutively injured adult patients who sustained severe traumatic brain injury in the Cape Town, Metropole area.

The prehospital airway management methods that were researched were:

1. Rapid sequence intubation (RSI)
2. Sedation-assisted intubation
3. Intubated without sedation
4. Basic airway management (without intubation)
5. Attempted intubation but unsuccessful (failed intubation)

3.3. Study Setting

The study setting was at the two central hospitals in Cape Town: Tygerberg and Groote Schuur hospital. These are specialist centres where patients with severe TBI are transported for further care and management. The researcher also works in Cape Town which made the data collection easier.

There aforementioned hospitals utilise standard software to document basic demographics (sex, age, and race), clinical data (time, date, location of hospital admission, length of stay and discharge status). The study used two sources of data: the EMS patient care record form and the data on the patient's file at the abovementioned hospitals.

3.4. Inclusion/Exclusion

The study included adult trauma patients (age ≥16 years) admitted to Groote Schuur and Tygerberg hospital for the treatment of closed severe TBI (GCS ≤8); suspected TBI by mechanism of injury or physical examination during the period of 01 January 2009 till 31 August 2011. The study excluded patients that were transferred to Tygerberg and Groote Schuur hospital from another facility. Patients sustaining penetrating head trauma were also excluded. Patients who were declared dead on scene were also not included in the study.

3.5. Sample size

During the period of 1 January 2009 to 31 August 2011, the investigator took all patients with severe TBI from the above-mentioned hospitals. Patients with the most complete available data were selected and 124 patients were included in this study. With these 124 patients, the investigator was able to describe the prevalence of a good outcome with the precision shown in Annexure 1; the precision will depend on what the proportion with a good outcome is in the study. The 95% confidence interval ranges from 0.1-0.23. Due to the low proportion, the confidence interval is asymmetrical.

Prior to the study it was not known how these 120 patients would be distributed according to the various methods. Annexure 2 shows the precision with which the proportions with a good outcome were described for five subsamples ranging from 10–100 patients.

In order to determine whether one method results in a significant difference in proportion of a good outcome compared to another method, the overlap of the 95% confidence interval was explored. The overlapping 95% confidence interval indicates no statistical significance between two proportions while non-overlapping indicates statistically different proportions. This was formally explored by performing a logistic regression analysis. The time to get the patient to hospital was compared in those with poor and good outcomes using the t-test.

3.6. Outcome measures

Patient survival to hospital discharge was the primary outcome determined from the patient's clinical record at the aforementioned hospitals. This was done in comparison to other studies of a similar nature [90, 92, 100]. The secondary outcomes were determined at the time of discharge using the Glasgow Outcome Scale. These were neurologic and functional status. Glasgow Outcome Score of 1-3 denoted a "poor outcome" while 4-5 suggested a "good neurological outcome".

1.	Dead
2.	Persistent vegetative state
3.	Severe disability
4.	Moderate disability
5.	Good recovery

Table 2: Glasgow Outcome Scale

3.7. Data collection

Completed folders of patients admitted to Groote Schuur and Tygerberg hospitals' resuscitation units for suspected severe TBI were reviewed for eligibility. The data was collected both retrospectively (1 January 2009 to 31 March 2011) and prospectively (1 April 2011 to 31 August 2011). Both hospitals have a trauma register at the resuscitation unit in which the patients with severe TBI were filtered from. Patients were filtered using the following criteria: working diagnosis of TBI indicated on the register, GCS ≤ 8, intubated, patient sent for CT scan. If one of the following was present, the folder was then requested at medical records for a more detailed evaluation. The primary selection was broad by design so that patients with suspected TBI were not missed. Patients were finally included if they had a confirmed TBI using a CT scan or had a prehospital GCS ≤ 8.

The following data was collected by the investigator at the two hospitals from the patient's hospital folders which also contain the EMS service patient care record form. The selection of patients was made from the most complete available data. 62 patients were then included in this study from each hospital. The CT imaging was taken from the neurosurgical report available from the patients' hospital records. A

data collection form (see Annexure 3) was created in Microsoft Access where the data was captured. The form was password protected and only accessible to the researcher.

Approval to peruse the documents was sought from the hospital management.

1. Time of arrival at scene, injury data collected on scene arrival initial GCS, mechanism of injury, sex, age, heart rate, blood pressure, haemoglucose test, pupil findings, presence of decorticate/decerebrate posture, pulse oximetry **(Available from the patient care record form)**
2. Airway management of the patient (RSI/sedation-assisted intubation/ basic airway management/attempted intubation but unsuccessful/intubated without sedation) **(Available from the patient care record form)**
3. Post airway management data (defined as data collected after the airway management procedure/en-route to hospital following the airway management of the patient) GCS, heart rate, end-tidal carbon dioxide reading (mandatory with RSI as per HPCSA regulation), blood pressure, pupil findings, pulse oximetry (**Available from the patient care record form**)
4. Time of arrival at hospital, data on hospital arrival (GCS, heart rate, respiratory rate, end-tidal carbon dioxide reading, blood pressure, CT scan and/or X-ray findings), pupil findings, pulse oximetry **(Emergency Centre notes)**
5. Prehospital transport times were taken from the patient care record form which were all calculated in minutes
    - Response time was defined as the interval from dispatch to the arrival of the ambulance crew on scene
    - Scene Time was defined as the period from arrival of the ambulance on scene till departure at the scene
    - Transport time was defined as the period from departure of the scene till arrival at the hospital
    - Total time was defined as the interval from dispatch to arrival at hospital
6. Data on hospital discharge (length of hospital stay, length of ICU stay, discharge destination, neurological and functional outcome at discharge)

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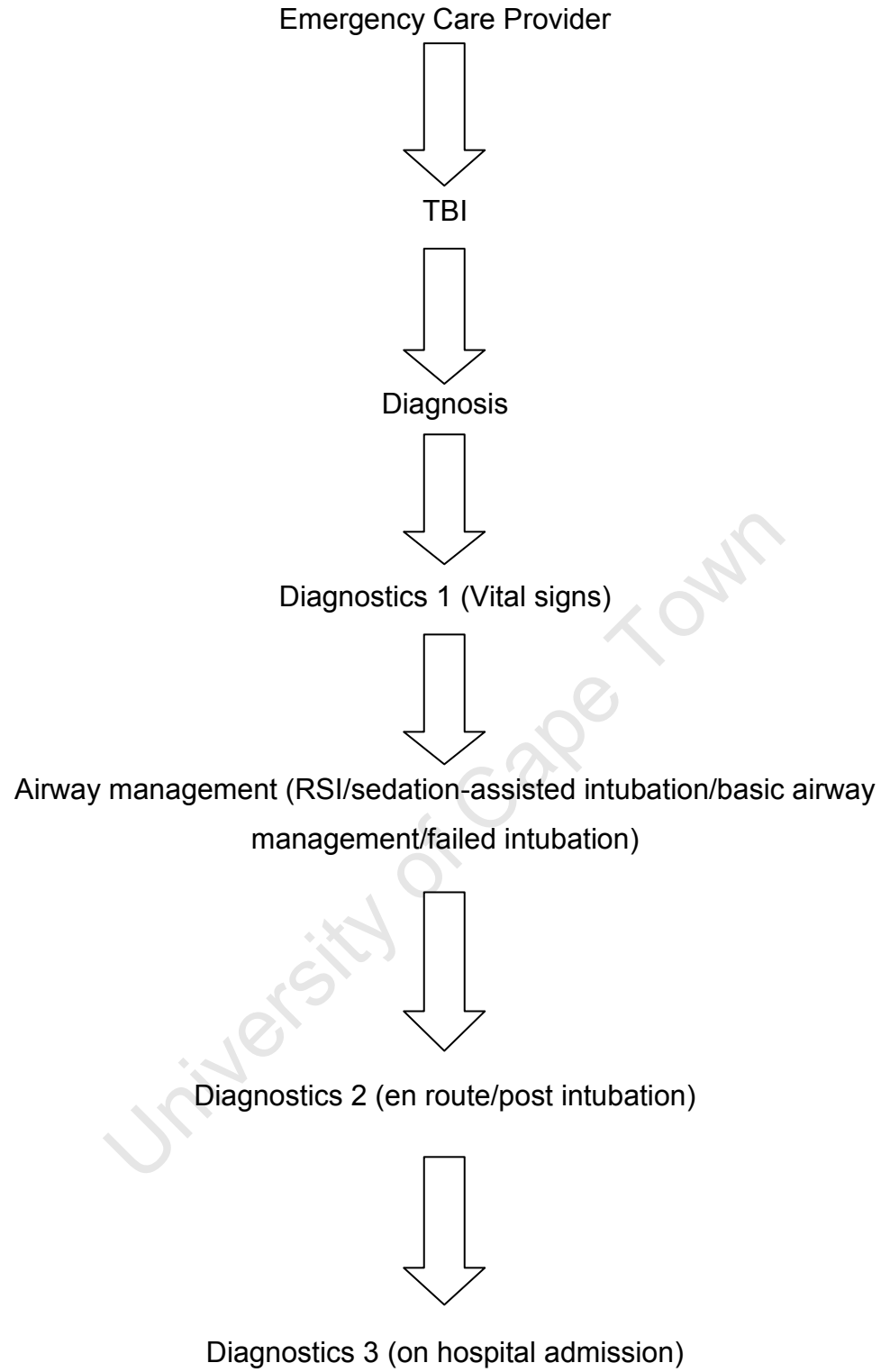

3.8. Data analysis

Data was analysed using SPSS (Statistical Programs for Social Sciences). The sample was described using basic frequencies and descriptives. Outcome measures were compared univariately against a selection of independent variables. Lastly the outcome measures were modelled using a binary logistic regression adjusting for age, GCS, pupillary reaction, and initial SpO_2. The Pearson chi-square and Fisher's exact test were used where appropriate with statistical significance set at $p < 0.05$.

3.9. Ethical Considerations

The patients were numerically coded to ensure anonymity throughout the entire research process, so that no names were to appear on the data collection sheet or in the result report forms. Patients were not contacted nor was their care in the prehospital or hospital setting affected by the study, thus no consent was needed. Emergency care provider details were not recorded on the data collection sheet. Permission from the hospital management was sought after ethics approval from the University of Cape Town human research ethics committee (HREC REF: 096/2011) and prior to commencement of the data collection.

CHAPTER 04

RESULTS

The previous chapter discussed the methodology utilised in this study with reference to the data collection and data analysis. This chapter describes the results that have emanated from the analysis by using basic frequencies and descriptives. The times in the study are presented in minutes. Results of the outcome were determined at discharge.

4.1. Hospital Distribution

		Frequency	Percent
Hospital	GSH	62	50
	TBH	62	50
	Total	124	100

Table 3: Frequency and percentage of patients from GSH and TBH

Table 3 illustrate the frequency and the percentage of patients in the study population admitted to GSH and TBH following TBI.

4.1.1. Patient Characteristics

4.1.2. Gender

		Frequency	Percent
Gender	Male	110	88.7
	Female	14	11.3
	Total	124	100

Table 4: Gender distribution among the study population

Table 4 demonstrate the gender distribution among the study population. Males made up the larger proportion in the study (n=110; 88.7%) compared to females (n=14; 11.3%).

4.1.3. Age

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Age	124	100.00%	0	0.00%	124	100.00%

Descriptives

			Statistic	Std. Error
Age	Mean		32.32	1.011
	95% Confidence Interval for Mean	Lower Bound	30.32	
		Upper Bound	34.32	
	5% Trimmed Mean		31.74	
	Median		30	
	Variance		126.63	
	Std. Deviation		11.253	
	Interquartile Range		16	
	Skewness		0.814	0.217

Table 5: Mean age of the study patients

The basic descriptives for the mean age is depicted in Table 5. The mean age of the study population was 32. The true population mean age falls within 30.32 and 34.32 with 95% confidence.

4.2. Mechanism of injury of the study population

		Frequency	Percent	Valid Percent
	Assault	30	24.2	24.4
	Fall from	9	7.3	7.3
	RTA	82	66.1	66.7
	Train casualty	2	1.6	1.6
	Total	123	99.2	100
Missing	1	1	0.8	
Total		124	100	

Table 6: Mechanism of injury of the study population

The distribution of the cause of injury in the study population is illustrated in Table 6. The major cause of injury in the study was road traffic accidents (n=82; 66.7%), followed by assaults (n=30; 24.4%); falls (n=9; 7.3%) and train casualties (n=2; 1.6%).

4.2.1. Distribution of airway management methods among the study population

		Frequency	Percent
	Basic airway management	37	29.8
	Failed intubation	11	8.9
	Intubated without drugs	8	6.5
	RSI	13	10.5
	Sedation-assisted intubation	55	44.4
	Total	124	100

Table 7: Distribution of airway management methods among the study population

Sedation-assisted intubation was the airway management method that was performed the most (n=55; 44.4%); followed by basic airway management (n=37; 29.8%). RSI was performed 13 times in the study (10.5%). There were 11 failed intubations (8.9%) recorded in the study. Three practitioners resorted to using the laryngeal mask airway (2.4%) as the bailout device after the failed airway while the

rest continued with bag-valve mask ventilation. Eight patients (6.5%) were intubated without the use of pharmacological agents. This is illustrated in Table 8.

4.2.2. Outcome and Mortality

		Frequency	Percent
	Dead	48	38.7
	Discharged	74	59.7
	Transferred to other hospital	2	1.6
	Total	124	100

Table 8: Discharge destination of the study population

4.2.3. Discharge Destination

Table 8 present the discharge destination of the study population. 74 patients in the study were discharged (59.7%), 49 were discharged home (39.5%), 24 were discharged to a rehab centre (19.4%), 2 were transferred to another hospital (1.6%). 48 patients died following hospital admission (38.7%).

4.2.4. Glasgow Outcome Scale (GOS)

		Frequency	Percent
GOS	Dead	48	38.7
	Severe Disability	2	1.6
	Moderate Disability	33	26.6
	Good Recovery	41	33.1
	Total	124	100

Table 9: Outcome of the study population using the Glasgow Outcome Scale upon hospital discharge

The mortality rate for the study population was 38.7% (n= 48), all of which occurred following hospital admission. 41 patients had a good recovery (33.1%) while 33 (26.6%) had a moderate disability with complaints of various grades. The remaining 2 survivors had a severe disability upon hospital discharge (1.6%). This is illustrated in Table 9.

4.2.5. Prevalence of a Good Outcome

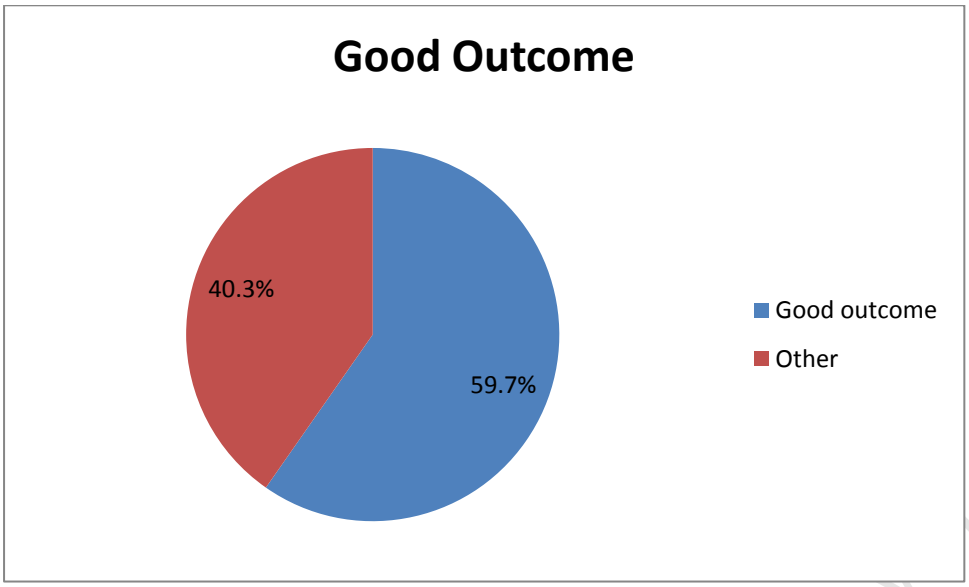

Figure 8: Prevalence of a good outcome in the study population

The prevalence of a good outcome in this study was 59.7% (n= 74). This is portrayed in Figure 8.

4.3. Airway Management and Outcome

		Outcome		Total
		Poor	Good	
Airway Management	Basic airway management	10	27	37
	Failed intubation	4	7	11
	Intubated without drugs	7	1	8
	RSI	8	5	13
	Sedation-assisted intubation	21	34	55
Total		50	74	124

Table 10: Association of airway management methods and outcome

Table 10 presents the cross tabulation for outcome and airway management. It is important to note that basic airway management had the highest proportion of a good outcome (72.9%) followed by the failed intubation group. Patients intubated without drugs had the highest proportion of a poor outcome (88%) followed by the RSI group (62%). A good outcome of 62% and 38.4% was the result in patients where sedation-assisted intubation and RSI was employed respectively. Patients intubated without drugs had a 12.5% proportion of a good outcome.

With 95% confidence, there Fisher's exact test found an association between airway management and outcome in this study (p = 0.013).

4.3.2. Mean Systolic Blood Pressure for Sedation-assisted intubation

		Mean	N	Std.	Std. Error Mean
	Arrival Systolic	123.28	54	25.185	3.427
	Systolic post airway management	124.93	54	25.575	3.48

Table 11: Mean Systolic Blood Pressure for Sedation-assisted intubation

Table 11 illustrates that there is no significant drop in the mean systolic blood pressure from arrival to post airway management (p= 0.434). As a matter of fact, there is a slight increase in the mean systolic blood pressure.

4.4. Scene times for each airway management method

Airway management method	N	Mean (Minutes)	Std. Deviation	Variance	Std. Error of Mean
Basic airway management	35	22.46	9.385	88.079	1.586
Failed intubation	8	28.88	10.274	105.554	3.632
Failed intubation-LMA	3	42	3.606	13	2.082
Intubated without drugs	7	26.29	13.388	179.238	5.06
RSI	12	33.67	16.03	256.97	4.628
Sedation-assisted intubation	53	40.08	18.464	340.917	2.536
Total	118	32.67	16.697	278.804	1.537

Table 12: Basic descriptives indicating the scene times for each airway management method

Airway Management	N	Mean (Minutes)	Std. Deviation	Variance	Std. Error of Mean
Basic airway management	35	22.46	9.385	88.079	1.586
Failed intubation	11	32.45	10.680	114.073	3.220
Intubated without drugs	7	26.29	13.388	179.238	5.060
RSI	12	33.67	16.030	256.970	4.628
Sedation-assisted intubation	53	40.08	18.464	340.917	2.536
Total	118	32.67	16.697	278.804	1.537

Table 13: Basic descriptives indicating the scene times with each airway management method

Table 12 and 13 demonstrate the time spent on scene with each airway management method. Patients who were intubated using Midazolam and/or Midazolam and Morphine Sulphate spent the most time on scene; 40 minutes (n=

53). Patients who underwent basic airway management spent the least amount of time on scene; 22 minutes (n= 22) followed by patients who were intubated without drugs; 26 minutes (n= 7). The mean scene time for RSI was 33 minutes (n= 12) and 3 patients who had a laryngeal mask airway (LMA) as a result of a failed airway had a scene time of 42 minutes while those who had a failed airway with no supraglottic device had a scene time of 28 minutes (n= 8). There was 6 missing data for prehospital transport times. Thus data reflecting prehospital transport times will have a total of 118 patients.

4.4.1. Total time for each airway management method employed in the study

Airway management method	N	Mean (Minutes)	Std. Deviation	Variance	Std. Error of Mean
Basic airway management	35	51.14	18.335	336.185	3.099
Failed intubation	8	58.25	21.312	454.214	7.535
Failed intubation-LMA	3	74.67	23.029	530.333	13.296
Intubated without drugs	7	63.43	12.765	162.952	4.825
RSI	12	61.25	25.602	655.477	7.391
Sedation-assisted intubation	53	70.25	23.038	530.766	3.165
Total	118	62.56	22.601	510.813	2.081

Table 14: Mean total time for each airway management method employed in the study

Airway Management	N	Mean (Minutes)	Std. Deviation	Variance	Std. Error of Mean
Basic airway management	35	51.14	18.335	336.185	3.099
Failed intubation	11	62.73	21.973	482.818	6.625
Intubated without drugs	7	63.43	12.765	162.952	4.825
RSI	12	61.25	25.602	655.477	7.391
Sedation-assisted intubation	53	70.25	23.038	530.766	3.165
Total	118	62.56	22.601	510.813	2.081

Table 15: Mean total time for each airway management method employed in the study

Table 14 and 15 depict the descriptives for the mean total time for the various airway management methods employed on the scene during the study. The group that underwent basic airway management had the quickest mean total time of 51 minutes (n= 35) followed by the failed intubation group with 58 minutes (n= 8). Patients who underwent RSI had a mean total time of 61 minutes (n= 12) and the group who had an LMA in-situ due to a failed intubation had a mean total time of 74 minutes (n= 3). The group that encountered sedation-assisted intubation had a mean total time of 70 minutes (n= 53).

4.5. Mean Prehospital Transport Times

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Transport times	118	95.20%	6	4.80%	124	100.00%

4.5.1. Response Time

			Statistic	Std. Error
Response time	Mean (Minutes)		10.43	0.638
	95% Confidence Interval for Mean	Lower Bound	9.17	
		Upper Bound	11.7	
	5% Trimmed Mean		9.84	
	Median		10	
	Variance		48.008	
	Std. Deviation		6.929	
	Interquartile Range		8	
	Skewness		1.726	0.223
	Kurtosis		5.169	0.442

Table 16: Mean response time of the study population

Table 16 presents the basic descriptives for the response times in the study. The mean response time of the study population was 10.43. The true mean falls within 9.17 and 11.70 with 95% confidence.

4.5.2. Scene Time

			Statistic	Std. Error
Scene time	Mean (Minutes)		32.67	1.537
	95% Confidence Interval for Mean	Lower Bound	29.63	
		Upper Bound	35.71	
	5% Trimmed Mean		31.42	
	Median		31.5	
	Variance		278.804	
	Std. Deviation		16.697	
	Interquartile Range		19	
	Skewness		1.323	0.223
	Kurtosis		3.292	0.442

Table 17: Mean scene time of the study population

The basic descriptives for the mean scene time is illustrated in Table 17. The mean scene time in the study was 32.67. At 95% significance, the true mean falls within 29.63 and 35.71.

4.5.3. Total Time

			Statistic	Std. Error
Total Time	Mean (Minutes)		62.56	2.081
	95% Confidence Interval for Mean	Lower Bound	58.44	
		Upper Bound	66.68	
	5% Trimmed Mean		61.29	
	Median		60	
	Variance		510.813	
	Std. Deviation		22.601	
	Interquartile Range		32	
	Skewness		0.734	0.223
	Kurtosis		0.093	0.442

Table 18: Mean total time of the study population

The basic descriptives for the total time is depicted in Table 18. The mean total time of the study population was 62.56. The true mean of the total prehospital transport time falls between 58.44 and 66.68 with 95% confidence.

4.6.1. Response Time 15 minute cut-off

		Frequency	Percent
Valid	Under 15 minutes	92	78
	15 minutes or more	26	22
	Total	118	100
Missing	System	6	
Total		124	

Table 19: Response Time 15 minute cut-off

The frequencies for response-times under 15 minutes are demonstrated in Table 19. An emergency care practitioner was present on scene in less than 15 minutes in 78% of the cases. 22% missed the 15 minute cut-off response time.

4.6.2. Response Times with 15 minute-cut off and Outcome

		Outcome		Total
		Poor	Good	
Response Time	Under 15 minutes	41	51	92
	15 minutes or more	7	19	26
Total		48	70	118

Table 20: Association between response times and outcome

48 patients had a poor outcome in the study. 41 out of the 48 patients had an ambulance on scene in less than 15 minutes while the other 7 missed the 15 minute cut-off response time. 70 patients had a good outcome and 73% had a response time under 15 minutes. 27% of the patients with a good outcome had a response time above 15 minutes. This is illustrated in Table 20.

There Pearson chi-square test was done to determine an association between the two variables. There appears to be no correlation between response times and

outcome in TBI (p = 0.106). Odds of a poor outcome when response time is under 15 minutes relative to poor outcome when the response time is over 15 minutes is 2.18; 95% confidence interval (CI) 0.77 to 6.38.

4.6.3. Total Time with 60 minute-cut off and Outcome

		Outcome		
		Poor	Good	Total
Total time	Less than 60 minutes	24	32	56
	60 minutes or more	24	38	62
Total		48	70	118

Table 21: Association between total time under 60 minutes and outcome

42.9% (n= 24) of the patients that were in hospital within an hour from dispatch time had a poor outcome and 57.1% (n= 32) had a good outcome. In the group that missed the 60 minute cut-off, 38.7% (n= 24) had a poor outcome and 61.2% (n= 38) had a good outcome. This is shown in Table 21. There Pearson chi-square test found no association between the patients that were in hospital < 60 minutes vs > 60 minutes and outcome (p = 0.647). Odds of a poor outcome when the total response time is under 60 minutes relative to poor outcome when the total response time is over 60 minutes is 1.19; 95% (CI) 0.53 to 2.65.

4.6.4. Mean Total Transport Time for both Outcome Groups

	Outcome	N	Mean	Std. Deviation	Std. Error Mean
Total Time	Poor Outcome	48	61.94	22.652	3.27
	Good Outcome	70	62.99	22.72	2.716

Table 22: Mean Total Transport Time for both Outcome Groups

Table 22 shows no significant difference between the mean transport times for the good or poor outcome group of patients (p= 0.806).

4.7.1. Pupillary Findings of the Study Population

		Frequency	Percent
	Pupils equal and reactive	43	34.7
	Pupils unreactive	18	14.5
	Slow pupil reaction	31	25
	Pupils fixed and dilated	12	9.7
	Unilateral pupillary reaction	8	6.5
	Total	112	90.3
	Eye or eyes swollen preventing evaluation of pupils	12	9.7
Total		124	100

Table 23: Pupillary findings of the study population

Pupillary size and reaction to light was also documented for all patients in the study from the EMS patient care record form. This is depicted in Table 23. Bilateral reactive pupils were recorded in 34.7% of patients whereas 9.7% patients had fixed and dilated pupils. 14.5% had bilateral unreactive pupils and in 9.7% of cases, the pupils could not be assessed due to peri-orbital edema.

4.7.2. Head CT Findings

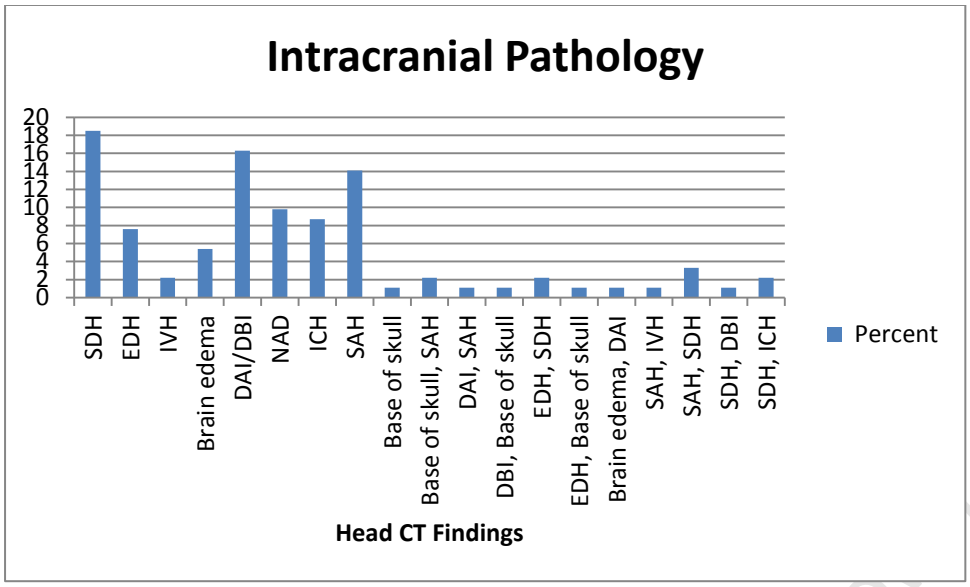

Figure 9: Head CT findings from the study population

The various head CT scan findings are highlighted in Figure 9. Subdural haematomas were the most frequent intracranial pathology detected (18.5%), followed by diffuse brain injuries (16.3%) and subarachnoid haemorrhages (14.1%).

4.8.1. Association between Cause of Injury and Intracranial Pathology

			Head CT Scan				Total
			Only SDH	Only EDH	Only DAI/DBI	Multiple Pathology	
Cause of Injury	Assault	Count	4	3	4	11	22
		% within Cause of Injury	18.20%	13.60%	18.20%	50.00%	100.00%
	Fall from height	Count	5	0	0	3	8
		% within Cause of Injury	62.50%	0.00%	0.00%	37.50%	100.00%
	RTA	Count	8	4	11	37	60
		% within Cause of Injury	13.30%	6.70%	18.30%	61.70%	100.00%
	Train casualty	Count	0	0	0	1	1
		% within Cause of Injury	0.00%	0.00%	0.00%	100.00%	100.00%
Total		Count	17	7	15	52	91
		% within Cause of Injury	18.70%	7.70%	16.50%	57.10%	100.00%

Table 24: Association between cause of injury and intracranial pathology

Frequencies for the cause of injury and intracranial pathology are illustrated above in table 24. In acceleration/deceleration injuries as seen in RTAs, diffuse brain injuries (petechial haemorrhages, intracerebral haematoma and brain swelling) and subdural haematomas were commonly seen. There are 32 cases missing due to patients who died before getting to the CT scan.

4.8.2. Association between Cause of Injury and Outcome

		Outcome		
		Poor	Good	Total
Cause of Injury	Assault	8	22	30
	Fall from height	5	4	9
	RTA	35	47	82
	Train casualty	2	0	2
Total		50	73	123

Table 25: Cause of injury and Outcome

Table 25 shows the cross tabulation which seeks to identify a relationship between cause of injury and outcome. There Pearson chi-square test was performed to determine an association between these variables which found no statistical significance linking the two at the 0.05 level (p= 0.095). It was significant at the level of 0.1.

4.8.3. Association between Outcome and Intracranial Pathology

		Outcome		
		Poor	Good	Total
Head CT	Only SDH	7	10	17
	Only EDH	2	5	7
	Only DAI/DBI	4	11	15
	Multiple pathology	15	38	53
Total		28	64	92

Table 26: Intracranial pathology and outcome

The cross tabulation for intracranial pathology and outcome is tabled above. There are 32 cases missing due to patients who died before getting to the CT scan. Patients with an isolated subdural haematoma suffered the most number of deaths (41.2%) whereas those with an isolated extradural haematoma had the most number of patients with a good recovery (42.9%). There chi-square test found no association between the two variables (p=0.746).

4.8.4. Scene times per GCS score

GCS on Arrival	N	Mean	Std. Deviation	Std. Error of Mean
3	30	35.93	17.512	3.197
4	6	30.67	22.420	9.153
5	18	36.22	20.698	4.879
6	10	34.80	12.709	4.019
7	20	31.25	15.334	3.429
8	34	28.47	14.305	2.453
Total	118	32.67	16.697	1.537

Table 27: Scene times per GCS score

The basic descriptives highlighting the scene time with each GCS score is illustrated in Table 27. Patients with a GCS of 8 spent the least amount of time on scene (28.47) followed by those with a GCS of 4 (30.67). Patients with a GCS of 5 spent the most amount of time on scene (36.22) followed by those with a GCS of 3. There were 6 prehospital transport times missing out of the 124 patients.

4.8.5. Concomitant injuries

		Frequency	Percent
	No Concomitant Injury	76	61.3
	Some Concomitant Injury	48	38.7
	Total	124	100

Table 28: Presence of concomitant injuries

A total of 76 patients (61.3%) were found to have isolated head injuries. 48 patients (38.7%) were found to have some concomitant injuries i.e. femur fracture/s, thoracic injuries. This is depicted in Table 28.

4.9. Evaluating a relationship amongst the variables

			Age	Airway Management	Glasgow Outcome Scale	Arrival Systolic BP	Pupillary findings
	Age	Correlation Coefficient	1	0.1	-0.076	0.173	0.019
		Sig. (2-tailed)	.	0.269	0.401	0.056	0.845
		N	124	124	124	123	112
	Airway Management	Correlation Coefficient	0.1	1	-0.039	-.239**	0.046
		Sig. (2-tailed)	0.269	.	0.668	0.008	0.627
		N	124	124	124	123	112
	Glasgow Outcome Scale	Correlation Coefficient	-0.076	-0.039	1	-0.097	-.455**
		Sig. (2-tailed)	0.401	0.668	.	0.286	0
		N	124	124	124	123	112
	Arrival Systolic BP	Correlation Coefficient	0.173	-.239**	-0.097	1	-0.023
		Sig. (2-tailed)	0.056	0.008	0.286	.	0.806
		N	123	123	123	123	112
	Pupillary findings	Correlation Coefficient	0.019	0.046	-.455**	-0.023	1
		Sig. (2-tailed)	0.845	0.627	0	0.806	.
		N	112	112	112	112	112

Table 29: Spearman's correlation table

** Correlation is significant at the 0.01 level (2-tailed)

The Spearman's correlation revealed a positive weak relationship among the variables. The closest relationship was between the pupil findings and the Glasgow Outcome scale (p= 0.001, p < 0.01). This is depicted in Table 29.

4.9.1. Binary Logistic Regression

Tests of Model Effects			
Source	Type III		
	Wald Chi-Square	df	Sig.
GCS on Arrival	17.495	5	0.004
Age	1.949	1	0.163
SpO2 on arrival	4.319	1	0.038
Total Time	2.795	1	0.095
Pupils	6.796	1	0.009
Dependent Variable: Outcome: Poor/Good			
Model: GCS on Arrival, Age, SpO2 on arrival, Total Time, Pupils			

Parameter	B	Std. Error	95% Wald Confidence Interval		Hypothesis Test		
			Lower	Upper	Wald Chi-Square	df	Sig.
[GCS on Arrival=3]	7.297	3.2623	.903	13.690	5.003	1	.025
[GCS on Arrival=4]	6.368	3.1044	.283	12.453	4.208	1	.040
[GCS on Arrival=5]	7.418	3.3305	.890	13.946	4.961	1	.026
[GCS on Arrival=6]	5.514	3.2282	-.813	11.841	2.918	1	.088
[GCS on Arrival=7]	5.589	3.1947	-.673	11.851	3.060	1	.080
[GCS on Arrival=8]	5.016	3.2522	-1.358	11.391	2.379	1	.123
Age	.030	.0215	-.012	.072	1.949	1	.163
SpO2onarrival	-.065	.0314	-.127	-.004	4.319	1	.038
Total Time	-.019	.0112	-.041	.003	2.795	1	.095
[Pupils	-1.399	.5366	-2.450	-.347	6.796	1	.009
			.	.	.	.	.

Table 30: Binary Logistic Regression I

Dependent Variable: Poor/Good Outcome

Model: GCS on Arrival, Age, SpO2 on arrival, Total Time, Pupils

Table 30 illustrates the binary logistic regression which tested the influence of the predictor variables on the dependent variable (good/poor outcome). GCS 3, 4, 5, SpO2 on arrival and pupillary findings on arrival were significant at the level of p= 0, 05.

4.9.2. Binary Logistic Regression II

Variables in the Equation									
		B	S.E.	Wald	df	p-value	Exp (B) Adjusted Odds Ratio	95% C.I.for EXP(B) Lower	Upper
	SpO2	1.147	0.513	5.006	1	0.025	3.148	1.153	8.598
	BP Systolic	1.259	1.258	1	1	0.317	3.52	0.299	41.458
	GCS	1.631	0.449	13.17	1	0	5.108	2.117	12.323
	Pupil Reactivity	1.483	0.516	8.252	1	0.004	4.405	1.602	12.115
	Concomitant Injury	-0.164	0.46	0.127	1	0.721	0.849	0.345	2.09
	Constant	-2.745	1.301	4.448	1	0.035	0.064		

a. Highlighted p-values show significant effect.

Table 31: Binary Logistic Regression II

Another logistic regression was done to show the association between the Glasgow Outcome scale and the other variables: GCS, SpO_2, Systolic blood pressure, concomitant injury and pupil reactivity in a multivariate model. 72.6% of the outcomes were correctly predicted by this model.

Odds Ratio of a good outcome when SpO2 ≥ 90 = 3.148; 95% CI 1.153 to 8.598. Thus having a SpO2 measurement of 90 or above increases the odds of a good outcome by 214.8%.

Odds Ratio of a good outcome when the systolic blood pressure ≥ 90 = 3.520; 95% CI 0.299 to 41.458. Thus having a systolic blood pressure measurement of 90 or above increases the odds of a good outcome by 252%.

Odds Ratio of a good outcome when (GCS = 6 – 8) = 5.108; 95% CI 2.117 to 12.323. Thus having a GCS measurement of 6 – 8 increases the odds of a good outcome by 410.8%.

Odds Ratio of a good outcome when pupils are bilaterally reactive = 4.405; 95% CI 1.602 to 12.115. Thus having bilateral reactive pupils increases the odds of a good outcome by 340.5%.

Odds Ratio of a good outcome when the patient has no concomitant injuries = 0.849; 95% CI 0.345 to 2.09. Thus having no concomitant injury decreases the odds of a good outcome by 15.1%.

$$\ln \frac{P(D)}{1-P(D)} = \alpha + \beta_1(X_1) + \beta_2(X_2) + \ldots$$

$$odds\ of\ a\ good\ outcome = \frac{P(D)}{1-P(D)} = e^{\alpha + \beta_1(X_1) + \beta_2(X_2) + \ldots}$$

$$Probability = \frac{Odds\ Ratio}{1 + Odds\ Ratio}$$

The odds of disease increase multiplicatively by $e^{ß}$ for for every one-unit increase in the exposure, controlling for other variables in the model.

Or

The odds of a good outcome decrease multiplicatively by $e^{ß}$ for for every one-unit decrease in the exposure, controlling for other variables in the model

Odds Ratio for this study =

$$e^{-2745 + 1.631 * \text{GCS} + 1.147 * \text{SpO2} \geq 90 + 1.259 * \text{BP Sys} \geq 90 + 1.483 * \text{Bilateral Reactive pupils} - 0.164 * \text{No Concomitant Injury}}$$

All the values in the equation above come from the column labelled “B” in the table above. These are the estimated parameters in the model.

Using this equation we can calculate the probability of a good outcome for various situations:

E.g. if a patient has a GCS of 6 – 8, both SpO2 and Systolic blood pressure of above 90, eyes bilaterally reactive and no concomitant injury then the odds of the person having a good outcome on the Glasgow outcome scale is 13.603 to 1 and the chances of a good outcome is 93%.

The interesting (odd really) statistic is that if a patient has a GCS of 6 – 8, both SpO2 and systolic blood pressure of above 90, Eyes bilaterally reactive and **Some** concomitant injury then the odds of the person having a good outcome on the Glasgow outcome scale is 16.026 to 1 and the chances of a good outcome is 94%.

The table detailing this follows.

GCS	SpO2	BP Sys	Pupils Reactive	Concomitant Injury	Odds Ratio (Good Outcome)	Probability (Good outcome)
GCS 6 - 8	90 or above	90 or above	Eyes bilaterally reactive	**Some Concomitant Injury**	16.026	0.941
GCS 6 - 8	90 or above	90 or above	Eyes bilaterally reactive	No Concomitant Injury	13.603	0.932
GCS 6 - 8	**Below 90**	90 or above	Eyes bilaterally reactive	**Some Concomitant Injury**	5.091	0.836
GCS 6 - 8	90 or above	**Below 90**	Eyes bilaterally reactive	**Some Concomitant Injury**	4.553	0.82
GCS 6 - 8	**Below 90**	90 or above	Eyes bilaterally reactive	No Concomitant Injury	4.321	0.812
GCS 6 - 8	90 or above	**Below 90**	Eyes bilaterally reactive	No Concomitant Injury	3.864	0.794
GCS 6 - 8	90 or above	90 or above	**All other findings with eyes**	**Some Concomitant Injury**	3.638	0.784
GCS 3 - 5	90 or above	90 or above	Eyes bilaterally reactive	**Some Concomitant Injury**	3.138	0.758
GCS 6 - 8	90 or above	90 or above	**All other findings with eyes**	No Concomitant Injury	3.088	0.755
GCS 3 - 5	90 or above	90 or above	Eyes bilaterally reactive	No Concomitant Injury	2.663	0.727
GCS 6 - 8	**Below 90**	**Below 90**	Eyes bilaterally reactive	**Some Concomitant Injury**	1.446	0.591
GCS 6 - 8	**Below 90**	**Below 90**	Eyes bilaterally reactive	No Concomitant Injury	1.227	0.551
GCS 6 - 8	**Below 90**	90 or above	**All other findings with eyes**	**Some Concomitant Injury**	1.156	0.536
GCS 6 - 8	90 or above	**Below 90**	**All other findings with eyes**	**Some Concomitant Injury**	1.033	0.508
GCS 3 - 5	**Below 90**	90 or above	Eyes bilaterally reactive	**Some Concomitant Injury**	0.997	0.499
GCS 6 - 8	**Below 90**	90 or above	**All other findings with eyes**	No Concomitant Injury	0.981	0.495
GCS 3 - 5	90 or above	**Below 90**	Eyes bilaterally reactive	**Some Concomitant Injury**	0.891	0.471
GCS 6 - 8	90 or above	**Below 90**	**All other findings with eyes**	No Concomitant Injury	0.877	0.467
GCS 3 - 5	**Below 90**	90 or above	Eyes bilaterally reactive	No Concomitant Injury	0.846	0.458
GCS 3 - 5	90 or above	**Below 90**	Eyes bilaterally reactive	No Concomitant Injury	0.757	0.431
GCS 3 - 5	90 or above	90 or above	**All other findings with eyes**	**Some Concomitant Injury**	0.712	0.416
GCS 3 - 5	90 or above	90 or above	**All other findings with eyes**	No Concomitant Injury	0.605	0.377
GCS 6 - 8	**Below 90**	**Below 90**	**All other findings with eyes**	**Some Concomitant Injury**	0.328	0.247
GCS 3 - 5	**Below 90**	**Below 90**	Eyes bilaterally reactive	**Some Concomitant Injury**	0.283	0.221
GCS 6 - 8	**Below 90**	**Below 90**	**All other findings with eyes**	No Concomitant Injury	0.279	0.218
GCS 3 - 5	**Below 90**	**Below 90**	Eyes bilaterally reactive	No Concomitant Injury	0.24	0.194
GCS 3 - 5	**Below 90**	90 or above	**All other findings with eyes**	**Some Concomitant Injury**	0.226	0.185
GCS 3 - 5	90 or above	**Below 90**	**All other findings with eyes**	**Some Concomitant Injury**	0.202	0.168
GCS 3 - 5	**Below 90**	90 or above	**All other findings with eyes**	No Concomitant Injury	0.192	0.161
GCS 3 - 5	90 or above	Below 90	All other findings with eyes	No Concomitant Injury	0.172	0.147
GCS 3 - 5	Below 90	Below 90	All other findings with eyes	Some Concomitant Injury	0.064	0.06
GCS 3 - 5	**Below 90**	**Below 90**	**All other findings with eyes**	No Concomitant Injury	0.055	0.052

Table 32: Table indicating odds ratio of a good outcome with various predictor variables

4.9.3. Bivariate analysis

4.9.3.1. Systolic Blood Pressure and Outcome

		Outcome		Total
		Poor	Good	
Systolic Arrival on scene	Below 90	6	1	7
	90 or above	44	72	116
Total		50	73	123

Table 33: Systolic blood pressure and outcome

Table 33 illustrates a cross tabulation between the systolic blood pressure taken on arrival at the scene and outcome. A Pearson chi-square test was performed to determine an association between the two. At 95% significance, there is an association between the systolic blood pressure taken on arrival at the scene and outcome ($p = 0.018$).

4.9.3.2. Age and Outcome

		Outcome		Total
		Poor	Good	
Age	Below 40	35	55	90
	40 and above	15	19	34
Total		50	74	124

Table 34: Age and Outcome

61% of patients below the age of 40 had a good outcome while 56% of those above 40 had a good outcome. This is depicted in Table 34. At 95% significance, there

Pearson chi-square test indicated no statistical significance linking age and outcome (p= 0.596).

4.9.3.3. SpO_2 and Outcome

		Outcome		Total
		Poor	Good	
SpO_2 arrival on scene	Below 90%	18	15	33
	90% or above	28	56	84
Total		46	71	117

Table 35: SpO_2 and Outcome

Table 35 shows a poor outcome was the result in 55% of patients with SpO_2 below 90% in contrast to the 33% who had SpO_2 above 90. At 95% significance, there Pearson chi-square test found no observed relationship between the SpO_2 and outcome in this study (p= 0.35).

4.9.3.4. GCS and Outcome

		Outcome		Total
		Poor	Good	
GCS on Arrival	3	21	12	33
	4	3	3	6
	5	11	8	19
	6	4	6	10
	7	6	15	21
	8	5	30	35
Total		50	74	124

Table 36: GCS and Outcome

64% of patients with GCS 3 had a poor outcome in contrast to 14% who had a GCS of 8. The higher, the GCS score, the better the outcome. A good outcome was the result in 42%, 60%, 71% and 86% of patients with GCS 5, 6, 7 and 8 respectively. This is illustrated in Table 36.

With 95% confidence, there Pearson Chi-square test revealed a positive strong relationship concerning the GCS and outcome (p= 0.001).

4.9.3.5. Pupils and Outcome

		Outcome		Total
		Poor	Good	
Pupils	Pupils bilaterally reactive	8	35	43
	All other findings with pupils	42	39	81
Total		50	74	124

Table 37: Pupils and Outcome

Table 37 illustrates a crosstabulation between pupillary findings on scene and outcome. To determine an association between the two variables, the Pearson chi-square test was done.

With 95% confidence, there was a strong association between the pupillary findings on scene and outcome (p= 0.001).

CHAPTER 05

DISCUSSION

5.1. Introduction

Chapter four presented the findings of the various airway management methods currently utilised in the Cape Town Metropole for the management of severe traumatic brain injury. The association of each method and prehospital transport times was also presented. This chapter discusses the findings of this study in the context of other similar studies conducted on this topic.

There are several publications describing out-of-hospital intubation and outcome in severe TBI [89-92]. However, most of them are from developed countries who offer superior healthcare services compared to developing nations. Their findings might slightly differ to what we have as a developing nation. With a high incidence of road traffic accidents and violence, a high prevalence of TBI is inevitable in South Africa. As discussed in chapter 2, there is no scientific evidence for the ALS interventions performed in the pre-hospital setting for the management of severe TBI in South Africa and this study seeks to answer some of the questions related to the management of this group of critically injured patients.

There was a 50% split among the patients collected from GSH and TBH. There was no significant difference in outcome from the two hospitals. The prevalence of TBI was higher among the male group in this study (88.7%) compared to females (11.3%). These findings were similar to other studies of patients presenting with TBI as males are more likely to partake in acts of violence [4, 90]. The mean age of the participants was 32 years with road traffic accidents (67%) the most common cause of injury. These figures are similar to the CRASH trial which had a mean age of 37 years and 64% were enrolled secondary to a road traffic accident.

The main findings of this study were that sedation-assisted intubation is the most practiced technique of airway management for TBI patients presenting with GCS ≤8 in the Cape Town Metropole. Patients who underwent basic airway management had the highest proportion of a good outcome in this study with respect to the intubated groups. The former was also found to spend the least amount of time on

scene in comparison to those intubated in the prehospital setting. It is unclear whether this could have resulted in improved outcomes as we found no association between the total-out-of hospital interval and outcome.

5.2. Limitations

This study has certain limitations that need to be well-thought-out prior to considering the contributions of this study. The issue surrounding out-of-hospital intubation and outcome in severe TBI is a complex one that presents a challenge for prehospital research. One would ideally conduct a randomised controlled trial with a large sample size to provide a definite answer on the method of airway management for this group of critically injured patients. However this was not possible due to time and resource constraints. Though we found an association between airway management and outcome in this study, there was no significant difference between injury severity amongst the airway groups. This could be attributed to the relatively small sample size. This however still raises the important question about the added benefit of early pre-hospital intubation in severe TBI. It was evident in chapter 04 that patients intubated in the pre-hospital milieu spend more time on scene compared to non-intubated patients. Unfortunately, the design of the study does not give us the insight into the reasons for delay on scene besides the airway management. This was beyond the scope of this study and a bigger study is recommended to establish all the possible scenarios that lead to scene delay in patients with severe TBI. The design of the study relied heavily on record keeping from other health care professionals thereby increasing their probability of random error. One of the limitations of the study is the total out-of hospital interval is taken from dispatch to arrival. It is not possible to get the exact time of an incident and thus the decision to utilise the dispatch time.

5.3. Airway Management Practices in the Cape Town Metropole

As discussed in chapter 2, the airway of the severe TBI patient can be managed in various ways depending on the emergency care practitioner's scope of practice. The BLS and ILS practitioner can only employ basic airway techniques while the ALS practitioner can perform endotracheal intubation which is perceived to be the more ideal airway intervention as it secures the airway [107, 108].

Sedation-assisted intubation was the airway method that was mostly employed in this study (44.4%) followed by basic airway management (29.8%). It must be considered that the majority of the ALS practitioners in Cape Town hold the ANT registration with the HPCSA. These practitioners can only intubate with the assistance of benzodiazepines and morphine sulphate for synergism. The PGWC Metro EMS has some ALS practitioners who hold the ECP registration with the HPCSA and can thus perform RSI within their scope of practice. However, performing this skill is not permitted by the organisation and practitioners have to utilise benzodiazepines to intubate patients with severe TBI. PGWC Metro EMS is currently working on the policy governing the safe practice of prehospital rapid sequence intubation. There were 10.5% of patients who underwent RSI in this study. This procedure was performed by ALS practitioners from the private sector who received the neuromuscular blocking agents from the employers.

There were thirty-seven patients that underwent basic airway techniques. The practitioners either used an oxygen face mask or a bag-valve mask for supplementation of oxygen. This was due to no paramedic being available for the incident and thus the BLS/ILS practitioner had to transport the patient to GSH/TBH. This can be attributed to the skills shortage of advanced life support paramedics in South Africa [109]. Depending on the location of the incident, the ambulance can take anything from five to thirty minutes to GSH/TBH hence some ALS practitioners choose to transport the patient to hospital without the insertion of an endotracheal tube in an attempt to minimise transportation time.

There were 8.9% failed airway cases reported in this study. This is similar to the international literature which reports up to 18.5% of failed intubations [110]. The practitioners were found to have administered benzodiazepines and morphine sulphate or hypnomidate and suxamethonium but failed to insert the endotracheal tube. Some patients were found to still have an intact gag reflex following the administration of midazolam. A bag-valve mask was used in 72.7% of the failed airway cases to optimize oxygenation and ventilation. The use of supraglottic devices is widely advocated as a 'bailout device' during endotracheal intubation [107, 108]; however the laryngeal mask airway (LMA) was only used in 27.3% of the failed airway cases. Laryngeal mask airways are made available by the EMS service providers in the Cape Town metropole. It is not clear whether the ALS practitioners

are not comfortable with the insertion of the LMA or oblivious to the use of the device.

The LMA is often criticised as it does not completely secure the airway [107, 108]. The position of the LMA can increase the risk of regurgitation which would result in pulmonary aspiration or oesophageal rupture. Patients in the prehospital setting are not fasted and thus have a high risk of aspiration.

On the other hand, the LMA has been shown to provide better oxygenation compared to the bag-valve mask (BVM) device with an oral airway which also doesn't offer the added benefit of airway protection [108]. The LMA does not require the continuous maintenance of a mask seal as seen with the BVM device. The BVM also causes gastric insufflation which results in diaphragmatic splinting and vomiting [108]. The BVM requires two practitioners to ensure adequate ventilation of the severe TBI patient; one practitioner to perform the jaw-thrust technique while the second one squeezes the bag. This requires more personnel for one particular skill who are not always available. The latest embellishments of the LMA such as the Pro-Seal include a gastric port to drain the stomach contents in the emergency setting. The LMA should therefore be the device of choice after failed endotracheal intubation in the prehospital setting [107, 108].

The maintenance of the endotracheal intubation skill amongst South African ALS practitioners is thought to be reasonably high due to the high burden of disease, road traffic accidents and violence. As a result, one would perceive them as highly proficient in advanced airway management as endotracheal intubation is arguably more difficult in the uncontrolled pre-hospital setting. Botha [111] found a 3% rate of misplaced endotracheal tubes in an analysis of out-of-hospital success of endotracheal tube placement in Johannesburg. However the exact number of failed airways was unknown in the latter study as it only looked at unrecognised misplaced endotracheal tubes.

5.4. Outcome of TBI in the Study Population

The mortality of the study population was 38.7% with 60% having a good outcome. These findings are similar to Wang et al [92] who had a mortality of 37% in their analysis of 4,098 TBI patients. This is in contrast to Bochicchio et al [90] who had a

mortality of 17%. 40% were discharged home while 19.4% were discharged to a rehab centre.

5.5. Airway Management and Outcome

A correlation between airway management and outcome was found in this study. Patients who underwent basic airway management had the highest proportion of a good outcome (72.9%) compared to patients who were intubated in the prehospital setting. There was no significant difference in injury severity amongst the airway groups. A good outcome was the result in 61.8% and 38.4% of patients who experienced sedation-assisted intubation and RSI respectively. Secondary to patients intubated without drugs, RSI had the highest proportion of a poor outcome (61.5%) followed by the sedation-assisted group (38.2%). Despite the advantages of endotracheal intubation in TBI, multiple studies have demonstrated adverse outcomes from this complex procedure in severe TBI.

Murray et al [91] demonstrated that patients who were intubated in the prehospital setting had increased mortality when compared to non-intubated patients in severe TBI. Similarly, Bochicchio and colleagues [90] demonstrated increased morbidity and mortality in the prehospital intubated patients. They also found an increased incidence of pneumonia and ventilator days with the prehospital intubated patients which have been found in other similar studies [102, 112]. In this study, the RSI group had the longest ICU stay (14 days) whilst the sedation-assisted group had the shortest stay (7 days) in ICU. A study of 31,464 paediatric patients with severe TBI also failed to demonstrate better survival in the patients who were intubated in the out-of-hospital milieu compared to bag-valve mask ventilation [113]. Wang et al [92] also found increased mortality, poorer functional and neurological outcome in patients who were intubated in the prehospital environment.

Nevertheless, Winchell and Hoyt [89] found a 21% increase in survival in an analysis of 1,098 TBI patients in the out-of-hospital intubation group. Similarly, Suominen et al [114] discovered that prehospital intubation resulted in a 34% increase in survival over patients who were intubated in the emergency centre in an evaluation of 59 paediatric TBI patients. However both studies did not adjust for severity of injury or illness which limits the conclusions that can be made from these studies. Having said

this, with all the above-mentioned studies, it is unclear whether any pharmacological agents were used to facilitate endotracheal intubation as not all paramedics are permitted to carry drugs for this advanced skill. In an analysis of 486 trauma patients intubated in the field without any pharmacological agent, Lockey et al [115] had one survival in that study. Likewise, we had only one survival in this study from the group that was intubated without drugs. One might argue that if the patients were intubated without drugs in the prehospital setting in these afore-mentioned studies, they may already have had very low GCS scores and therefore a high probability of death expected.

RSI is thought to be the airway management technique of choice in the patient with severe TBI as it attenuates the intracranial pressure response during laryngoscopy. It has also been used by some EMS systems to increase the success rate of endotracheal intubation [98, 116]. Despite its advantages, patients receiving RSI in this study had significantly poorer outcomes compared to other airway methods. Despite a small group that received RSI, a larger study found similar findings. Davis et al [101] evaluated 209 TBI patients receiving RSI and matched them to 627 non-intubated controls, comparing prehospital RSI with the alternative of no intubation in the pre-hospital milieu. The RSI group had 33% mortality rate vs 24.2% and a decreased prevalence of good outcome 45.5% vs 57.9%. These findings contradict those by Bernard et al [104] who found that prehospital RSI increases favourable outcome at 6 months compared with hospital intubation in their randomised controlled trial. Sloane and colleagues [102] compared patients who underwent prehospital RSI with those that underwent emergency department RSI. They found no difference in mortality, length of ICU and hospital stay. These findings are counterintuitive to the assumption that early advanced airway management is associated with improved outcomes in TBI.

Davis et al [117] demonstrated that hyperventilation is a common phenomenon following pre-hospital RSI. This results in cerebral vasoconstriction and subsequently reduces cerebral perfusion. They subsequently noted a correlation between hyperventilation and increased mortality. A ventilator is a scarce commodity in the Cape Town EMS setting. ALS practitioners often have to rely on the BVM to ventilate intubated patients. They have no control over the minute volume and hyperventilation is therefore a very likely scenario. The ventilator is a mandatory adjunct during pre-hospital RSI as per HPCSA regulation. However Christopher [118]

found that South African EMS providers were non-compliant with the HPCSA protocols for various reasons. It is not clear whether ALS practitioners take hyperventilation into account when setting a ventilator for the severe TBI patient. Likewise, patients with associated thoracic injuries might require lower pressures or volume in order to minimise the risk of increased intrathoracic pressure. This condition impedes venous drainage from the cerebral vasculature resulting in decreased cerebral blood flow and cause a rise in intracranial pressure.

5.6. Relationship between Airway Management Methods and Prehospital Transport Times

It is not surprising that advanced airway management was associated with increased pre-hospital transport times in this study. Practitioners who inserted an LMA after a failed airway spent the longest time on scene (42 min) followed by the sedation-assisted intubation group (40 min). This appears to suggest that an additional 2 minutes was spent on scene to insert the supraglottic device after the failed intubation. The rationale for the delay on scene is not clear. However, 38.7% of patients in the study had concomitant injuries (i.e. long bone fractures, thoracic injuries) and thus required additional time and care to be stabilised on scene. Interestingly, there was no significant difference in the scene interval between patients with concomitant injuries and those without concomitant injuries. There was also no significant difference in the mean total transport interval between the poor and good outcome group. This could be attributed to the large number of emergency care personnel at accident scenes' in the Cape Town Metropole who assist each other with patient care. However Spaite et al [119] argue that no fragment of the pre-hospital process should be adjudged as more/less important as the other. The pre-hospital process should be understood as a continuum encompassing everything before the incident risk through to rehabilitation.

During sedation-assisted intubation, patients are not always flaccid and the gag reflex is sometimes still present following the administration of the benzodiazepine. The dosage and administration of Midazolam according to the HPCSA ALS protocol is 1mg/min slowly IVI with an onset of action of 1-3 minutes. Dosages of between 15-30mg of Midazolam were noted in the EMS patient care record forms. Boluses of 5-15mg were given slowly IVI and additional dosages had to be administered at times

as the patient still had a gag reflex. This predisposes the patient to hypotension which has been shown to be detrimental to TBI patients. Likewise, this study found an association between hypotension (systolic blood pressure <90) and outcome. On the other hand, we found no significant drop in blood pressure after the administration of Midazolam and Morphine in this study. As a matter of fact, we found a slight increase in systolic blood pressure.

It is unknown whether the patients were being adequately ventilated via the BVM during this period of sedation. One study noted periods of hypoxia during prehospital intubation which has also been linked to poor outcome in TBI [120]. Nonetheless there was no association between hypoxia and outcome in this study.

There is also the added danger of increasing ICP during airway manipulation which the TBI patient is most vulnerable as autoregulation is often impaired. There is also an expectation from the trauma unit doctor that the severe TBI patient needs to be intubated prior to arrival at the unit if brought in by a paramedic. It would appear that ALS practitioners feel compelled to intubate these patients to avoid conflict at the receiving hospital despite shorter transport times or simply struggling to secure the airway.

Secondary to the basic airway management group, patients that were intubated without drugs spent the least amount of time on scene. The latter had the shortest stay on scene (22.46 min) and were in hospital quicker (51 min) than the sedation-assisted intubation group (70 min). As noted above, they also had the highest proportion of a good outcome. It is unclear whether the airway intervention or the decreased prehospital transport time was the result of the good outcome. This certainly raises questions on how to best approach the airway of critically injured patients. Should one should load and go or stay and play.

Despite being a new skill, the RSI group had shorter scene and total pre-hospital transport times, 33.67 and 61.25 minutes respectively. The implementation of this new skill appears to shorten scene and overall transport time compared to sedation-assisted intubation.

5.7. Association of Prehospital Transport Times and Outcome in Severe TBI

Time has always been a critical factor in prehospital emergency care. Trauma patients are thought to require expedient transport to the nearest appropriate medical facility as they require the least possible time from injury to definitive care [121]. The 'golden hour' concept is that trauma patients have improved outcomes if they are provided definitive care within 60 minutes from the time of injury. The 'golden hour' concept appears to have originated from Cowley [121] who stated that "the first hour after injury will largely determine a critically-injured person's chance of survival". There is however no explicit research findings for this statement and the basis for the statement remains unclear.

While it appears intuitive that less pre-hospital time is better for trauma patients, various studies have failed to demonstrate an association between total out-of hospital time and mortality [84, 122 - 125]. In fact, this study also failed to demonstrate a relationship between response times, total out-of hospital time and outcome. This makes "innate" sense as the pre-hospital care is aimed at reversing any physiologic abnormalities prior to hospital arrival thus giving patients the possibility to benefit from definitive care.

78% of response times were under 15 minutes in this study with an average response time of 10 minutes. It must be noted however that the response time was calculated from dispatch and not from call incident time. In those who had a response time under 15 minutes, 55% had a good outcome. Good outcome was the result in 73% of patients with response times over 15 minutes. Pons and Markovchick [124] found no difference in outcome after exceeding the 8 minute response time after a traumatic injury. In a similar study, Pons et al [125] found a survival benefit when the response time was less than 4 minutes in patients experiencing cardiac arrest. They subsequently found no survival advantage to hospital discharge based on the 8-minute cut-off point. Newgard et al [84] prospectively evaluated 3,656 trauma patients who were transported by EMS with physiologic abnormality. Likewise, they found no association between prehospital transport times and mortality.

5.8. Intracranial Pathology

In isolated intracranial pathologies, subdural haematoma was the most common (18.5%), followed by diffuse axonal injuries (16.3%) and subarachnoid haemorrhages (14.1%). These findings are in contrast with the TCDB series which noted that 56% and 24% of patients with severe TBI will suffer from DAI and SDH respectively. Some patients had multiple intracranial pathologies which included subdural haematomas and DAIs. The reason for this finding may be that road traffic accidents was the most common cause of injury in this study. With a high number of patients sustaining acceleration/deceleration injuries, it is plausible that there might be more patients who sustained DAIs in this study. DAI is a histologic finding consisting of shearing and tearing of the white matter tracts which is not visible on CT scan. As noted in chapter 2, there is a considerable delay from hospital arrival to CT scan at TBH. This is contrary to the ATLS guidelines [105] which advocate a target of 30 minutes from injury to CT scan. This however is feasible in developed countries which have shorter distances to hospital with better trauma systems.

Death occurred in 26% of patients while waiting for CT scan in this study. It is unclear whether the delay contributed to the patient's outcome. A study by Seelig et al [106] found 30% mortality for patients operated within four hours compared to 90% mortality to those operated after four hours after an acute subdural haematoma. The TCDB noted 50% mortality in patients with SDH. This is contrary to 41% in this study where SDH had the highest mortality. We subsequently found no association between intracranial pathology and outcome. The latter contradicts numerous studies that have found an association between intracranial pathology and outcome [126 - 129]. This finding might be due to a smaller sample in this study.

5.9. Association of Predictor Variables and Outcome

We performed a binary logistic regression to identify risk factors for outcome in TBI. The predictor variables were age, GCS and SpO_2 on arrival at the scene, pupil reactivity and time from dispatch to hospital arrival. Most of these variables have been associated with outcome in TBI in other studies and were thus included in our regression model [6, 20, 52, 57, 59, 60, 61, 62, 72, 73]. Blood pressure was not included in the regression as it had no effect on the model. However the bivariate analysis between blood pressure and outcome indicated a strong association between the two

variables (p=0.001). These findings concur with that of Chesnut et al [6] who found 150% increase in mortality in patients with a single episode of hypotension.

In this study, GCS on arrival, pupillary findings and SpO_2 on arrival were significant at 5% level while total time was significant at 10% level. There was no association between patients with GCS scores 6 - 8 suggesting that the higher the GCS score, the better the prognosis. In fact, the second logistic regression model suggests that having a GCS 6 – 8 increases the odds of a good outcome by 410.8%. Increasing age was not associated with poor outcome in this study. This is contrary to other studies who have found increasing age to be associated with adverse outcome [70, 71]. This could be due to the fact that patients < 16 years of age were not included in the study. In one study, this association was present at the age of 40 years [71].

CHAPTER 06

CONCLUSION and RECOMMENDATIONS

6.1. Conclusion

This study was unique in that it looked at all the various airway techniques utilised in the prehospital emergency setting for the management of severe TBI. Despite a small sample, this study was able to provide us with some key answers and serves as a good platform for larger studies of a similar nature to be conducted in the future. There has been great success over the last two decades regarding the thorough understanding of physiological derangements leading to secondary brain injury. It is likely however that some patients are not treated to current best practise guidelines for managing severe TBI. Current literature supports the prevention of secondary brain injury from hypoxia and hypotension while minimising on-scene time and providing rapid transport to the nearest facility which can monitor and treat intracranial hypertension. While this study clearly demonstrates an association between airway management and outcome, there are still lots of unanswered questions regarding the value of endotracheal intubation versus basic airway management for patients maintaining SpO_2> 90% in an urban setting.

It is clear that pre-hospital intubation without the use of drugs is linked to poorer outcomes compared with no intubation. Sedation-assisted intubation increases the chances of patients who may be intubated by suppressing the gag reflex. It is plausible that this procedure may prove to be disastrous in the TBI patient as it can aggravate intracranial hypertension during conventional laryngoscopy thus reducing cerebral perfusion. It is interesting that despite the use of sedation, there was no statistical significant drop in blood pressure that could account for this poorer outcome. Failure to intubate might result in increased risk of aspiration due to a probable depressed gag reflex. This method appears to be inappropriate as there is no published pre-hospital data suggesting positive outcomes following sedation-assisted intubation. It is also associated with prolonged pre-hospital transport times. Based on the rationale that early aggressive airway management reverses the deleterious effects of hypoxia, it is reasonable to expect that RSI would have a positive impact on TBI. This study however found adverse outcomes following RSI in patients with severe TBI. This appears to be in contrast with the assumption that

aggressive airway management is related with better outcomes. A large prospective, randomized trial is warranted to yield some insight into the airway management of a TBI patient and how these methods influence outcome. However, ethics approval is a challenge in conducting such a trial as many consider endotracheal intubation to be a standard of care in severe TBI. The results of this study should in fact favour ethics approval as there appears to be worse outcome in the current airway management techniques employed and for this reason it has to be investigated further with a class one study. It is important to note that the relationship between airway management and outcome in this study is not causal but associative.

Response time's ≤ 15 minutes were not associated with improved outcomes in this study. Meeting this expectation can result in more emergency vehicle accidents and put the public and emergency service's personnel at risk. Thus adherence to the 15-minute response time is not supported by this study. In conclusion, given that prolonged pre-hospital transport times are not associated with adverse outcomes, this study supports stabilisation of the critically-injured patient prior to transportation to hospital.

6.2. Recommendations

A class one study is required to answer the question as to whether endotracheal intubation, as part of advanced life support pre-hospital airway management, in severe TBI reduces morbidity and mortality.

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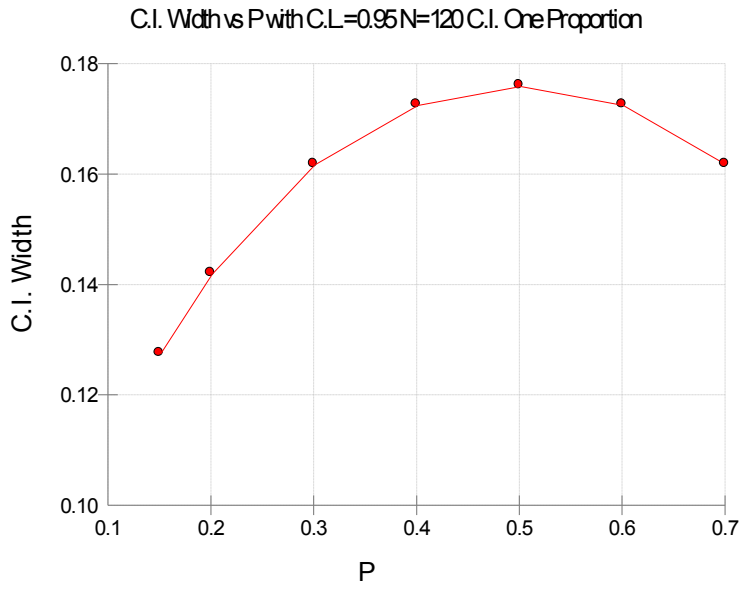
ANNEXURE 1

Proportion (P)	Lower Limit	Upper Limit
0.150	0.097	0.225
0.200	0.138	0.280
0.300	0.225	0.387
0.400	0.317	0.489
0.500	0.412	0.588
0.600	0.511	0.683
0.700	0.613	0.775

Chart Section



ANNEXURE 2

Confidence Level	Sample Size (N)	Target Width	Actual Width	Proportion (P)	Lower Limit	Upper Limit	Width if P = 0.5
0.950	10		0.423	0.150	0.035	0.459	0.527
0.950	10		0.453	0.200	0.057	0.510	0.527
0.950	10		0.495	0.300	0.108	0.603	0.527
0.950	10		0.519	0.400	0.168	0.687	0.527
0.950	10		0.527	0.500	0.237	0.763	0.527
0.950	10		0.519	0.600	0.313	0.832	0.527
0.950	10		0.495	0.700	0.397	0.892	0.527
0.950	20		0.308	0.150	0.052	0.360	0.401
0.950	20		0.335	0.200	0.081	0.416	0.401
0.950	20		0.373	0.300	0.145	0.519	0.401
0.950	20		0.395	0.400	0.219	0.613	0.401
0.950	20		0.401	0.500	0.299	0.701	0.401
0.950	20		0.395	0.600	0.387	0.781	0.401
0.950	20		0.373	0.700	0.481	0.855	0.401
0.950	30		0.253	0.150	0.063	0.316	0.337
0.950	30		0.278	0.200	0.095	0.373	0.337
0.950	30		0.312	0.300	0.167	0.479	0.337
0.950	30		0.331	0.400	0.246	0.577	0.337
0.950	30		0.337	0.500	0.332	0.668	0.337
0.950	30		0.331	0.600	0.423	0.754	0.337
0.950	30		0.312	0.700	0.521	0.833	0.337
0.950	40		0.220	0.150	0.071	0.291	0.296
0.950	40		0.243	0.200	0.105	0.348	0.296
0.950	40		0.274	0.300	0.181	0.454	0.296
0.950	40		0.291	0.400	0.263	0.554	0.296
0.950	40		0.296	0.500	0.352	0.648	0.296
0.950	40		0.291	0.600	0.446	0.737	0.296
0.950	40		0.274	0.700	0.546	0.819	0.296
0.950	50		0.197	0.150	0.076	0.274	0.267
0.950	50		0.218	0.200	0.112	0.330	0.267
0.950	50		0.246	0.300	0.191	0.438	0.267
0.950	50		0.262	0.400	0.276	0.538	0.267
0.950	50		0.267	0.500	0.366	0.634	0.267
0.950	50		0.262	0.600	0.462	0.724	0.267
0.950	50		0.246	0.700	0.562	0.809	0.267
0.950	60		0.180	0.150	0.081	0.261	0.245
0.950	60		0.200	0.200	0.118	0.318	0.245
0.950	60		0.226	0.300	0.199	0.425	0.245
0.950	60		0.241	0.400	0.286	0.526	0.245
0.950	60		0.245	0.500	0.377	0.623	0.245
0.950	60		0.241	0.600	0.474	0.714	0.245
0.950	60		0.226	0.700	0.575	0.801	0.245
0.950	70		0.167	0.150	0.085	0.252	0.228
0.950	70		0.185	0.200	0.123	0.308	0.228
0.950	70		0.210	0.300	0.205	0.415	0.228
0.950	70		0.224	0.400	0.293	0.517	0.228
0.950	70		0.228	0.500	0.386	0.614	0.228
0.950	70		0.224	0.600	0.483	0.707	0.228

Confidence Level	Sample Size (N)	Target Width	Actual Width	Proportion (P)	Lower Limit	Upper Limit	Width if P = 0.5
0.950	70		0.210	0.700	0.585	0.795	0.228
0.950	80		0.156	0.150	0.088	0.244	0.214
0.950	80		0.173	0.200	0.127	0.300	0.214
0.950	80		0.197	0.300	0.211	0.408	0.214
0.950	80		0.210	0.400	0.300	0.510	0.214
0.950	80		0.214	0.500	0.393	0.607	0.214
0.950	80		0.210	0.600	0.490	0.700	0.214
0.950	80		0.197	0.700	0.592	0.789	0.214
0.950	90		0.147	0.150	0.091	0.238	0.202
0.950	90		0.164	0.200	0.130	0.294	0.202
0.950	90		0.186	0.300	0.215	0.401	0.202
0.950	90		0.198	0.400	0.305	0.503	0.202
0.950	90		0.202	0.500	0.399	0.601	0.202
0.950	90		0.198	0.600	0.497	0.695	0.202
0.950	90		0.186	0.700	0.599	0.785	0.202
0.950	100		0.140	0.150	0.093	0.233	0.192
0.950	100		0.155	0.200	0.133	0.289	0.192
0.950	100		0.177	0.300	0.219	0.396	0.192
0.950	100		0.189	0.400	0.309	0.498	0.192
0.950	100		0.192	0.500	0.404	0.596	0.192
0.950	100		0.189	0.600	0.502	0.691	0.192
0.950	100		0.177	0.700	0.604	0.781	0.192

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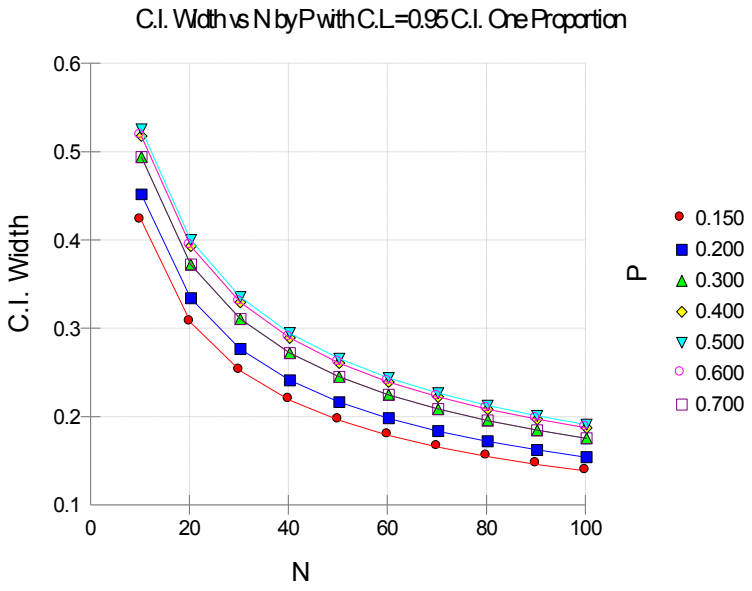
Chart Section



Annexure 3

Data Collection Sheet

Outcome Form

1. **I.D.** 1, 2, 3, 4 etc.
2. **Hospital name** ☐
3. **Folder no**. ☐
4. **Sex:** M / F
5. **Cause of injury:** ☐ RTA ☐ Fall Other: ☐
6. **Airway Management method:** ☐
7. **Outcome:** ☐ Dead ☐ discharged home ☐ discharged to police custody

   ☐ Discharged to rehab centre ☐ signed out of hospital against medical advice

   ☐ Discharged to nursing home/long-term facility care centre

8. Yes ☐ No ☐ Admitted to ICU, if **Yes**, how many days in ICU ☐
9. **Glasgow Outcome scale:** 1 = Dead
   2 = Vegetative
   3 = Severe disability
   4 = Moderate disability
   5 = Good recovery
   ☐
10. Yes ☐ No ☐ Head CT scan: **Result:** ☐
11. ☐ Length of stay in hospital

Annexure 4

Data Collection Sheet

Prognostic variables

Folder no:			
GCS on arrival:		**Airway management:**	
GCS on hospital arrival:		**M score post airway management:**	
M score on arrival:		**M score on hospital arrival**	
SpO2 on arrival:		**SpO2 post airway management:**	
SpO2 on hospital arrival:			
BP on arrival:		**BP post airway management:**	
BP on hospital arrival:			
HR on arrival:		**HR post airway management:**	
HR on hospital arrival:			
RR on arrival:		**RR post airway management:**	
RR on hospital arrival:			
HGT:			
(ETCO2) post airway management:		**(ETCO2): on hospital arrival:**	
Pupillary findings:			
Response time:		**Scene Time:**	
Scene – Hospital time:			