PRE-HOSPITAL MANAGEMENT AND PATIENT OUTCOME IN SEVERE TRAUMATIC HEAD INJURY

BY

HELEN L. WEBB

A THESIS

SUBMITTED FOR THE FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

CHARLES STURT UNIVERSITY

2012
ACKNOWLEDGEMENTS

I wish to acknowledge the contribution made by my supervisor, Professor Lexin Wang. I would like to convey my sincere appreciation for the assistance and guidance provided by Professor Wang during his time as my Principal Supervisor.

I acknowledge my Associate Supervisors: Dr Ken Abraham, Dr Marianne Vonau and Dr Ron Manning for their on-going support and encouragement. I thank you: without your assistance, this research would not have been possible. I wish to thank Dr Rosemary Ford for her espousal and guidance.

To Charmane Lofts, I offer my sincere appreciation for her continued support, encouragement, enthusiasm and camaraderie. Charmane was there for me every step of the way. Thank you.

For their assistance with data entry I acknowledge and thank Charmane Lofts, Belinda Bruce, Garrick Burgess, Christine Meek, Tom McPherson and Brinda Rowe.

To my parents, Maureen and Bill, I thank you for your love and support. I am forever grateful. I dedicate this thesis to Maureen, Bill and Charmane.

Helen Webb

July 2012
ABSTRACT

Background
The treatment of persons with severe traumatic head injury often begins with the initial management provided by ambulance paramedics. The early assessment and treatment provided by paramedics is thought to be a crucial step in the ongoing care of individuals with severe traumatic head injury. Appropriate and timely pre-hospital care is an important element which may potentially affect the functional outcome of the patient. In Australia, few studies have been undertaken to investigate the efficacy of critical interventions such as airway management, ventilation, oxygenation and fluid resuscitation on functional outcome of persons with severe traumatic brain injury.

Objective
The objective of this research is to investigate the relationship between pre-hospital management and functional outcome of persons with severe traumatic head injury in the Sydney and metropolitan area.

Methods
Data were retrospectively collected from patient care records of the Ambulance Service of New South Wales patient care records and hospital medical records of 326 patients with severe traumatic head injury. Pre-hospital and in-hospital data were collected and analysed using Statistical Packages for Social Sciences (SPSS) versions 14 to 18. Data was analysed using stepwise linear regression analysis with confidence interval (CI) set at 95% for the regression coefficient. Correlation analysis applied included 2-tailed
Spearman’s rho for categorical variables and 2-tailed Pearson’s correlation for continuous variables. Independent samples t-test was utilised to compare means. All p-values were set at the p < 0.05 level to be considered statistically significant.

**Results**

No significant relationship was demonstrated between the presence of intensive care paramedics at the scene and improved neurological outcome following severe traumatic head injury ($r = -0.005$, $p = 0.931$). Analysis showed the presence of intensive care paramedics at the scene was a significant predictor of scene time greater than or equal to 10 minutes ($r = 0.198$, $p = 0.001$, 95% CI = 0.087 to 0.319). Regression analysis found scene time less than or equal to 10 minutes to be associated with improved neurological outcomes (GOS 4 or 5) ($r = -0.122$, $p = 0.036$, 95% CI = −1.024 to −0.023).

A significant correlation was found between pre-hospital endotracheal intubation and Glasgow Outcome Scale ($r = -0.433$, $p < 0.001$, 95% CI = 1.011 to 1.858). Pre-hospital endotracheal intubation was associated with high incidences of injury severity, morbidity and mortality and was significantly associated with poor neurological outcome (GOS 1, 2 or 3).

Airway obstruction on arrival at the emergency department was observed in 25.2% of patients ($n = 82$). Regression analysis revealed that airway obstruction was a significant predictor of SpO2 range (< 85%; 85-89%; 90-94%; 95-100%) on arrival at the emergency department ($r = -0.247$, $p < 0.001$, 95% CI = −0.728 to −0.266) and SpO2
greater than or equal to 95% on arrival at the emergency department to be a significant predictor of improved outcome \( (r = 0.202, p = 0.001, 95\% \text{ CI} = 0.159 \text{ to } 0.591) \).

A statistically significant correlation for the administration of pre-hospital fluid volume replacement in hypotensive patients (systolic blood pressure < 90 mmHg) and improved outcomes for persons with severe traumatic head injury was not demonstrated in this study. While pre-hospital hypotension was found to be a significant predictor of poorer outcome \( (r = 0.250, p < 0.001, 95\% \text{ CI} = 0.538 \text{ to } 1.342) \), pre-hospital fluid volume replacement did not demonstrate improved outcomes for hypotensive patients with severe traumatic head injury \( (r = 0.019, p = 0.742) \).

**Conclusions**

Analysis failed to support the hypothesis that persons with severe traumatic head injury will benefit from the attendance of intensive care paramedics. Intensive care paramedics were associated with increased scene time, and scene time greater than 10 minutes was a significant predictor of poor outcome. The study found no significant correlation between pre-hospital endotracheal intubation and improved oxygen saturation levels or time to endotracheal intubation and improved outcome.

Patients who experienced pre-hospital hypoxaemia demonstrated poorer outcomes than those who did not experience pre-hospital hypoxaemia. A significant correlation between endotracheal intubation and reduction in hypoxaemia was not demonstrated. Persons with severe traumatic head injury should have oxygen saturations maintained at 95% or
greater during pre-hospital management and all ambulance vehicles should be equipped with pulse oximetry monitoring devices.

Improved outcomes were demonstrated in cases where time at scene was less than or equal to 10 minutes. Time at scene should be limited, where possible, to 10 minutes and patients expedited to a trauma facility. Outcomes for those who received pre-hospital fluid resuscitation were poorer than those who did not receive fluid resuscitation, regardless of the presence or absence of hypotension. Further research is required to investigate the efficacy of pre-hospital fluid volume resuscitation in persons with severe traumatic head injury.
CONFERENCE PRESENTATIONS

Australian College of Ambulance Professionals International Conference
(Auckland, New Zealand, 2009) Webb, H. Do paramedics over manage cervical spine and under manage airway?

Australian College of Ambulance Professionals International Conference

Rural Critical Care Conference

Australian College of Ambulance Professionals National Conference
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Certificate of Authorship

“I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the award of any other degree or diploma of a university or other institution of higher learning, except where due acknowledgement is made in the acknowledgements.”

__________________________

Helen L. Webb
CHAPTER ONE

INTRODUCING THE STUDY

1.1 Introduction

This study investigated the relationship between pre-hospital management and functional outcome of persons with severe traumatic head injury in the Sydney and metropolitan area. All patients enrolled into this study incurred significant injury to the brain as a result of their head injury. For the purposes of this study the term severe traumatic head injury should also signify the presence of a concurrent severe traumatic brain injury (TBI).

Severe traumatic head injury is one of the leading causes of injury and death and as a consequence results in immense financial burden in Western societies. Research in the area of neurological trauma has shown that the totality of damage to the brain following trauma does not occur at the moment of injury, but evolves over hours to days. The brain is subject to further insult from secondary causes including hypoxia, hypocapnoea, hypotension and expansion of the mass lesion (Brain Trauma Foundation, 2000a; Brain Trauma Foundation, 2007). The incidence of mortality due to severe traumatic brain injury has decreased from 50% to 35% to 25% over the past 30 years, largely due to greater understanding of pathophysiological processes in severe brain trauma and advancements in pre-hospital and in-hospital management. The critical interventions for improved outcomes have been in delaying or preventing secondary insults and maintaining cerebral perfusion. Evidence-based guidelines for the management of severe traumatic brain injury have been developed and implemented over the past decade,
particularly within the intensive care unit (ICU) environment (Brain Trauma Foundation, 2007).

However, the efficacy of pre-hospital advanced life support interventions remains uncertain. Pre-hospital care is an inexact science executed in an uncontrolled environment (Boulger & Werman, 2010). Standard management procedure for persons with severe traumatic brain injury varies on a case-by-case basis as a result of divergent scene conditions and characteristics. Multiple variables such as time in the pre-hospital setting, incident location, mechanism of injury, patient injury pattern and severity and paramedic experience and skill level contribute to a varied response to persons with severe traumatic brain injury (Boulger & Werman, 2010).

As a consequence of uncertainty in the efficacy of pre-hospital advanced life support, further investigation is needed. While pre-hospital management of severe traumatic brain injury has become progressively more sophisticated, there are many challenges remaining, especially in the early recognition and treatment of severe brain trauma by ambulance paramedics. The expeditious transport of patients with brain injuries to an appropriate facility and the prevention of secondary insult are of increasing importance in the reduction of mortality and morbidity in brain trauma patients (Badjatia, 2007). It is crucial that paramedics be familiar with the complex presentation of severe traumatic brain injury patients in the initial stages after injury. Early recognition and response to traumatic brain injury can significantly impact on neurological outcome. Paramedics must have a sound knowledge of the interventions which may minimise secondary insult
with pre-hospital treatment of hypoxaemia and hypotension being crucial components of traumatic brain injury management (Badjatia, 2007).

My interest in traumatic brain injury began after observing the poor outcomes of brain-injured persons I had treated and transported as an ambulance paramedic. In several cases not only did I transport the patient to a trauma facility following their accident but also transferred them to a rehabilitation facility or to the brain injury unit following their discharge many weeks later. I became very aware of the considerable time these people had spent in the ICU and of the devastating effects of brain trauma on the individual and their family. As a result of my experiences I wanted to understand more deeply how pre-hospital interventions impacted on those we treated. These events sparked my desire to undertake further study. Therefore, the purpose of this research is to investigate the relationship between pre-hospital management and functional outcome of persons with severe traumatic head injury in the Sydney and metropolitan area.

1.2 The Research Problem

The treatment of persons with severe traumatic head injury often begins with the initial management provided by ambulance paramedics. The early assessment and treatment provided by paramedics is thought to be a crucial step in the ongoing care of individuals with severe traumatic head injury. Appropriate and timely pre-hospital care is an important element which may potentially affect the functional outcome of the patient. Critical interventions in the pre-hospital management of head and brain trauma include airway management, ventilation, oxygenation, fluid resuscitation and the selection of the
hospital destination (Brain Trauma Foundation, 2000b). Few studies have been undertaken to assess the efficacy of these critical interventions on functional outcome and a literature search failed to identify any Australian studies that investigated the pre-hospital management of severe traumatic brain injury. Furthermore, little is known of the efficacy of paramedic clinical judgement and level of clinical skill and the relationship to functional outcome in individuals with severe brain trauma (Badjatia, 2007).

Four research questions were developed to investigate the relationship between the pre-hospital management of persons with severe head injury and their functional outcome at discharge from the acute facility. It was hypothesised that:

1. Persons with severe traumatic head injury will benefit from the attendance of intensive care paramedics and pre-hospital endotracheal intubation provided by intensive care paramedics.

2. Maintenance of a clear and patent airway and maintenance of oxygen saturation of greater than or equal to 95% will benefit patients with severe traumatic head injury.

3. Patients with severe head injuries and hypotension will benefit from early intravenous fluid volume replacement to maintain a systolic blood pressure of greater than 90 mmHg.

4. Functional outcome of patients with head injuries is inversely related to pre-hospital response time, time at scene, the total time in the pre-hospital setting and time to neurosurgical intervention (craniotomy): as pre-hospital time and time to craniotomy increases, functional outcome decreases.
A conceptual model was developed to illustrate the independent and dependent variables considered in this study which are presented in Figure 1.

**Figure 1** Conceptual Model: a Guide for the Study of Pre-Hospital Management of Severe Traumatic Head Injury

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>Dependent Variable</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Airway Management &amp; Oxygenation:</strong></td>
<td></td>
</tr>
<tr>
<td>Patency of airway (airway obstruction on arrival at the emergency department – yes/no)</td>
<td>Glasgow Outcome Scale</td>
</tr>
<tr>
<td>Endotracheal intubation in the pre-hospital setting (yes/no)</td>
<td>1. Death</td>
</tr>
<tr>
<td>Pre-hospital ventilation and oxygenation (SpO2 on arrival at the emergency department)</td>
<td>2. Persistent vegetative state</td>
</tr>
<tr>
<td><strong>Fluid resuscitation and correction of hypotension</strong> (systolic BP &lt; 90 mmHg)</td>
<td>3. Severe disability</td>
</tr>
<tr>
<td><strong>Time in the pre-hospital setting:</strong></td>
<td>4. Moderate disability</td>
</tr>
<tr>
<td>Response time</td>
<td>5. Good recovery</td>
</tr>
<tr>
<td>Scene time</td>
<td></td>
</tr>
<tr>
<td>Treatment time</td>
<td></td>
</tr>
<tr>
<td>Transport time</td>
<td></td>
</tr>
<tr>
<td>Total pre-hospital time</td>
<td></td>
</tr>
<tr>
<td>Time to craniotomy</td>
<td></td>
</tr>
<tr>
<td><strong>Hospital Destination</strong> (trauma centre / non-trauma centre)</td>
<td></td>
</tr>
<tr>
<td><strong>Paramedic Skill Level</strong> (intensive care paramedic / non-intensive care paramedic)</td>
<td></td>
</tr>
</tbody>
</table>
1.3 Significance of the Study

Few studies have been undertaken in the Australian context to investigate the affect of management provided by ambulance paramedics on the outcome of those with severe traumatic brain injury. The significance of this study is to present original information to clarify the efficacy of the pre-hospital interventions on outcome of persons with severe traumatic head injury. Bunn, Kwan, Roberts and Wentz (2001) suggest that if pre-hospital interventions increase the risk of poor outcomes by only a few per cent, the interventions could be responsible for a substantial increase in mortality and morbidity. Similarly, if those interventions were shown to reduce the incidence of poor outcomes by a few per cent, then this would decrease the incidence of preventable death and disability following severe traumatic brain injury.

1.4 Structure of the Thesis

This thesis addresses the above objective through a series of ordered processes and is presented as follows:

Chapter One provides an introduction to the purpose of the research and presents an overview of the scientific rationale for investigation into the pre-hospital management of severe traumatic head injury.

Chapter Two provides an extensive review of literature in the area of severe traumatic head injury. Related subjects under discussion within the review of literature include the epidemiology of severe traumatic brain injury in Australia, trauma systems, functional

Chapter Three explains the study design of a retrospective cohort study and describes the methodology of the study. The research hypotheses are presented in this chapter along with information describing data collection, inclusion criteria, exclusion criteria and the final sample. Details of the pilot study, ethics submissions, area trauma hospital overview and statistical analysis are presented.

Chapter Four presents the information on the characteristics and representativeness of the sample. Age and gender distributions are discussed and the validity and reliability of the Glasgow Coma Scale as an inclusion criteria and the Glasgow Outcome Scale as a measure of outcome are also discussed.

Chapter Five presents the results and discussion for Research Question 1: Persons with severe traumatic head injury will benefit from the attendance of intensive care paramedics and pre-hospital endotracheal intubation provided by intensive care paramedics.
Chapter Six presents the results and discussion for Research Question 2: Maintenance of a clear and patent airway and maintenance of oxygen saturation of greater than or equal to 95% will benefit patients with severe traumatic head injury.

Chapter Seven presents the results and discussion for Research Question 3: Patients with severe head injuries and hypotension will benefit from early intravenous fluid volume replacement to maintain a systolic blood pressure of greater than 90 mmHg.

Chapter Eight presents the results and discussion for Research Question 4: Functional outcome of patients with head injuries is inversely related to pre-hospital response time, time at scene, the total time in the pre-hospital setting and time to neurosurgical intervention (craniotomy): as pre-hospital time and time to craniotomy increases, functional outcome decreases.

Chapter Nine presents the results and discussion for other findings including the effects of age and gender in the outcome of persons with severe traumatic head injury.

Chapter Ten presents the limitations of the study and the key findings and implications for paramedic practice. Recommendations for future research are also presented in this chapter.

Chapter Eleven presents the conclusions of the study.
1.5 Literature Search Methods

Literature search and review was ongoing throughout the project. The literature search commenced in July 1998 and the final review of literature was completed in May 2011. A timeline of the project outlines when literature search and review were undertaken and when the final literature review was completed (refer to Table 1, Project Timeline).

Literature was identified through the following databases; MEDLINE, OVID, MEDLINE PubMed, and CINAHL. Systematic reviews published by the Brain Trauma Foundation; Management and prognosis of severe traumatic brain injury (2000), Guidelines for prehospital management of traumatic brain injury (2000) and Guidelines for the management of severe traumatic brain injury 3rd edition (2007) were reviewed during this study. No ‘gray’ literature was accessed or reviewed.

Database search from 1970 to 2011 was undertaken using the following key words: (“paramedic” or “pre-hospital or “prehospital or “emergency medical technician” or “EMT” or “field” or “ambulance”) and/or (“brain injury” or “head injury” or “intracranial”) and (“head trauma” or “brain trauma” or “trauma” or “severe”) and (“management” or “treatment” or “resuscitation”). Database searches resulted in over 700 articles being cited. Of those 155 were determined to be directly relevant to the study. Literature searches were limited to English language only studies.
<table>
<thead>
<tr>
<th>Year</th>
<th>1998</th>
<th>1999</th>
<th>2000</th>
<th>2001</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Aug: Ethics application approved by ASNSW.</td>
<td>Sep-Oct: Review and refine Data Collection Form.</td>
<td></td>
<td>2. Central Sydney Area Health Service</td>
<td>2. South Western Sydney Area Health Service</td>
</tr>
<tr>
<td></td>
<td>Oct-Dec: Review and revision of Data Collection Form.</td>
<td></td>
<td></td>
<td>3. Western Sydney Area Health Service</td>
<td>3. Hunter Area Health Service</td>
</tr>
<tr>
<td>Year</td>
<td>Activities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>---------------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2003</td>
<td>Apr-Dec: Data collection and data entry into SPSS database</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Apr-Dec: Data collection and data entry into SPSS database</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Apr-Dec: Data collection and data entry into SPSS database</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Jun-Sep: Preliminary data analysis.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Feb-Sep: Leave of Absence from project.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mar-Jun: Completion of revised thesis.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Jul: Submission of thesis for examination.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Feb: Return of examiner reports.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mar-May: Revision and completion of final Review of Literature.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Apr-Dec: Revision and re-writing of thesis.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Aug: Submission of final version of thesis.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Jan-Mar: Revision and restructure of thesis.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Aug: Submission of final version of thesis.</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
CHAPTER TWO

REVIEW OF LITERATURE

2.1 Introduction

This chapter presents an analysis of the literature relevant to the study. First I embed the study within the context of the paramedic response to traumatic brain injury. An historical overview presents the early policy and practice recommendations that were in the paramedicine field at the time of data collection (1997 to 2003). These recommendations are taken from the Brain Injury Foundation (2000a) and the Ambulance Service of NSW (1997) documents, and research evidence in the fields of paramedicine and nursing and medical literature (general and emergency clinical research).

Second, using the conceptual model (Figure 1) as an organising framework, a range of clinical features of brain injury will be presented. The small amount of literature that focuses on paramedics’ pre-hospital interventions is highlighted. The literature begins with an overview of traumatic brain injury and pre-hospital response.

2.2 Overview of Traumatic Head and Brain Injury and Pre-Hospital Response

Severe traumatic brain injury (TBI) is associated with significant mortality and morbidity and is a leading cause of hospitalisation, disability and mortality worldwide (Australian Institute of Health and Welfare, 2011). Stein et al. (2010) undertook a meta-analysis of
207 case series of traumatic brain injury comprising more than 140,000 cases admitted to hospital since the late 1800s. The authors found that mortality associated with severe closed traumatic brain injury had fallen by almost 50% over 150 years. Between the period from 1885 to 1930 mortality fell by a rate of 3% per decade (p < 0.05) and from 1970 to 1990 mortality decreased by 9% per decade (p < 0.05). However, no significant decreases in mortality occurred between 1930 and 1970 and from 1990 onwards. Since 1990 significant improvements in traumatic brain injury survival have not been demonstrated (Stein, Georgoff, Meghan, Mizra & Sonnad, 2010).

The Australian Institute of Health and Welfare estimated that in 1999 there were 338,700 Australians (1.9% of the Australian population) with a disability related to acquired brain injury and of those 160,200 were cases of severe brain injury requiring daily assistance (Brain Injury Centre Australia, 2011). The prevalence of acquired brain injury in Australian States and Territories in 1999 is presented in Table 2.
Table 2  Acquired Brain Injury in Australian States and Territories 1999

<table>
<thead>
<tr>
<th>State or Territory</th>
<th>Number of Residents</th>
<th>Percentage of Total State or Territory Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>New South Wales</td>
<td>113,300</td>
<td>1.8%</td>
</tr>
<tr>
<td>Victoria</td>
<td>72,700</td>
<td>1.9%</td>
</tr>
<tr>
<td>Queensland</td>
<td>74,300</td>
<td>2.6%</td>
</tr>
<tr>
<td>Western Australia</td>
<td>29,200</td>
<td>2.0%</td>
</tr>
<tr>
<td>South Australia</td>
<td>33,600</td>
<td>2.2%</td>
</tr>
<tr>
<td>Tasmania</td>
<td>7,800</td>
<td>1.8%</td>
</tr>
<tr>
<td>Australian Capital Territory</td>
<td>4,700</td>
<td>2.3%</td>
</tr>
<tr>
<td>Northern Territory</td>
<td>3,100</td>
<td>3.6%</td>
</tr>
</tbody>
</table>

(Brain Injury Centre Australia, 2011)

Hospitalisation rates for traumatic brain injury remained stable for the 1999 and 2000 period and increased by 7% during the 2004 and 2005 period. Rate of mortality was estimated at 4.3% of all TBI cases during this period. The estimate of direct cost of hospital care was $184 million during 2004 and 2005 (Australian Institute of Health and Welfare, 2011). Hospital admissions directly related to head injury in 2004 to 2005 exceeded 300 per 100,000 populations (Kennedy & Annunziata, 2004).

Australian statistics are comparable with other developed societies. The Brain Trauma Foundation (BTF) estimates that 1.5 million head injuries occur every year in the United States of America (USA) and 2% of the population of the USA live with disabilities resulting from traumatic brain injury. Hospitalisation rates for traumatic brain injury increased by 8.9% from 79% per 1000,000 to 87.9% per 100,000 from 2002 to 2003. Traumatic brain injury is responsible for 52,000 deaths annually in the USA (Brain
The incidence of traumatic brain injury in the European Union and United Kingdom is estimated at 2.7 million and 1 million per annum respectively (Brain Injury Centre Australia, 2011). Annual rates of traumatic brain injury for Australia, the United Kingdom, United States and the European Union are presented in Table 3.

Table 3  Annual Rates of Traumatic Brain Injury in Australia, United Kingdom, United States and the European Union in 2011

<table>
<thead>
<tr>
<th>Location</th>
<th>Annual Rate of Traumatic Brain Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>135,000 – 160,000</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>1,300,000</td>
</tr>
<tr>
<td>United States</td>
<td>2,000,000 to 2,700,000</td>
</tr>
<tr>
<td>European Union</td>
<td>3,700,000</td>
</tr>
</tbody>
</table>

(Brain Injury Centre, 2011)

The Brain Trauma Foundation first published guidelines for the pre-hospital and in-hospital management of brain trauma in 1995 with revised editions published in 2000 and 2007. The BTF works in collaboration with the American Medical Association, the American Association of Neurological Surgeons (AANS) and the Congress of Neurological Surgeons to review class I, II and III evidence in neurotrauma literature to
formulate recommendations for the management of neurotrauma. The recommendations are general recommendations which reflect published scientific evidence (Brain Trauma Foundation, 2007). The BTF suggests that the pre-hospital management regimes for patients with brain injuries have not advanced to the same extent as those for other trauma and non-trauma conditions (Brain Trauma Foundation, 2007).

Adherence to BTF guidelines for the management of severe traumatic brain injury have increased over the past decade and throughout the world. TBI survey research has shown that increasing numbers of patients with severe traumatic brain injury are being managed according to the Brain Trauma Foundation Guidelines with rates increasing from 32% in 1995 to 78% in 2005 (Brain Trauma Foundation, 2007).

The Ambulance Service of New South Wales (Ambulance Service of NSW) paramedic clinical practice is guided by protocols for all drug administration and procedures which have been developed and accepted as the standard of practice by the Ambulance Service of NSW Medical Advisory Committee and the Medical Director of the Service. These protocols are evidence-based and reflect current treatment regimes and practices (Ambulance Service of New South Wales, 2007). Revision of the protocols was undertaken in 1997, 2007 and 2009 (Ambulance Service of New South Wales, 1997, 2007, 2009). The head injury protocol has remained the same during the three revisions. The protocols stipulate that hypoventilation should be corrected, spinal immobilisation should be considered if there is risk of spinal injury, and urgent transport to a hospital is required if the patient has a depressed level of consciousness. The pre-hospital
management of head injury has remained unchanged over the past decade, and during the time of data collection for this study (Ambulance Service of New South Wales, 1997, 2007, 2009).

Pre-hospital management of persons with severe traumatic brain injury is the first critical link in the continuum of care (Baxt & Moody, 1987; Brain Trauma Foundation 2000b). Neurological damage evolves over minutes to hours following the primary injury so pre-hospital treatment is aimed at minimising the evolution of secondary injury and reducing mortality and morbidity (Brain Trauma Foundation 2000b).

Traumatic brain injury is a major cause of disability in young, previously healthy adults, with males out-numbering females by a ratio of 2:1 and incidences range from 106 per 100,000 population to 180-200 per 100,000 population (Tate, McDonald & Lulham, 1998). Statistics describing the incidence of severe traumatic brain injury in Sydney and metropolitan areas for this study (1997–2003) were not found in the literature. Trauma statistics presented below refer to total incidence of brain trauma, including mild, moderate and severe brain trauma. Sub-categories were not presented as an individual set of statistics.

A key early study conducted in New South Wales (NSW) found the rate of brain injury per 100,000 head of population to be 180 (Lyle, Quine, Bauman & Pierce, 1990). Further analyses of the cohort of patients with brain injuries found the following: 20 per 100,000 died in the pre-hospital setting; a further 160 per 100,000 were hospitalised; 131 per
100,000 sustained minor injuries; 29 per 100,000 sustained serious injuries. Of the 143 per 100,000 population who made an initial recovery, 4.2 per 100,000 sustained a residual moderate disability; 1.9 per 100,000 were left severely disabled; 0.8 per 100,000 remained in a persistent vegetative state and 9.5 per 100,000 died (Lyle et al., 1990).

Actual cases of brain injury were suggested by Lyle et al. (1990) to be 10,500 per year for New South Wales. Of these cases Lyle and colleagues suggested that 2,850 would suffer serious injury with moderate to severe disability and 1,150 would die in the pre-hospital setting. In terms of hospital care, 1,700 persons with serious brain injury would be admitted to hospital and 7,900 would receive hospitalisation for minor injuries. The majority of patients with brain injuries would have some degree of residual disability including 110 with severe disability, 245 with moderate disability and 45 would remain in a persistent vegetative state (Lyle et al., 1990).

Hillier, Hiller and Metzer (1997) in their study of brain injury in South Australia reviewed a wider range of International Classification of Diseases (ICD-9) codes and estimated the rate of brain injury per 100,000 population to be 322. The distribution of cause of injury by gender is outlined in Table 4.
### Table 4  
**Cause of Injury by Gender**

<table>
<thead>
<tr>
<th>Cause</th>
<th>Males</th>
<th>Females</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fall</td>
<td>34 (28%)</td>
<td>18 (33%)</td>
<td>52 (29%)</td>
</tr>
<tr>
<td>Car Driver</td>
<td>36 (29%)</td>
<td>13 (24%)</td>
<td>49 (28%)</td>
</tr>
<tr>
<td>Car Passenger</td>
<td>10 (8%)</td>
<td>12 (22%)</td>
<td>22 (12%)</td>
</tr>
<tr>
<td>Pedestrian</td>
<td>12 (10%)</td>
<td>6 (11%)</td>
<td>18 (10%)</td>
</tr>
<tr>
<td>Assault</td>
<td>14 (11%)</td>
<td>2 (4%)</td>
<td>16 (9%)</td>
</tr>
<tr>
<td>Motorcyclist</td>
<td>11 (9%)</td>
<td>1 (2%)</td>
<td>12 (7%)</td>
</tr>
<tr>
<td>Drug Abuse</td>
<td>1 (1%)</td>
<td>1 (2%)</td>
<td>2 (1%)</td>
</tr>
<tr>
<td>Gunshot</td>
<td>1 (1%)</td>
<td>0 (0%)</td>
<td>2 (1%)</td>
</tr>
<tr>
<td>Other</td>
<td>4 (3%)</td>
<td>1 (2%)</td>
<td>5 (3%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>123 (100%)</td>
<td>54 (100%)</td>
<td>177 (100%)</td>
</tr>
</tbody>
</table>

(Hillier, Hiller & Metzer 1997, p. 653)

Similarly the Brain Trauma Foundation (2007) has identified falls, motor vehicle crashes and assaults respectively as the leading causes of TBI in the USA, and males are twice as likely to experience TBI (Brain Trauma Foundation 2007).

The NSW Department of Health (2004) identified similar trends to Lyle et al. (1990). The NSW Department of Health (2004) in the Metropolitan Trauma System Monitoring Report found trauma to be the major cause of death in the 15 to 44 year age group; representing more than 50% of all serious injuries. The ratio of males to females with serious traumatic injury was found to be 3:1 respectively. Further, one quarter (average 8 per week) were incorrectly transported to a district hospital rather than an area trauma hospital in the Sydney and metropolitan area. Other estimates suggest that the rate of traumatic brain injury is approximately 150 per 100,000 population per year, with 12-14 per 100,000 population being severe brain injury (Khan, Baguley & Cameron, 2003).
Hillier, Sharpe and Metzer (1997) reviewed functional outcomes five years after traumatic brain injury ($n = 67$). The study was undertaken in South Australia and data were collected using medical records, personal interview/questionnaire and neurophysical assessment. The aims of the study were to investigate broad outcomes following traumatic brain injury as well as the prevalence of any residual physical impairment or disability. Study participants’ living arrangements were found not to change during the post-injury period, and nearly half had returned to some form of paid work (the remainder were reliant on the welfare system). Fifty seven percent (57%) of participants reported functional improvements in all areas, while others reported partial improvement (19%) or deterioration (8%). The most commonly reported residual impairments were headaches, balance difficulties and fatigue or weakness, while upper limb deficit (30%) and transfer difficulty (9%) were also reported. Participants reported residual deficits in mobility (34%): namely, overall balance impairment and altered gait and of those, 9% were reliant on wheelchairs for mobility (Hillier et al., 1997). The results of this study highlight the considerable residual deficit experienced by more than half of individuals in this cohort following severe brain injury.

Further, according to Myburgh et al. (2008), mortality associated with traumatic brain injury has remained at approximately 30% to 35% in developed countries over the past 20 years despite improvements in pre-hospital management, diagnostic technology, intensive care and extensive research into the pathology of primary brain injury. The literature reviewed (Kennedy et al., 2004; Brain Injury Centre Australia, 2011; Australian Institute for Health and Welfare, 2011; Stein et al., 2010; Lyle et al., 1990; Khan et al.,
2003 & Hillier et al, 1997) indicate the rates of morbidity and mortality associated with traumatic brain injury to be high. As such, further investigation into the development, evolution and management of severe brain injury is required.

### 2.2.1 Trauma Systems

Ambulance Service of New South Wales (1997, 2007) pre-hospital trauma guideline stipulates that persons with severe traumatic brain injury be transported directly to a trauma centre, even if the trauma centre is not the closest hospital. This focus on transport to a trauma centre is intended for persons with one or more of the following indicators: head injury with depressed level of consciousness (Glasgow Coma Score 14 or less), serious trauma to any body region, systolic blood pressure less than 90 mmHg, and/or respiratory distress (rate < 10 or > 30 breaths per minute).

According to Ambulance Service of New South Wales (1997, 2007) pre-hospital trauma guidelines the transportation of persons with head injury and a depressed level of consciousness should be considered urgent and emergency beacons and sirens should be used during transportation. The 1997 protocol indicates that paramedics should carry out intubation, defibrillation, haemorrhage control and cannulation at the scene prior to transportation (Ambulance Service of New South Wales, 1997). In the 2007 protocol cannulation has been removed. Both the 1997 and 2007 protocols suggest that if transport time to hospital is very short then treatment should be given en route rather than at the scene. However, actual time frames or distances are not specified in either protocol (Ambulance Service of New South Wales, 1997, 2007).
Early studies in San Diego County in the USA (Shackford, Hollingworth-Fridlund, Cooper & Eastman, 1986) and in Quebec, Canada (Sampalis et al., 1995) found improved outcome for trauma patients who had expeditious transport to a trauma centre. Sampalis et al. (1997) studied the impact of direct transport to a trauma centre versus transfer from a lower level facility on mortality and morbidity among patients with major trauma ($n = 2,756$). The majority of patients in the study (63%) were transported directly to a trauma centre and 1,608 patients (37%) were transferred from a lower level facility. The direct and transfer groups were similar with respect to age, gender, body regions injured and mechanism of injury. They found significantly lower risk for overall mortality for the direct transport group (4.8%) compared with the transfer group (8.9%) (odds ratio, 1.96, 95% CI, $p < 0.003$). Emergency room mortality was significantly higher in the transfer group (transfer 3.4%; direct 1.2%; odds ratio, 2.96, 95% CI, $p < 0.003$). Mean time in the intensive care unit (transfer, 2.0 days; direct 0.95 days, $x^2$, $p = 0.001$) and in hospital admission (transfer, 16.0 days; direct 13.2 days, $x^2$, $p = 0.02$) was significantly shorter for patients who were taken directly to a trauma centre.

A multicentre study was undertaken by Harlt et al. (2006) to explore pre-hospital management decisions on early mortality of patients with traumatic brain injury in New York State between 2000 and 2004 ($n = 1,123$). After controlling for arterial hypotension, age, pupillary status and initial Glasgow Coma Score recorded by paramedics, a significantly lower mortality was found in the direct transport group. Logistic regression showed an association between direct transport to a trauma centre and a lower 2-week mortality compared with non-direct transport (odds ratio = 1.48, CI 95%, $p < 0.04$). A
50% increase in mortality was associated with the indirect transfer group. Variables such as transport mode (air versus ground), time to admission to a trauma centre and pre-hospital intubation showed no association with improved outcomes.

In summary, it can be seen that direct transport to a trauma facility is associated with reduced mortality in trauma patients. However other determinants of survival are not identified in the available literature. The current study investigates this issue by analysing the impact of variables including paramedic level of clinical skill and maintenance of respiratory and cardiovascular function on functional outcome.

2.2.2 Paramedic Triage and Clinical Judgement

The ability of paramedics to effectively triage trauma patients is uncertain. Shaban, Wyatt Smith and Cumming (2004) suggest that research into human clinical judgement in medicine and health is mainly limited to the medical and nursing professions. Much of ambulance practice including paramedic judgement and decision-making have not undergone systematic and sustained research and therefore there is a paucity of evidence that examines the mechanics of human error in paramedic practice. Further, paramedic practice is regulated by clinical practice guidelines, protocols and procedures. Paramedics are not independent practitioners and must work within specified guidelines, therefore the relationship between knowledge, judgement and decision making has not been established.
Mulholland, Gabbe and Cameron (2005) critically reviewed 12 studies of paramedic practice and found no clear evidence that paramedics can accurately triage severe traumatic injury and paramedics’ ability to predict anatomical injuries requiring trauma services is largely unknown. The authors could not determine paramedic processes for reaching triage decisions, and postulated that paramedics gauge injury severity using the Glasgow Coma Scale, decreased blood pressure, mechanisms of injury, anatomic injury and penetrating injury. The ability to triage accurately is thought to be a complex relationship between clinical experience, repeated clinical assessment of trauma patients and feedback from trauma experts (Mulholland et al., 2005).

Early research in the USA (Billittier, Lerner, Moscati & Young, 1998) investigated the accuracy of paramedic triage, transportation and destination decisions. A survey consisting of 14 patient care scenarios was administered to paramedics \( n = 311 \) from 20 randomly selected ambulance services. The paramedics with less experience and/or were operating at a basic or intermediate life support level (BLS/ILS) \( n = 108 \) demonstrated a mean correct score of only 32.6%. Whereas paramedics operating at advanced life support (ALS) level \( n = 203 \) had a mean correct score of 41.1%. Using Spearman correlation analysis, variables found to correlate with improved decision making were: agency call volume \((r = 0.20, p < 0.001)\), agency level of service \((r = 0.27, p < 0.001)\), agency transports \((r = 0.25, p < 0.001)\), subject provider level \((r = 0.39, p < 0.001)\), subject hours in emergency medical systems \((r = 0.25, p < 0.001)\) and subject provider type \((r = 0.23, p < 0.001)\). Correlation analysis suggests that improved decision-making amongst paramedics was associated with high call volume and higher level clinical
training. Results of the Billittier et al. study suggest that out-of-hospital providers frequently make inappropriate triage, transportation and destination decisions when presented with mock scenarios. However, the results may be limited due to the sizeable variation amongst the agencies and therefore care should be exercised when extrapolating findings to the total population of paramedics in the USA or to paramedic populations in other countries.

In the Sydney and metropolitan areas paramedics are dispatched to the scene of an incident involving severe traumatic head injury in accordance with a number of factors. Importantly, the closest available ambulance crew will be initially responded to the scene which may be an intensive care crew or a non-intensive care crew. Dual response, with at least one intensive care crew, may be initiated by the ambulance dispatcher if sufficient crews are available and the dispatcher considers from the information available that intensive care interventions are required; or a responding non-intensive care crew who arrive at the scene first may request intensive care paramedics to be responded based on the Ambulance Service triage principles and presenting injuries. Ambulance crews may choose to wait at scene for intensive care assistance or to expedite rapid transportation of the patient under the auspice of the Urgent Transport protocol (Ambulance Service of New South Wales, 1997).

The current study investigates the issue of paramedic triage and clinical judgement further by analysing the triage and transport decisions of a sample of paramedics from a
single ambulance service in a large metropolitan area on patient outcome following severe head injury.

2.3 Severe Traumatic Head Injury – Clinical Features

Clinical features of severe traumatic head and brain injury include reduced consciousness and cognition, alteration to oxygen saturation causing hypoxaemia, alteration to carbon dioxide levels causing hypercapnoea or hypocapnoea, and alteration to intracranial pressure, mean arterial blood pressure (MABP) and cerebral perfusion pressure (CPP) (Brain Trauma Foundation, 2000a). The following section provides an overview of key pathophysiological features evident in severe brain injury, the assessment measures used in the pre-hospital treatment and the validity of these measures.

2.3.1 Cerebral Blood Flow (CBF), Cerebral Ischaemia and Cerebral Perfusion Pressure (CPP)

It is thought that the most important secondary occurrence influencing outcome following severe traumatic brain injury is cerebral ischaemia. Cerebral ischaemia may result from impaired cerebral blood flow (CBF), post-traumatic vasospasm and alterations in blood pressure, metabolic auto-regulation, and in alterations in cerebral vascular resistance (Brain Trauma Foundation, 2000a). Following traumatic brain injury, CBF is usually very low and may approach the ischaemic threshold; in addition, CBF in the area of subdural haematomas and/or post-traumatic contusions is reduced to a greater degree than global CBF. Post-traumatic vasospasm is thought to occur in as much as 40% of persons with severe traumatic brain injury. Other causes of reduced CBF are compressed cerebral
vessels, the effect of mass lesions and reduced cerebral metabolism (Brain Trauma Foundation, 2000a).

A relationship between cerebral blood flow and neurological outcome following traumatic brain injury was established using the xenon intravenous method. Bouma et al. (1991) studied arteriovenous oxygen difference (AVDO2), cerebral blood flow, cerebral metabolic rate of oxygen (CMRO2) and motor score in patients with brain injuries \( (n = 153) \) (Bouma et al., 1991). CBF measurement was taken at 4, 8, 12 and 24 hours post-injury. It was found that CBF rates were significantly lower at 4 hours post-injury than at any other time (Newman-Keuls’ test, \( p < 0.05 \)). CBF gradually increased over the 24-hour period to hyperaemic levels. AVDO2 however, was significantly above average at the 4-hour period and rapidly decreased over the 24-hour period. Significantly higher AVDO2 values were found in patients with low motor scores (GCSM 1 or 2) and correlation was found between CBF and motor score (Spearman correlation coefficient, \( r = 0.69, p < 0.001 \)). Higher mortality was also found in the group of patients with low CBF (Chi square test for trends, \( p < 0.04 \)).

Ischaemia following brain trauma therefore occurs in early stages following injury, generally in the first few hours, and ischaemia then subsides over 24 hours. Early ischaemia is considered an important factor in determining neurological outcome with maintenance of CBF crucial in the management of severe traumatic brain injury. The Bouma et al. study presents robust evidence relating to CBF post trauma, however intermittent monitoring may fail to spot transitory periods of ischaemia and the xenon
intravenous method capacity to accurately measure deeper areas of the cerebral circulation is uncertain.

Cerebral perfusion pressure (CPP) refers to the pressure gradient within cerebral vasculature that directly influences cerebral blood flow and delivery of essential metabolic requirements for cerebral tissue. CPP is determined by mean arterial blood pressure (MABP) minus intracranial pressure (ICP): CPP = MABP – ICP. CPP less than 70 mmHg are thought to have deleterious effects on areas of the brain with pre-existing trauma (Brain Trauma Foundation, 2000a).

CPP is directly related to mean arterial blood pressure. Episodes of hypotension (systolic BP < 90 mmHg) have been linked to deleterious outcomes (Brain Trauma Foundation, 2000a). With a drop in systolic and diastolic BP, a concurrent drop in mean arterial BP occurs. Blood pressure is maintained through the combined use of vasopressor agents and intravenous infusion of fluids and/or blood products within the in-hospital setting (Brain Trauma Foundation, 2000a). In the pre-hospital setting, blood pressure is maintained through the manipulation of intravascular volume via the administration of fluid, predominantly crystalloid infusion (Ambulance Service of New South Wales, 1997).

CPP is now considered to be the most important factor in determining a sufficient partial pressure of oxygen within cerebral tissue. A CPP of greater than 70 mmHg is recommended to ensure an adequate partial pressure of oxygen. To maintain CPP greater
than 70 mmHg, hypotension (systolic blood pressure < 90 mmHg) must be avoided (Brain Trauma Foundation, 2000a).

McGraw (1989) in a retrospective analysis of patients with severe traumatic brain injury \((n = 221)\) found a CPP of 80 mmHg or greater was associated with improved outcomes. The relationship between the initial CPP and outcome at one year post-injury were analysed. It was found that if CPP was maintained at a level greater than 80 mmHg, mortality rates were reduced and the likelihood of improved outcome increased. When CPP was maintained at levels greater than 80 mmHg, the mortality rate was 35%-40%. For those patients whose CPP dropped below 80 mmHg, mortality rates increased by 20% for each 10 mmHg drop in CPP. CPP less than 60 mmHg was associated with a mortality rate of 95%.

The issue of maintenance of blood pressure and avoidance of hypotension through fluid resuscitation in the pre-hospital setting will be further investigated in this study. The efficacy of fluid resuscitation with both crystalloid and colloid fluids (Hartmann’s Solution and Haemaccel) in the presence or absence of hypotension will be analysed.

**2.3.2 Intracranial Pressure (ICP)**

Marmarou et al. (1991) studied the relationship between raised intracranial pressure, hypotension and outcome in patients with severe brain injuries who underwent intracranial pressure monitoring \((n = 428)\). The research was a prospective study that obtained observational data from the Traumatic Coma Data Bank. Outcome was
measured using the Glasgow Outcome Scale at 6 months post-injury. The study considered ICP readings from 0 – 80 mmHg by increments of 5 mmHg and systolic blood pressure from 120 – 20 mmHg by increments of −5 mmHg. Stepwise regression and chi-square analysis revealed an ICP > 20 mmHg and systolic BP < 80 mmHg were significantly associated with poorer neurological outcome (p < 0.001 for both variables). Results suggest that an ICP of 20 mmHg or greater and a systolic BP of 80 mmHg or less were the critical levels that were most indicative of poorer outcome.

The Brain Trauma Foundation (2000a) considered the Marmarou et al. study to contribute clinically valuable results due to its prospective design, a relatively large sample (n = 428) and the capacity to control for confounding prognostic variables. The study was influential in establishing the optimal predictive values of an ICP of 20 mmHg and systolic BP of 80 mmHg on outcome.

Other studies, while not as robust as the Marmarou et al. study due to their retrospective design and small sample sizes, also supported the premise of an ICP of greater than 20 mmHg as a predictor of poorer outcome (Eisenberg, Frankowski, Contant, Marshall & Walker, 1988; Marshall, Smith & Shapiro, 1979; Saul & Ducker, 1982). Eisenberg et al. (1998) studied the efficacy of high dose barbiturate therapies in patients with head injuries (n = 73) to control ICP. ICP control threshold was set at 20 mmHg. The authors noted improved outcomes when ICP was controlled at 20 mmHg or less. Similarly, Marshall et al. (1979) in their retrospective review of patients with head injuries (n = 100) found an ICP of 15 mmHg or less to be associated with improved outcome. Saul and
Ducker (1998) found an ICP of 25 mmHg or greater associated with high mortality rates (84% mortality rate, p < 0.001) in their retrospective study of 127 patients with severe head injuries who underwent ICP monitoring.

Raised ICP (ICP > 25 mmHg) has been associated with diminished functional outcome following severe traumatic brain injury (Marmarou et al, 1991; Bourma et al., 1991; Saul et al., 1998). ICP was initially thought to be positively related to blood pressure. Studies have since failed to establish a direct positive relationship between ICP and blood pressure (Brain Trauma Foundation, 2000a).

While the relationship between blood pressure, ICP and CBF remains uncertain, the weight of evidence suggests an ICP of less than 20 mmHg and a systolic blood pressure of greater than 80 mmHg may be associated with improved outcomes (Eisenberg et al., 1998; Saul et al., 1998). While some findings suggest a systolic blood pressure of greater than 80 mmHg is associated with improved outcomes (Eisenberg et al., 1998; Saul et al., 1998) and the Brain Trauma Foundation suggest that to maintain CPP greater than 70 mmHg, a systolic blood pressure < 90 mmHg must be avoided (Brain Trauma Foundation, 2000a). The disparity in what is recommended as the minimum systolic blood pressure in the presence of brain trauma intimate that further research is required to substantiate the impact of blood pressure on cerebral perfusion pressure and cerebral blood flow. Current practice in the pre-hospital management of severe TBI is to correct hypotension (BP < 90 mmHg) and this management aim has not changed for several years (Ambulance Service of New South Wales, 1997, 2007).
The subject of raised ICP and pre-hospital management will be investigated in this study. The relationship between the independent variables of time in the pre-hospital setting, pre-hospital resuscitation, hospital destination and time to craniotomy, and the dependent variable of functional outcome (GOS) will be analysed.

2.3.3 Hypotension

Hypotension (systolic blood pressure < 90 mmHg) has been associated with poor neurological outcome following severe traumatic brain injury (Brain Trauma Foundation, 2000a). Following review of outcomes in several studies the Brain Trauma Foundation (2000a) found the 5 most powerful predictors of outcome in traumatic brain injury were hypotension, age, intracranial lesion diagnosis, pupillary reactivity and post-resuscitation Glasgow Coma Scale result.

Chesnut (1997) prospectively studied individuals with severe brain trauma (n = 717) and compared outcome among those with neither pre-hospital hypotension nor hypoxia, those with pre-hospital hypoxia alone, those with pre-hospital hypotension alone and those with both pre-hospital hypoxia and hypotension. Pre-hospital hypotension was found to be singularly the most significant predictor of poor outcome. Patients who were hypotensive at the time of admission had a mortality rate twice that of patients who were not hypotensive. The mortality rate for those with hypotension alone was 60.2%; hypoxia alone, 33.3%; neither, 27.0%; and both hypotension and hypoxia was 75.0%. The presence of hypotension and hypoxia in the field was strongly associated with increased risk of mortality. Chesnut (1997) recommends that avoidance or minimisation of
hypotensive episodes in the pre-hospital and acute phases of severe brain trauma has the highest likelihood of improving outcome as any one single therapeutic intervention. This study with a large sample size demonstrated the deleterious effects of pre-hospital hypotension on neurological outcome in severe TBI. A limitation of the study was that the magnitude and duration of episodes of hypotension in individual patients were not considered. However, the study did establish that any episode of pre-hospital hypotension (systolic BP < 90 mmHg) was associated with poorer outcome.

Pietropaoli et al. (1992) retrospectively selected a cohort of 53 patients with head injuries for analysis of outcome in the presence or absence of intraoperative hypotension. Data was collected from the Department of Surgery Head Injury Data Base. Intraoperative hypotension was defined as systolic blood pressure less than 90 mmHg. All patients were normotensive on arrival at hospital and none had experienced pre-hospital hypotension. Of the 53 patients, 17 experienced intraoperative hypotension (32%) and 36 remained normotensive throughout surgery (68%). Significantly higher injury severity scores and lower admission GCS were noted in the intraoperative hypotension group. No patient in the intraoperative hypotension group had a good outcome and it was established using Fisher’s Exact Test that intraoperative hypotension was inversely related to Glasgow Outcome Score (R = –0.30, R² = 0.009, F = 5.2, p = 0.02). Intraoperative hypotension in excess of 5 minutes occurred in 14 patients and of those, 13 died and 1 patient was severely disabled. Within the entire intraoperative hypotensive group, a mortality rate of 82% was noted as compared with a mortality rate of 25% in the normotensive group. While this study was retrospective and had a small sample size, the findings were
comparable to the more robust study of Chesnut (1997). Episodes of hypotension in brain trauma patients were associated with poorer outcomes and high mortality rates.

Shackford (1995) suggests that not only an incident of hypotension but also the duration of hypotension post-brain injury is an important factor in determining outcome. Persons suffering hypotensive post-traumatic brain injury should be treated aggressively to restore cerebral blood flow (CBF) and cerebral perfusion pressure (CPP) in order to prevent secondary insults. Shackford (1995) warns that treatment of hypotension requires rational and judicious use of volume expanders as over-infusion can also lead to reduced cerebral perfusion pressure. Over-infusion of volume expanders causes an increase in intracranial pressure (ICP). As ICP rises, CPP falls, leading to the commencement of a secondary insult. Inadequate fluid infusion also leads to secondary insult and reduction of CPP through a different mechanism. Inadequate fluid resuscitation causes a reduction in MABP and therefore a reduction in CPP. It is therefore imperative that hypotension in the pre-hospital setting be corrected as early as possible and that over-infusion not occur.

However, while the exact relationships between blood pressure, ICP, CCP and MABP are uncertain, there is consensus in several studies that a systolic blood pressure of less than 90 mmHg is associated with deleterious outcomes and correction of hypotension should occur as soon as possible in individuals with TBI (Chesnut, 1997; Pietropaoli et al., 1992; Brain Trauma Foundation, 2000a). The Brain Trauma Foundation (2000a, 2007) recommends that systolic blood pressure be maintained above 90 mmHg; however, the upper limits of appropriate systolic blood pressure have not been established. Cerebral
perfusion pressure is possibly more important than systolic blood pressure, as systolic blood pressure can be an unreliable indicator as the relationship between CPP and systolic blood pressure is not consistent. The actual target values of upper and lower systolic blood pressure in the management of severe traumatic brain injury remain unclear (Brain Trauma Foundation, 2007).

2.3.4 Age and Gender Relationships in Severe Traumatic Head Injury

Age is thought to impact on outcome from traumatic head and brain injury for several reasons. Firstly as the brain ages, its ability to recover from TBI is reduced and secondly, the aged are predisposed to co-morbid conditions and complications post-injury (Brain Trauma Foundation, 2000a).

Pennings, Bachulis, Simons and Slazinski (1993) used a retrospective cohort study to compare outcome following severe trauma of 42 older patients (60 years or greater) with 50 younger patients (aged 20-40 years). It was observed that differences in mechanism of injury and pattern of brain injury existed between the two cohorts. The older patients were predominately injured as a result of a fall (52%), compared with only 6% in the younger patient group. Injuries in the younger group were largely due to high speed motor vehicle accidents (80%), compared with 24% in the older group. In the younger group the ratio of males to females was 4:1 as opposed to 2:1 in the older group. The older group also had a higher incidence of subdural haematoma (52% vs 34%), intracerebral haematoma (41% vs 30%), midline shift (26% vs 14%), cerebral contusion (40% vs 26%) and 3 or more lesions (52% vs 24%).
Cause of death in all younger patients was solely attributed to brain injury whereas 33% of deaths in the older group were attributed to pulmonary, cardiac or multisystem organ failure. Only one of the elderly patients experienced a good outcome, compared with 59% of the younger group. That elderly patient was aged 61 years had a good recovery from an isolated epidural haematoma. Decreased physiological reserves of the ageing brain and other organ systems, and higher complication rates in the elderly were cited as reasons for the differences in outcome (Pennings et al., 1993).

Vollmer et al. (1991) had similar findings to Pennings et al. (1993) in their prospective study of 661 patients with traumatic coma. Results showed that advancing age is directly related to poorer outcome following traumatic coma, with a marked increase in mortality for patients 55 years of age or greater. Older groups had higher rates of pre-existing systemic disease, and mass lesions were more common in older patients. Trend analysis showed significant relationship between advancing age and outcome ($x^2$, $p < 0.001$) across all age groups.

Functional outcome of elderly patients (age 65 years or greater) with acute head injury was considered by Ritchie, Cameron, Ugoni and Kaye (2000). Outcome was measured by overall mortality and the Glasgow Outcome Scale. The inclusion criteria were met by 191 patients. Of those, 59 had a GCS of $<11$ on arrival at hospital and 132 presented with a GCS $\geq 11$. The overall mortality rate was 33.5% ($n = 64$). It was observed that all 59 patients with a GCS $< 11$ had poor outcomes compared with 41 of the 132 patients with a GCS $\geq 11$ (Fisher’s Exact Test, $p < 0.001$). Poor outcome was measured as a GOS 1, 2 or
3 and satisfactory outcome a GOS 4 or 5. No significant differences were observed between males and females. It was concluded that elderly patients presenting with a GCS < 11 have poor functional outcomes following acute head trauma.

Masson et al. (2003) considered outcome in 248 patients with severe brain trauma in the south-west region of France. It was found that 128 (52.0%) of the patients died, with 13.3% of the deaths occurring within 2 hours post-injury, 17.2% occurring within 6 hours, 39.9% within 24 hours and 50% within 36 hours following injury. In those who died, the median age was higher (48 years) than those who survived (33 years). Death rate was highest in patients over 55 years-of-age (70.5%).

While the effect size is uncertain, all studies indicated that advancing age is associated with poorer outcome following TBI (Pennings, Bachulis, Simons & Slazinski, 1993; Vollmer et al., 1991; Ritchie, Cameron, Ugoni & Kaye, 2000; Masson et al., 2003).

Gender is also thought to be related to outcome following severe traumatic brain injury, with women displaying worse outcomes than men in several studies (Farace & Alves, 2000; Kraus, Peek-Asa & McArthur, 2000). A meta-analysis of gender differences in eight studies of outcome in traumatic brain injury revealed that women had poorer outcome in 85% of the 20 measured variables classified by gender; however, the size effect for the meta-analysis was relatively small (F = −0.15) (Farace & Alves, 2000). While the exact relationship was unknown, suggested gender differences that may have affected outcome include pre-morbid factors, symptom reporting, injury factors,
cognition and psychosocial factors, gender differences in brain function, sex hormones and treatment effects (Farace & Alves, 2000).

A prospective cohort study of patients with severe to moderate head injuries over a 3.5 year period was undertaken by Kraus et al. (2000). Data was collected from two medical teaching centres in Los Angeles, California. Glasgow Outcome Scale was used to measure outcome at discharge and at 18 months post-discharge. A total of 795 patients with brain injuries were studied with 652 (82.0%) being male and 143 (18.0%) being female. Mortality rates in females were found to be higher than in males (75.8% versus 69.0% respectively) and females were 1.75 times more likely to die than males. Females were also 1.57 times more likely than males to have poorer outcomes (persistent vegetative state or severe disability); however, statistical significance was not achieved using logistic analysis.

Of the initial 795 brain injured patients, 313 were assessed at 18 months post-discharge with 263 being male (84%) and 50 being female (16%). Poor outcomes were more prevalent in females (60.0%) than in males (51.4%) (Kraus et al., 2000). It should be noted that the sample size in this study was extremely uneven. Males were over-represented by a ratio of as much as 16:1. The vast difference in numbers of males versus females may have confounded results. A more equitable sample distribution of males and females would be required before the results could be generalised to the total population of brain-injured individuals.
When studying epidemiological accounts of brain trauma, Slewa-Younan, Green, Baguley, Gurka and Marosszeky (2004) found that men are significantly over-represented in severe traumatic brain injury samples of subjects less than 65 years of age. In order to obtain an equal sample the authors identified 54 females who had been involved in high speed motor vehicle collisions from the Brain Injury and Rehabilitation Data Base at the Westmead Hospital in Sydney, Australia. The female sample was then matched by age, years of education, speed of motor vehicle collision and mode and type of injury to a sample of 54 males from the same data base. All patients had been involved in high speed motor vehicle collisions between 1990 and 1993. No significant difference in Glasgow Outcome Score was found between men and women; however, men had significantly greater levels of injury. Results of a two-tailed Mann-Whitney U Test found men within the under-65 age group demonstrated higher injury severity, with injury severity determined by lower GCS scores ($U = 971.5, p < 0.005$) and longer periods of post-traumatic amnesia ($U = 858.0, p < 0.05$).

The precise relationship between gender and outcome following severe brain trauma remains unclear. Studies by Farace and Alves (2000) and Kraus et al. (2000) found women to have poorer outcomes that men following brain trauma; however, the sample sizes were considerably uneven with men appreciably out-numbering women; which may have resulted in type I error. Conversely, Slewa-Younan et al. (2004), when controlling for disproportionate sample sizes between males and females, found no significant differences in Glasgow Outcome Scale between the genders. However, the sample size was much smaller in this study. Further studies with larger samples would be required to
corroborate the findings. The functional outcome following severe head injury of males compared with females will be analysed in this study.

2.4 Measurement of Injury Severity in Severe Traumatic Head Injury

2.4.1 Glasgow Coma Scale (GCS)

The Glasgow Coma Scale was developed by Teasdale and Jennet (1974) to assess the depth and duration of impaired consciousness or coma. The scale measures 3 aspects of behaviour: motor responsiveness, verbal performance and eye opening. The GCS facilitates communication between different personnel managing brain injuries, for example, paramedics, nurses and doctors. The GCS is used in the acute stages for triage of patients from the scene of the incident to the receiving hospital to enable timely assessment and treatment regimes. Frequent assessment of impaired consciousness using the GCS has resulted in improved outcomes by decreasing the frequency of avoidable secondary insults due to delays in treatment. Avoidable delays in treatment are the most common cause of death and disability following brain trauma (Jennett, 2002).

The GCS should be assessed as soon as possible following injury and before sedation and endotracheal intubation (Jennett, 2002). The use of sedating and paralysing agents and endotracheal intubation in the pre-hospital setting is thought to taint the accuracy of the GCS (Marion & Carlier, 1994; Brain Trauma Foundation, 2000a). In addition, a multi-centre Scottish study \((n > 13,000)\) concluded that pre-sedation GCS was superior in determining the level of impaired consciousness (Jennett, 2002). However, the Ambulance Service of New South Wales does not permit the use of paralysing and sedating agents to facilitate endotracheal intubation and therefore the accuracy of GCS
assignment is not affected by the administration of pre-hospital pharmacology in NSW (Ambulance Service of New South Wales, 1997).

The GCS can be used in combination with other measures used to predict functional outcome following brain trauma. Such prediction measures can include the Injury Severity Scale, Glasgow Outcome Scale, Disability Rating Scale and Post-Traumatic Amnesia Scale (Jennett, 2002).

The GCS has been used with the Injury Severity Scale (ISS) to predict outcome in multi-trauma patients with head injury. Pal, Brown and Fleiszer (1989) found that using the two measures provided physicians with an accurate assessment tool for predicting outcome in traumatic brain injury. A retrospective cohort study of 170 patients was undertaken to determine to efficacy of the GCS and ISS in outcome prediction. Outcome was measured using the Glasgow Outcome Scale. In the cohort, 56 (35%) were mildly injured (ISS 0–9), 104 (61.2%) were moderately injured (ISS 20–50) and 10 (5.8%) were severely injured (ISS > 50). It was found that the more injured the patient and the higher the ISS, the lower the GCS, indicating a deeper level of impaired consciousness or coma. One month post-injury, a good recovery was seen in 86 of the 87 (98.9%) patients with a GCS of 15–13. A good recovery was only seen in 70.8% of the 24 patients with a GCS of 12–9, while mortality in this group was 12.5%. A mortality rate of 40.7% was noted in the patients with a GCS of 8 or less. Of the 59 in this group 23.8% (14 patients) had a poor recovery and only 35.5% (21 patients) experienced a good recovery. Findings suggest the GCS values of 8 or less could be used as a predictor of mortality (Pal et al., 1989). While
the findings support the premise that GCS can be used in conjunction with the ISS to predict outcome in multi-trauma patients with head injury, the sample size in this study was relatively small \( (n = 170) \) and the results should therefore be considered in combination with other studies before extrapolating findings to the general population of patients with traumatic head injuries.

Ross, Leipold, Terregino and O’Malley (1998) in a retrospective analysis of 1,410 adult patients transported to a Level 1 Trauma Centre found no statistical difference in the GCS and the GCS Motor (GCSM) score in predicting outcome. The motor component of the GCS was found to be a robust predictor of outcome. The authors postulated that GCSM could replace the GCS in pre-hospital triage of severe brain injury. More recent studies have corroborated the findings of Ross et al. (1989). Moore et al. (2006) found the GCS to be a strong predictor of in-hospital mortality. In a retrospective analysis of 20,494 trauma patients admitted to a major Canadian hospital, the GCS was found to have excellent discrimination (area under Receiving Operator Curve = 0.833, CI = 95%). It was also found that eye-opening component was not as significant a predictor of mortality as the motor and verbal components.

Generally the GCS has been found to be a robust predictor of outcome with the motor component being the most significant predictor of mortality. A GCS of 8 or less was associated with severe injury and poorer outcome (Pal et al., 1989; Moore et al., 2006; Ross et al., 1998). The GCS was found to inversely correlate with the ISS where lower
GCS scores (8 or less) were a reliable indicator of severe neurological injury (Pal et al., 1989).

Various studies have been undertaken to determine the level of agreement and inter-rater reliability of the GCS between pre-hospital and emergency department personnel. Menegazzi, Davis, Sucov and Paris (1993) considered the reliability of the GCS when used by emergency physicians and paramedics. A prospective, non-randomised trial was conducted in the classroom setting with 19 emergency physicians and 41 paramedics. Three videotaped scenes were used to determine initial GCS. All participants were volunteers. All paramedics had at least 5 years of on-road experience. Inter-rater reliability was determined using the Kappa statistic and the reliability coefficient.

Menegazzi et al. (1993) found statistically significant reliability between emergency physicians and paramedics. The inter-rater reliability for emergency physicians and paramedics was $r = 0.66$ ($p < 0.001$) and $r = 0.63$ ($p < 0.001$) respectively. The results demonstrated a strong inter-rater reliability for both emergency physicians and paramedics. The greatest variability between emergency physicians and paramedics was found in the period following intermediate depression of consciousness. Paramedic GCS scores were consistently lower than that of emergency physicians by an average of one point. It was noted that all scenes were of adult patients and that inter-rater reliability had not been explored for paediatric or pre-verbal patients and results could not be extrapolated to this population. However, results suggested that strong inter-rater reliability was demonstrated in the severe category (Menegazzi et al., 1993).
Correlation between emergency department GCS (ED-GCS) and field GCS (EMS-GCS) was investigated by Bazarian, Eirich and Salhanick (2003). A significant linear relationship between ED-GCS and EMS-GCS was found using linear regression analysis for 60 patients with traumatic brain injury ($r = 0.45$, $p = 0.003$). Further investigation was undertaken exploring the difference between emergency department and pre-hospital systolic blood pressure, respiratory rate and oxygen saturation. No significant difference was found between emergency department and field scores in all comparisons. It was found that field GCS scores were, on average, two points lower than emergency department scores. Results suggest a robust linear relationship between ED and EMS GCS. However, the sample size was relatively small with only 60 individuals.

The reliability and accuracy of the GCS was examined in a study of experienced and inexperienced nurses (Rowley and Fielding, 1991). Reliability was established through per cent agreement and the rate of disagreement and accuracy was established by comparing the ratings of each individual rater with an expert rater. Four groups were compared. Group 1 consisted of 3 experienced nurses with 2 or more years of post-graduate experience and 1 year or more of neuroscience nursing. Group 2 included 7 newly graduated nurses. Groups 3 and 4 included 5 and 6 student nurses respectively. All groups received instruction on the GCS before participating in the study. The experienced nurses in group 1 displayed a high level of reliability and their agreement rates were 96.4%. The remaining 3 groups displayed observer agreements of 60–80%. Rowley and Fielding (1991) concluded that experienced and well-trained practitioners can use the GCS with extremely high levels of accuracy and reliability. Practitioners with limited
training and experience can still use the GCS with high levels of reliability. It should be noted, however, that the sample sizes in this study were small and the results may not be representative of the general population of nurses.

The Menegazzi et al. (1993), Bazarain et al. (2003) and Rowley and Fielding (1991) studies are all limited by small sample sizes and therefore limitations may exist when extrapolating findings to the larger population of in-hospital and pre-hospital raters. However, while the three studies have limitations, they all demonstrate similar findings in that good inter-rater reliability was found between pre-hospital and in-hospital raters, and between experienced and inexperienced users. The studies suggest the GCS, when used by pre-hospital personnel, is a reliable tool for determining depth of impaired consciousness.

More recently, Kerby, MacLennan, Burton, McGwin and Rue (2007) enrolled 3,052 patients over a 3-year period into retrospective cohort study. Cases were retrieved from a trauma registry. Pre-hospital GCS (Ph-GCS) and emergency department GCS (ED-GCS) were compared using chi-square (x²) for categorical variables, Student’s t-test for continuous variables and κ-coefficient to assess observer agreement. Analysis showed that there was high agreement between Ph-GCS and ED-GCS in the mild head injury category (97.9%) and moderate agreement in the severe category (63.3%). Lowest agreement was noted in the moderate head injury category (9.3%). Overall, results suggested moderate agreement between Ph-GCS and ED-GCS. Kerby et al. (2007) also considered length of time in the pre-hospital setting and found that agreement worsened
for patients with longer transport times. It was uncertain as to whether this was due to an improvement in the patient’s condition during transport or a result of inter-rater variability.

Davis et al. (2006c) considered the predictive value of the field GCS (fGCS) versus initial in-hospital arrival GCS (aGCS); and also field and arrival trauma score and injury severity score (TRISS) in moderate to severe traumatic brain injury. Inclusion criteria included major trauma victim with an abbreviated injury score (AIS) of three or greater, with moderate to severe brain injury. The sample was selected from the San Diego County Trauma Register for a 16-month period. A total of 12,882 patients were enlisted into the study. Mean values for fGCS and aGCS were compared using the Student’s t-test and the association between fGCS and aGCS was also calculated using linear regression analysis. Field and arrival values for GCS, systolic blood pressure and respiratory rate were used to calculate TRISS scores. TRISS survival prediction was compared with observed survival. Patients intubated in the pre-hospital setting were excluded from the study as arrival GCS could not be assigned.

Results demonstrated that fGCS and aGCS mean values were similar (fGCS 11.4 and aGCS 11.5, p = 0.336) and a strong correlation existed between fGCS and aGCS (r = 0.67, 95% CI 0.69, p < 0.001). Pre-hospital GCS calculations were shown to be valid and fGCS calculations also correlated with both aGCS and patient outcome. Pre-hospital GCS is therefore highly predictive of survival and for the need for neurosurgical intervention (Davis et al., 2006c).
Holdgate, Ching and Angonese (2006) studied the variability in agreement between physicians and nurses when measuring the GCS in the emergency department. The study was undertaken in a tertiary referral hospital in New South Wales, Australia. The study design was a prospective observational study using a convenience sample of 108 patients who were aged 18 years or above. Holdgate et al. (2006) found inter-rater agreement to be excellent for verbal scores and total GCS scores (weighted kappa > 0.75) and intermediate for motor and eye scores (weighted kappa 0.40 – 0.75). Inter-rater agreement was not found to vary substantially across the range of actual numeric GCS scores. It should be noted that the sample included medical as well as trauma patients and that trauma represented only 18% of the total, a factor which limits the generalising of the results to a homogenous trauma population.

The preponderance of evidence suggests there is good inter-rater reliability amongst cohorts of paramedics, nurses and doctors when assigning the GCS. The GCS is therefore considered a reliable tool and an appropriate scale to be used as an inclusion criterion. A pre-hospital GCS of 8 or less was considered to be an indicator of severe traumatic head injury and an inclusion criterion into the cohort for this study.

2.4.2 Injury Severity Score (ISS)

The Injury Severity Score (ISS) was developed in 1971 by Susan Baker. Prior to the development of the ISS, the Abbreviated Injury Scale (AIS) was used as a measure of injury severity. The AIS is a 6-point scale that classifies individual injuries; however, the AIS does not assess the effects of multiple injuries or trauma to multiple body regions.
The ISS was therefore developed to assess the effects of multiple injuries. The ISS is calculated by adding the square of the highest AIS score in the three most severely injured body regions. It is a 75-point scale which considers injuries in six body areas including head or neck, face, chest, abdominal or pelvic contents, extremities or pelvic girdle and external injuries (Association for the Advancement of Automotive Medicine, 1998a, 1998b).

Foreman et al. (2007) enrolled 410 patients with brain trauma into a prospective study of outcome at 12 months post-injury. Of the 410, outcome data was obtained for 270, a follow-up rate of 66%. All patients were 13 years of age or older. The aim of the study was to assess the usefulness of the AIS, ISS and GCS in predicting outcome following brain trauma. The measure of outcome used was the Extended Glasgow Outcome Scale (GOSE). AIS, ISS and GCS scores were collected at admission. Univariate and multivariate analysis was undertaken using Mann-Whitney U and Kruskal-Wallis one-way analysis of variance with \( x^2 \) for categorical data. Spearman’s correlation coefficients were used for univariate analysis and linear multiple regression was performed on statistically significant variables (p < 0.05).

The ISS was the best predictor of outcome when using the GOSE (rs = −0.341, p < 0.001). GCS and AIS were also found to be significant (rs = 0.227, p < 0.001 and rs = −0.222, p < 0.001 respectively). When considered in combination, GCS and ISS showed significant correlation with GOSE (r = 0.355, p < 0.001) at 12 months post-injury (Foreman et al., 2007).
2.4.3 The GCS and ISS in the Current Study

This study uses the GCS as a criterion for inclusion. With reference to the studies reviewed in the preceding section (Section 2.4 Measurement of Injury Severity in Severe Traumatic Brain Injury), a GCS of 8 or less has been demonstrated to be a reliable indicator of severe brain injury and the GCS has an established as having good inter-rater reliability. The Injury Severity Scale is assigned post-admission by accredited assessors and is a 75-point scale used to measure injury severity in trauma patients. Both the ISS and GCS will be used in regression analysis as independent measures in injury severity.

2.5 Outcome Measures in Severe Traumatic Head Injury

2.5.1 Glasgow Outcome Scale (GOS)

The Glasgow Outcome Scale (GOS) is a five-category scale used to estimate functional outcome following neurotrauma. The GOS was first described in 1975 by Jennett and Bond. The components of the GOS as described by Jennett and Bond (1975) and Hall, Cope and Rappaport (1985, p. 36) are contained in Table 5.
Table 5  Five Categories of the Glasgow Outcome Scale

<table>
<thead>
<tr>
<th>Score/Category</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Death</td>
<td>(a) As a direct result of brain trauma. (b) Patient regained consciousness, died thereafter from secondary complications or other causes.</td>
</tr>
<tr>
<td>2. Persistent Vegetative State</td>
<td>Patient remains unresponsive and speechless for an extended period of time, weeks to months. He may open his eyes and show sleep/wake cycles but show an absence of function in the cerebral cortex as judged behaviourally.</td>
</tr>
<tr>
<td>3. Severe Disability (Conscious but disabled)</td>
<td>Dependent for daily support by reason of mental or physical disability, usually a combination of both. Severe mental disability may occasionally justify this classification in a patient with little or no physical disability.</td>
</tr>
<tr>
<td>4. Moderate Disability (Disabled but independent)</td>
<td>Can travel by public transport and work in a sheltered environment and can therefore be independent insofar as daily life is concerned. The disabilities found include varying degrees of dysphasia, hemi-paresis, or ataxia, as well as intellectual and memory deficits and personality change. Independence is greater than simple ability to maintain self-care within the patient’s home.</td>
</tr>
<tr>
<td>5. Good Recovery</td>
<td>Resumption of normal life even though there may be minor neurological and pathological deficits. Return to work is considered an unrealistic index of recovery due to varying degrees of socioeconomic circumstances between survivors of neurotrauma.</td>
</tr>
</tbody>
</table>

The GOS has been used as a widely used scale for assessing outcome following severe head injury since its introduction in 1975 (Teasdale, Pettigrew, Wilson, Murray & Jennett 1998). It is recommended that the GOS is best assigned at intervals 1 month, 3 months, 6
months and 1 year post-discharge from hospital. A GOS score can be assigned at discharge; however, long term outcome may not be accurately reflected as the degree of resumption of normal life cannot be reliably determined at that stage (Teasdale et al., 1998). Wilson, Pettigrew and Teasdale (1998) suggest a structured interview using a standard protocol is the most appropriate means of assigning a GOS score. When a structured interview was used to assess 50 patients with head injuries, inter-rater reliability was found to be high (K = 0.89).

Teasdale et al. (1998) measured the inter-rater reliability between a research psychologist and a research nurse in assigning a GOS for 50 patients admitted to a regional neurosurgical unit. Agreement was found to be 92% and calculation using weighted kappa values was K = 0.89, indicating very good agreement. Inter-rater reliability was also found to be high when the GOS was used by the nurse without specific training in outcome assessment and a trained psychologist (Teasdale et al., 1998). While this study demonstrated high inter-rater reliability, it is limited by the participation of only two raters and the study should therefore be considered in conjunction with other studies.

Satz et al. (1998) studied the validity of the GOS in relation to neuropsychological, psychological and vocational correlates at 6 months post-injury in 100 moderate to severe brain injury survivors. To control for potentially confounding affects of general trauma an ‘other injury’ group was included into the study. These patients had suffered trauma to body areas other than the head. Correlates considered were neuropsychological (motor and psychomotor), memory, attention, psychosocial and function domains. Results were
adjusted for age and education. Separate analysis of covariance was undertaken for each domain. A p value of < 0.05 was considered significant. Validity of the GOS was found in the following domains, motor (grooved peg board test $F = 12.0$, $p = 0.001$), psychomotor (colour trails test $F = 8.0$, $p < 0.001$), word list memory ($F = 4.83$, $p < 0.004$) and the functional domain (employability rating scale $F = 16.5$, $p < 0.001$). No significant results were identified in the attention and psychosocial domains. It was concluded that results demonstrated an overall support for the predictive and concurrent validity of the GOS six months post-injury (Satz et al., 1998). Although results suggest good predictive validity of the GOS, caution should be employed in generalising these results due to the relatively small sample size, moderate effect size of the results and the non-reporting of eta-squared statistics.

Wilson, Pettigrew and Teasdale (2000) studied the validity of the GOS in relation to the Extended Glasgow Outcome Scale (GOSE). The GOSE is an 8-point scale in which the last three categories of the GOS (severe disability, moderate disability and good recovery) are divided into upper and lower bands. The GOS and GOSE were used to assess 135 adult patients admitted to a regional neurosurgical unit at six months post-injury. Spearman correlations between the GOS and GOSE demonstrated substantial correlation in 39 of 40 demographic, clinical and outcome variables with correlation coefficients ranging from $-0.21$ to $-0.71$ ($p < 0.05$ and $p < 0.01$). Age at injury failed to reveal a significant correlation ($\text{GOS } r = -0.08$; $\text{GOSE } r = -0.10$). The results demonstrated that the GOS shows consistent correlation with the GOSE across a wide range of variables.
However it is highly recommended that the GOSE be used in conjunction with a structured interview at 3, 6 and 12 months post-injury. The GOSE is therefore reliant on a structured patient interview in order to assign an outcome category. Advantages of the GOS over the GOSE are its less complicated structure and its wide recognition and acceptance amongst researchers (Wilson, Pettigrew & Teasdale, 1998).

Although each study exhibited limitations, collectively the studies of Teasdale et al., (1998), Satz et al., (1998) and Wilson et al., (2000) support the predicative validity of the GOS in relation to other outcome measures and domains and indicate good inter-rater reliability.

2.5.2 Disability Rating Scale (DRS)

The DRS is a 30-point scale that ranges from 30 (death) to 0 (recovery without gross impairment) and consists of 8 items which are divided into 4 categories. The DRS was developed to provide more specific information about the level of on-going disability of an individual patient over time. Quantitative evaluation of level of disability, awareness and functioning of the brain-injured patient could be gained through the rehabilitation process from coma and to their return to the community (Rappaport, Hall, Hopkins, Belleza & Cope, 1982). The DRS is more specific than the GOS due to the larger range of categories: a one-point change on the DRS is reflective of a distinct clinical change in the patient’s status (Hall, Cope & Rappaport, 1985).
The parameters of the DRS as described by Hall et al. (1985, p. 35) are contained within Table 6.

**Table 6  Components of the Disability Rating Scale (DRS)**

<table>
<thead>
<tr>
<th>Arousability, Awareness &amp; Responsivity</th>
<th>Eye Opening</th>
<th>Verbalisation Communication Ability</th>
<th>Motor Response</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 Spontaneous</td>
<td>0 Orientated</td>
<td>0 Obeying</td>
<td>0 Complete independent</td>
</tr>
<tr>
<td></td>
<td>1 To Speech</td>
<td>1 Confused</td>
<td>1 Localising</td>
<td>1 Independent (special environment)</td>
</tr>
<tr>
<td></td>
<td>2 To Pain</td>
<td>2 Inappropriate</td>
<td>2 Withdrawing</td>
<td>2 Mildly dependent (non-resident helper)</td>
</tr>
<tr>
<td></td>
<td>3 None</td>
<td>3 Incomprehensible</td>
<td>3 Flexing</td>
<td>3 Moderately dependent (person in home)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>4 Extending</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>5 None</td>
<td></td>
</tr>
<tr>
<td>Cognitive ability for self-care activities (does Pt known how and when?)</td>
<td>Feeding</td>
<td>0 Complete</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 Partial</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 Minimal</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 None</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toileting</td>
<td>0 Complete</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 Partial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 Minimal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grooming</td>
<td>0 Complete</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 Partial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 Minimal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychosocial adaptability</td>
<td>Employability</td>
<td>0 Not restricted</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 Selected jobs - competitive</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 Sheltered workshop – non-competitive</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 Not employable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total DR Score</td>
<td>4 Markedly dependent (all activities at all times)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5 Totally dependent (24 hour nursing care)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Hall et al., 1985, p. 35)
A total DRS is calculated by adding the score attained for each of the eight items. DRS scores and outcomes categories or levels of disability are included in Table 7.

Table 7  Disability Rating Scale (DRS) Outcome Categories and Level of Disability

<table>
<thead>
<tr>
<th>Total DRS Score</th>
<th>Level of Disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>Death</td>
</tr>
<tr>
<td>25-29</td>
<td>Extreme Vegetative State</td>
</tr>
<tr>
<td>22-24</td>
<td>Vegetative State</td>
</tr>
<tr>
<td>17-21</td>
<td>Extremely Severe</td>
</tr>
<tr>
<td>12-16</td>
<td>Severe</td>
</tr>
<tr>
<td>7-11</td>
<td>Moderately Severe</td>
</tr>
<tr>
<td>4-6</td>
<td>Moderate</td>
</tr>
<tr>
<td>2-3</td>
<td>Partial</td>
</tr>
<tr>
<td>1</td>
<td>Mild</td>
</tr>
</tbody>
</table>

Hall et al. (1985) considered the comparative usefulness of the GOS and the DRS following traumatic brain injury. Inter-rater correlations were compared for 88 patients with head injuries at 2, 4, 6, 12 and 24 months post-injury for three trained raters. Each rater independently assessed the 88 patients. The Pearson correlation of the DRS and
GOS at admission was found to be $r = 0.50$ and at discharge was $r = 0.67$ ($p < 0.01$). The robust correlation coefficient suggests a significant correlation existed between DRS and GOS scores and that DRS is an appropriate and sensitive scale for monitoring persons with severe traumatic brain injury through the recovery phase.

Gouvier, Blanton, LaPorte and Nepomuceno (1987) also considered the reliability and validity of the DRS in monitoring recovery from severe brain trauma. The DRS and the Levels of Cognitive Functioning Scale (LCFS) were used to evaluate 40 head-injured inpatients. Each patient was independently assessed by the 3 different raters each week for the duration of their in-hospital stay. Spearman’s rho correlation was used to analyse inter-rater reliability, test-retest reliability, admission concurrent validity, discharge concurrent validity and predictive validity of the DRS and LCFS. Inter-rater reliability between the three raters when using the DRS were identical ($r = 0.98$) whereas some variance was noted between the 3 raters when using the LCFS ($r = 0.94$, $r = 0.88$, $r = 0.87$; mean correlation = 0.89). The DRS also surpassed the LCFS on test-retest reliability (DRS $r = 0.95$, LCFS $r = 0.82$). No difference was found between the DRS and LCFS in admission concurrent validity ($r = 0.92$); however, some difference was revealed between the DRS and LCFS when using the GOS and GOSE as measures of outcome (DRS GOS $r = 0.80$, DRS GOSE $r = 0.85$, LCFS GOS 0.76, LCFS GOSE $r = 0.79$). The predictive validity of the DRS with the GOS ($r = 0.62$) and GOSE ($r = 0.73$) was superior to the LCFS and GOS ($r = 0.59$) and the GOSE ($r = 0.68$). Very strong correlation coefficients indicate the DRS to be a reliable and valid tool for monitoring recovery in patients with head injuries.
Similarly, Hammond et al. (2001) found the DRS to be a superior predictor of functional outcome than the Functional Independence Measure (FIM) in their prospective study of 1,160 subjects with traumatic brain injury. The study was undertaken to assess the sensitivity of the DRS and FIM in predicting outcome at 1, 2 and 5 years post-injury. It was found that the DRS was more sensitive to changes during a shorter time period than the FIM and was more appropriate at detecting long-term deficits. Signed rank tests were used to compare patient functioning from year to year. In the years 1 and 3 the DRS only showed significant differences in rehabilitation status (p < 0.001) where as in years 1 and 5 both the DRS and FIM were sensitive to changes in patient functioning (DRS p < 0.001, FIM p = 0.001). Results suggest the DRS is sensitive to changes in patient functioning in both short and long term phases of the rehabilitation process in individuals with traumatic brain injury.

2.5.3 The Use of the GOS and DRS in the Current Study

The GOS was selected as the primary measure of outcome in this study. The GOS is a 5-point scale used to estimate functional outcome following neurotrauma. As demonstrated through the literature reviewed in this section (Section 2.5 Outcome Measures in Severe Traumatic Brain Injury) the GOS has been shown to be a valid and reliable predictor of functional outcome following trauma and has excellent inter-rater reliability. The GOS was selected in preference to the 8-point GOSE as it is recommended that the GOSE be used in conjunction with structured interviews at 6 and 12 months post-injury. The GOS has a less complicated structure than the GOSE and is widely accepted amongst researchers. The DRS is used as an additional measure of outcome in this study.
2.6 Pre-Hospital Management of Persons with Severe Traumatic Head Injury

2.6.1 Maintenance of a Patent Airway and Oxygen Saturation

Maintaining a patent airway and oxygen saturations (SpO2 > 95%) is critical in the management of persons with severe traumatic brain injury. Oxygen saturation of greater than 90% is imperative in reducing the risk of secondary insult (Brain Trauma Foundation, 2000a, 2007). The treating of airway compromise and ventilatory insufficiency should comprise the first steps in the pre-hospital management of the trauma patient. Obstructed airway and subsequent hypoxaemia may result in secondary insult and poor outcome (Brain Trauma Foundation, 2000a).

In an analysis of the efficacy of current trauma care in Victoria, Danne et al. (1997) found that patients with severe head injuries require augmented management to prevent secondary brain injury in both the in-hospital and pre-hospital settings. Further, it was suggested that definitive airway control in particular needs to be improved in the pre-hospital setting; however, the types of improvements were not specified. In an epidemiological study of outcomes following traumatic brain injury in Australia and New Zealand (n = 635) in 2000 to 2001 Myburgh et al. (2008) found pre-hospital airway obstruction to be documented in 19.6% of patients (n = 99 of 504) and oxygen desaturation to be documented in 8.5% of patients (n = 36 of 422).

Paramedics use suction, patient posturing and airway adjuncts to aid in airway maintenance (Ambulance Service of New South Wales, 1997, 2007). The airway adjuncts
used by the Ambulance Service of NSW include oropharyngeal airway, nasopharyngeal airway and endotracheal intubation. Oropharyngeal and nasopharyngeal airways may be used by all qualified paramedics; however, endotracheal intubation is only authorised for level 5, intensive care paramedics. Rapid sequence induction (RSI) in conjunction with endotracheal intubation is not an approved intervention for NSW paramedics (Ambulance Service of New South Wales, 1997, 2007).

The Ambulance Service of NSW (1997, 2007) specifies that if spinal injury is suspected or there is evidence of significant mechanism of trauma, then a cervical collar should be applied and spinal precautions implemented as part of the patient management routine. Therefore, the vast majority of trauma patients have a cervical collar applied and are postured supine on the stretcher.

Birnbaumer and Pollack (2002) advocate that maintaining a patent airway is essential for good outcome and failure to achieve a patent airway can lead to hypoxic neurological injury within minutes. Difficult airways occur in 1 in 10,000 patients and 1% of patients will have a failed airway. Characteristics which can give rise to a difficult airway can include previous head or neck surgery, facial hair, dental abnormalities, a narrow face, a high and arched palate, a short or thick neck and obesity. Regal, Stalp, Lehmann and Seekamp (1997) studied 1,223 multi-trauma patients with Injury Severity Scores greater than 20 and found that early pre-hospital intubation was associated with a lower rate of multiple organ failure in multi-trauma patients with Injury Severity Scores between 20 and 39 (Student’s t-test, p < 0.05); however, the results were not significant for those
with Injury Severity Scores greater than 40. The extent of influence of other variables such as pre-hospital fluid resuscitation, rescue time and the effect of different concomitant injuries on the development of multiple organ failure is unclear due to the lack of statistical control of potentially confounding variables. Therefore the results of this study should be considered in conjunction with other studies and care should be exercised when generalising these results to the general population of multi-trauma patients.

Chi et al. (2006) undertook a prospective, multi-centre cohort study to determine the incidence of hypoxia and hypotension in the pre-hospital setting of patients with traumatic brain injury and to examine the association between pre-hospital hypoxia or hypotension and mortality and functional outcome at discharge from hospital. The sample included 150 patients who were transported via helicopter to a 4 different level 1 trauma centres in the United States of America from February 1998 to January 2000. The mean flight time was 45 minutes. Analysis revealed that 37 patients (24.7%) had hypoxic episodes only, 14 (9.3%) had hypotensive episodes only and 6 (4.0%) had both hypoxia and hypotension. A total of 57 patients (38%) had episodes of hypoxia and/or hypotension during helicopter transport. The mean duration of the hypoxic episodes was 17 (± 16) minutes. Lung injury occurred in 56 patients and of these, 38% experienced hypoxia with an average duration of 18 minutes. Of those patients without lung injury (n = 94), hypoxia occurred in 23%. The mean duration of hypoxia in those patients was 15 minutes (Chi et al., 2006).
No significant difference in the episodes of hypoxia was found between intubated and non-intubated patients. Among the 99 patients who were intubated, 28% (n = 30) experienced episodes of hypoxia, compared with 30% (n = 15) of the 51 non-intubated patients. Mortality rates for patients experiencing hypoxia alone was 37%, hypotension alone 8% and both hypoxia and hypotension was 33%. Overall mortality was 23.3% and mortality odds ratio for those patients who experienced an episode of hypoxia was 2.66 (p = 0.043) (Chi et al., 2006). The outcome measure used for the Chi et al. study was mortality; therefore, functional outcome (GOS) post-hypoxia was not reported. While the prospective methodology and good sample size provide sound evidence of the deleterious effects of pre-hospital hypoxaemia, the study is limited by the single outcome measurement of mortality only.

Stocchetti, Furlan and Volta (1996) studied the effects of pre-hospital hypoxaemia and hypotension in 50 patients with head injuries. Data analysis found that 50% of patients experienced hypoxaemia (SpO2 < 90%) and 24% experienced hypotension (systolic blood pressure < 90 mmHg). Results established that both hypoxaemia and hypotension adversely affect outcome, however the extent to which each contributed to outcome was not determined. Cooke, McNicholl and Byrnes (1995) in a prospective audit of 131 patients with severe head injuries found 27% were hypoxic on arrival at the emergency department. Jones et al. (1994) in their prospective analysis of 124 patients with head injuries found that episodes of hypoxaemia were significantly associated with poor outcome (p = 0.024).
The current study investigates this issue further by analysing the effect of airway management and oxygen saturations on functional outcome of patients with severe traumatic brain injury.

### 2.6.2 Ventilation

Respiratory dysfunction is common following severe brain trauma. Atkinson, Anderson and Murray (1998) studied the effects of mild, moderate and severe brain trauma in a sample of 3 groups of 6 rats. An induced mild, moderate or severe brain injury was delivered to each group, using fluid percussion. Mild brain injury was achieved using a force of 1.5 atmospheres of pressure (atm), while moderate and severe injuries were attained using 2.5 atm and 3.5 atm respectively. Continuous monitoring of electroencephalograph (EEG) activity, MABP, ICP and respiratory tidal volumes were undertaken for a 45-minute period post-injury. The observed degree of respiratory dysfunction and apnoea following brain trauma was directly proportional to the magnitude of energy delivered to the brain: the higher the level of energy, the higher the rate of dysfunctional or absent respiratory response. This was thought to be a result of failure or disorganised function of the medullary respiration centre.

Within the mild injury group, all animals experienced brief apneustic pauses over one minute. Pre-injury respiratory rate and tidal volume resumed following one minute. The EEG waveform became dysrhythmic, MABP increased slightly and arterial blood gases and ICP remained stable and relatively unchanged. All animals survived the 45 minutes of the experiment. The moderate injury group experienced severe disruption to their pre-
injury ventilation responses. Four of the six animals did not regain organised respiratory effort within 5 minutes of the injury. All four animals became progressively hypoxic and underwent cardiovascular collapse. All died within 5 to 8 minutes. One animal regained an organised respiratory effort, however it was not sustained. Tidal volume diminished and cardiovascular collapse was experienced. Only two animals regained organised respiratory control to survive the 45 minutes of the experiment. All animals displayed severe EEG changes and tachycardia. Significantly elevated MABP and ICP were demonstrated in the dysfunctional respiratory group and increases in partial pressure of carbon dioxide (pCO2) were noted 5 minutes post-injury. All animals died in less than five minutes within the severe injury group. All failed to regain any respiratory effort and displayed severe EEG changes and marked elevation in MABP and ICP (Atkinson et al., 1998).

Results of the study suggested that brain-stem mediated respiratory dysfunction following brain trauma should be managed with early mechanical ventilation to prevent cardiovascular collapse. Many victims of brain trauma could be salvaged if early mechanical ventilation was instituted in the pre-hospital setting. Therefore response time is a critical factor in determining outcome following severe traumatic brain injury (Atkinson et al., 1998). However it should be noted that the Atkinson et al. study used an animal model and this may limit extrapolation of the findings to a human population. After review of existing scientific studies, the Brain Trauma Foundation (2000a) recommended that oxygen saturation levels in patients with severe brain trauma be maintained at a level greater than 90%. Hypoxaemia in severe traumatic brain injury
patients may lead to secondary brain injury and worsened outcome. While the Brain Trauma Foundation (2000a) recommends an appropriate level of oxygenation, optimal ventilation rates and tidal volume during intermittent positive pressure ventilation are not specified. Ambulance Service of New South Wales ambulance vehicles are not equipped with mechanical ventilators and therefore mechanical ventilation is not an intervention undertaken by paramedics. Paramedics manually ventilate patients who require ventilatory support (Ambulance Service of New South Wales, 1997). No study was found that investigated the affect of manual ventilation by paramedics on patients with severe traumatic brain injury.

Warner, Cuschieri, Copass, Jurkovich and Bulger (2007) studied the effect of pre-hospital ventilation on outcome after severe traumatic brain injury; however, the study did not isolate paramedic ventilation from emergency department ventilation, nor was it able to consider the respiratory status of the patient prior to the arrival of the paramedic crew. Despite the limitations of the study, some valuable results were noted. Carbon dioxide levels (pCO2) were measured in 423 trauma patients who were intubated in the pre-hospital setting. The pCO2 levels were determined on arrival at hospital via blood gas analysis. Target pCO2 was considered to be between 30 – 35 mmHg, mild hypercapnoea was 36– 45 mmHg, severe hypercapnoea was > 45 mmHg and hypocapnoea was considered to be < 30 mmHg. Chi-square analysis was used for categorical variables and one-way analysis of variance for continuous variables. The level of significance was set at p = 0.05 (Warner et al., 2007). It was found that 80 patients (16.3%) were hypocapnoeic: 155 (31.5%) were within the target ventilation group, 188 (38.2%) were
mildly hypercapnoeic. The severely hypercapnoeic group \( n = 69, 14.0\% \) were excluded as their injuries were considered to disproportionately affect alveolar ventilation due to chest trauma and therefore confound results (Warner et al., 2007).

Mortality rates were lower in the target group and patients with pCO2 between 30 – 45 mmHg were more likely to survive than those with pCO2 < 30 mmHg or > 45 mmHg \( (p < 0.05) \). It was also found that women were more likely to be hyperventilated and become hypocapnoeic than men \( (p < 0.05) \). This was thought to be a result of paramedics not adjusting rate and tidal volume to the smaller weights of the female sample. Further, mortality rate was high for isolated TBI when associated with hypocapnoea. However, it could not be determined if some of the hypocapnoeic group were not intentionally hyperventilated if paramedics thought the patient was displaying signs of raised intracranial pressure and impending brain-stem herniation (Warner et al., 2007).

Davis et al. (2006b) also found inadvertent hyperventilation and/or hypoventilation to be a deleterious factor in pre-hospital management of patients with severe brain injury. The increased incidence of hyperventilation was associated with endotracheal intubation. The outcomes of 890 intubated patients were compared with those of 2,914 non-intubated patients in a retrospective, registry-based analysis. Logistic regression was used to calculate the odds ratio of survival for increments in arrival pCO2 at a level 1 facility. Results showed an increase in survival for intubated patients with a pCO2 range of 30–49 mmHg. Those patients had an improved survival rate \( \text{odds ratio } 1.50 [1.15, 1.96], \) CI
95%, p < 0.05) and a higher incidence of good outcomes (odds ratio 1.34 [1.01, 1.78], CI 95%, p < 0.01) (Davis et al., 2006b).

Moreover, both hyperventilation and hypoventilation (pCO2 < 30 mmHg or > 49 mmHg) were associated with worse outcomes in intubated patients, but not in non-intubated patients. Davis et al. (2006b) concluded that endotracheal intubation in the pre-hospital setting was associated with increased incidence of hypercapnoea and hypocapnoea. The value of monitoring ETCO2 was advocated by Bernard (2006), who suggested that waveform capnography is essential to confirm tracheal placement of the endotracheal tube and to prevent inadvertent hyperventilation in patients with severe head injury.

Dumont, Visioni, Rughani, Tranmer and Crookes (2010) also found hypocapnoea to be associated with significant mortality. A retrospective review was undertaken to determine the effect of pre-hospital hyperventilation on in-hospital mortality following severe TBI (n = 65). Results demonstrated hypocapnoea to have an in-hospital mortality rate of 77%, hypercapnoea to have an in-hospital mortality rate of 61%, whereas patients who were normocapnoeic demonstrated an in-hospital mortality of 15%.

The integration of end-tidal carbon dioxide (ETCO2) monitoring into the pre-hospital environment has been advocated since the early 1990s. White and Asplin (1994) prospectively studied the pre-hospital ETCO2 of four patients during cardiopulmonary resuscitation. The cohort included one female and three male patients who were all over the age of 60 years. Results showed that ETCO2 monitoring was accurate in detecting
changes in pulmonary blood flow and that changes were identified rapidly. White and Asplin (1994) considered capnography in the pre-hospital setting was feasible and that clinically important events can be tracked using ETCO2 monitoring. It should be noted that ETCO2 monitoring is not used by paramedics in NSW and therefore changes in ETCO2 cannot be determined by ambulance paramedics (Ambulance Service of New South Wales, 1997).

Several studies have demonstrated that hypoxaemia, hypercapnoea and hypocapnoea are associated with poor outcome in severe TBI (Atkinson et al., 1998; Warner et al., 2007; Davis et al. 2006b & White et al., 1994). However, the ability of paramedics to manually ventilate trauma patients to maintain consistent oxygen and carbon dioxide levels within accepted parameters remains unclear. The current study investigates this issue further by analysing the effect of pre-hospital intermittent positive pressure ventilation on oxygen saturation on arrival at the emergency department and on functional outcome of patients with severe traumatic head injury. Further analysis of emergency department end-tidal carbon dioxide levels and functional outcome of persons with severe traumatic head injury will also be undertaken.

2.6.3 Hyperventilation

protocol for the management of head injury states that hypoventilation can aggravate brain injury by causing carbon dioxide retention. If hypoventilation is present, the patient should be hyperventilated. The protocol does not provide an indication of the length of time that the patient should be hyperventilated nor does it specify desired arterial CO2 during manual ventilation, and nor do paramedics have end-tidal carbon dioxide monitoring equipment (Ambulance Service of New South Wales, 1997, 2007)

Studies by Obrist, Langfitt, Jaggi, Cruz and Gennarelli (1984) and Muizelaar et al. (1991) have demonstrated the deleterious effects of aggressive hyperventilation in patients with brain injuries. Arterial carbon dioxide (PaCO2) should be maintained at 35 mmHg. Arterial carbon dioxide levels less than 30 mmHg reduces cerebral blood flow (CBF) without consistently reducing intracranial pressure (ICP) and may also lead to a loss of autoregulation (Brain Trauma Foundation, 2000a).

Aggressive and prolonged hyperventilation provides a significant risk of inducing cerebral ischaemia leading to cellular changes and secondary brain injury. Hyperventilation causes cerebral vasoconstriction and a decrease in CBF. Cerebral blood flow following traumatic brain injury is reduced to less than half of that of normal individuals for the first day post-injury. Hyperventilation augments the reduction in CBF by causing cerebral vasoconstriction (Brain Trauma Foundation, 2000a).

A cohort study was undertaken by Obrist et al. (1984) to measure CBF in 75 patients with brain injuries. Hyperaemia was noted in 41 patients (55%) with a mean duration of three
days. Consistent sub-normal CBF was found in 34 patients (45%). Obrist et al. discovered that hyperventilation had a more significant and direct effect on reducing CBF than it did on reducing ICP on 29 of 31 patients with severe traumatic brain injury. Muizelaar et al. (1991) in a prospective randomised controlled study of 77 patients with severe traumatic brain injury found that patients with an initial Glasgow Coma Score (GCS) of 4 or 5 had improved outcomes at 3 to 6 months post-injury when hyperventilation (PaCO₂ 20-30 mmHg) was not used (p < 0.05, correlation coefficient not reported). Patients who received periods of hyperventilation demonstrated worse outcomes in comparison.

Ambulance paramedics manually ventilate hypoventilating patients using a bag-valve-mask device. Mechanical ventilation and capnography are not routinely utilised in the pre-hospital setting in New South Wales (Ambulance Service of New South Wales, 1997, 2007). Ventilation in the pre-hospital environment can be affected by the airway management experience of the paramedic, distraction from the task of airway maintenance, the paramedic attempting to multi-task whilst ventilating the patient and movement of the patient during transfer to the stretcher and the vehicle. Aggressive and uncontrolled ventilation may occur during manual ventilation in the pre-hospital setting and the paramedic may not be aware of the presence of hypocapnoea due to the absence of CO₂ monitoring through capnography. Aggressive and prolonged hyperventilation in the pre-hospital setting may contribute to poor neurological outcomes of persons with severe traumatic brain injury (Muizelaar et al., 1991; Brain Trauma Foundation, 2000a).
The response to hyperventilation in a group of healthy individuals was studied by Raichle, Posner and Plum (1970). When partial pressure of carbon dioxide (PaCO2) was decreased by 15–20 mmHg, CBF was decreased by 40% within 30 minutes. CBF returned to 90% of baseline 4 hours later. When original PaCO2 was restored, there was a 31% increase in CBF. Fortune et al. (1995) found that a decrease in PaCO2 to 26 mmHg resulted in a 7.2% reduction in cerebral blood volume and moreover, a decrease of 30.7% in CBF. Results demonstrated that hyperventilation resulted in a change in CBF was far greater than the change in cerebral blood volume.

Hyperventilation was conventionally used to reduce or control ICP. The study by Raichle et al. (1970) highlighted that CBF is not directly related to ICP and yet CBF is the parameter directly affected by hyperventilation. This study revealed that CBF is not necessarily related to cerebral blood volume, but cerebral blood volume is directly related to ICP. Therefore hyperventilation will usually cause a significant reduction in CBF and thereby potentially limiting the flow of blood to ischaemic areas of the brain.

Crockard, Coppel and Morrow (1973) found an inverse relationship between hypocapnoea and ICP in some patients. PaCO2 between 25-30 mmHg resulted in an increase in ICP in four of 14 patients with severe traumatic brain injury. Conversely, Obrist et al. (1984) found that hyperventilation did reduce ICP in 15 of 31 patients, but there was also a reduction in CBF in 29 of the 31 patients.
A prospective, randomised clinical study was undertaken by Muizelaar et al. (1991) where 77 patients with severe traumatic brain injury were treated according to two protocols. One group was treated with chronic prophylactic hyperventilation for five days following injury (PaCO2 25 +/- 2 mmHg) and the other group was maintained in a normocapnoic state (PaCO2 of 35 +/- 2 mmHg). Patients were reviewed 3 and 6 months post-injury and it was found that patients who received chronic prophylactic hyperventilation had a significantly worse outcome than those patients who were maintained in a normocapnoic state. Significant results could not be established at 12 months post-injury as there were too few patients available for assessment.

Diringer et al. (2002) studied the effects of transient severe hyperventilation on four patients with severe traumatic brain injury one to five days post-injury using positron emission tomography (PET). Severe hyperventilation was defined as PaCO2 of 25 +/- 2 mmHg. Intracranial pressure was elevated to 20 – 30 mmHg. All patients had been stabilised through interventions in the emergency department and/or surgery. No patients had received early hyperventilation in the pre-hospital, emergency department or surgical environments.

It was found that while cerebral blood flow (CBF) fell, oxygen extraction fraction (OEF) for cerebral tissues rose and cerebral metabolic rate of oxygen (CMRO2) was unchanged. Moreover, in areas where CBF was less than 20 ml/100 g/min and 10 ml/100 g/min, CMRO2 did not change. In the uninjured brain, energy failure is reached when CMRO2 falls below 18 to 20 ml/100 g/min. It should be noted that in the injured brain, the
mitochondria are unable to efficiently use oxygen and move from aerobic to anaerobic metabolism, and oxygen demand and CMRO2 is low. Therefore, no evidence was found to indicate that hyperventilation to 25 mmHg during periods of elevated ICP produced global or regional ischaemia, even in areas where CBF fell to below 10 ml/100 g/min (Diringer et al., 2002).

Diringer et al. (2002) concluded there was no critical limitation of oxygen availability to the tissues in the injured brain and that the brain may tolerate transient hyperventilation. The authors also indicated that while transient severe hyperventilation may be safe, it does not necessarily imply that it is beneficial. The CBF threshold for energy failure following traumatic brain injury is therefore unknown.

Limitations to the Diringer et al. study include the small number of patients with traumatic brain injury enrolled into the study \( (n = 4) \) and the fact that not all patients had large contusions or mass lesions. At the time of the study all patients were normotensive and oxygen saturations were within normal limits, none were hypotensive or hypoxic. Severe hyperventilation was not administered in the pre-hospital setting or within the emergency department. Therefore the capacity to generalise the findings may be limited.

Oertel et al. (2002) also considered the efficacy of hyperventilation in controlling ICP after brain injury. The study included 33 patients with acute head injury with post-resuscitation GCS scores of 8 or less. Patients were subject to transient hyperventilation during the post-resuscitative period. All patients were haemodynamically stable and had undergone emergency craniotomy if required. It was found that hyperventilation did
reduce ICP in 96.5% of tests of the 33 patients and a decrease of 20% occurred in 77.2% of tests. It was also noted that saturation of jugular venous oxygen (SjO2) also decreased, but not to levels potentially dangerous to patient outcome (73 +/- % to 67 +/- 8%). The study concluded that transient hyperventilation may be used to control ICP in patients with head injuries, but should be performed in conjunction with SjO2 monitoring in order to avoid oxygen desaturation and potential secondary brain injury.

Nolan, Saxena, Burgess, Simmel and Braude (2004) studied the mechanism of post-hyperventilation hypoxaemia in healthy male volunteers (n = 7). The males were aged 29.1 ± 1.4 years and underwent voluntary hyperventilation for two 10-minute periods to reach end-tidal carbon dioxide values of 20 mmHg (severe hyperventilation), or 30 mmHg (moderate hyperventilation). Arterial oxygen saturation and arterial blood gas values were measured prior to hyperventilation and post-hyperventilation. It was found that maximal hypoxaemia occurred at approximately five minutes post-hyperventilation. Baseline arterial oxygen saturations were 97 ± 3 mmHg. Arterial oxygen saturations fell to 64 ± 7 mmHg following severe hyperventilation and 72 ± 6 mmHg following moderate hyperventilation. The study concluded that hypoxaemia following hyperventilation was largely due to an alteration in pulmonary blood flow distribution causing a fall in ventilation/perfusion (V/Q) ratio.

Davis et al. (2004) during the San Diego Paramedic Rapid Sequence Induction (RSI) Trial, found episodes of hyperventilation and hypocapnoea were common during pre-hospital rapid sequence induction and intubation of patients with head injury. The San
Diego Paramedic RSI Trial was undertaken between November 1998 and April 2002 to consider the efficacy of pre-hospital rapid sequence induction and intubation and outcome in patients with severe head injury. A sample of 59 hypocapnoeic patients from the trial was matched with 177 control patients. Logistic regression showed a weak but significant correlation between hypocapnoea and increased mortality ($r = -0.13$, $p < 0.001$). For the trial patients, overall mortality was 41% versus 22% for the control group (odds ratio, 2.51; 95% CI; $p = 0.004$). It should be noted that the sample size was small and the possibility of hidden bias between the sample and the control group may have confounded results.

Davis et al. (2005a) further evaluated the incidence of inadvertent pre-hospital hyperventilation (ETCO2 < 30 mmHg) and severe hyperventilation (ETCO2 < 25 mmHg) in 76 San Diego Paramedic RSI Trial patients. ETCO2 values of less than 30 mmHg and less than 25 mmHg were documented in 79% and 59% of the trial patients respectively. Results suggest that inadvertent hyperventilation was common following paramedic RSI and intubation, even though ETCO2 monitoring was used and target ETCO2 parameters of 30 – 35 mmHg were recommended during the trial. Reasons for high percentage of inadvertent hyperventilation were not explored.

Manley et al. (2000) studied the effects of hyperventilation (respiratory rate = 30) and hypoventilation (respiratory rate = 4) in swine haemorrhaged to 50% of estimated blood volume. Results indicated that tissue oxygenation decreased from $39.8 \pm 4.6$ mmHg to $11.4 \pm 2.2$ mmHg following haemorrhage. Hyperventilation resulted in a 56% mean
decrease in brain tissue oxygenation, whereas hypoventilation resulted in a 166% increase in brain tissue oxygenation \( (x^2, p = 0.001) \). Manley et al. concluded hyperventilation exacerbated brain injury by increasing brain tissue hypoxia through cerebral vascular constriction. Conversely, hypoventilation alleviated brain tissue hypoxia. The finding suggests that hyperventilation has a deleterious effect on brain oxygenation in patients with haemorrhagic shock and head trauma.

The preponderance of evidence in the studies reviewed found hyperventilation to be deleterious to outcome. Hyperventilation resulted in disruption of CBF and did not result in significant decreases in ICP. Hyperventilation was not found to be beneficial in individuals with severe brain trauma (Obrist et al., 1984; Muizelaar et al., 1991; Raichle et al., 1970; Diringer et al., 2002; Oertel et al. 2002; Nolan et al., 2004; Davis et al., 2005a & Manley et al., 2001). The analysis of intentional or inadvertent hyperventilation by paramedics is not possible in the current study as data relating to these parameters is not contained within ambulance service documentation. It should be noted that intentional or inadvertent hyperventilation may occur in the pre-hospital phase of management and this could significantly impact on patient outcome following severe brain trauma. However, at this point, there is no means of assessing this parameter.

The current study will provide limited analysis of end-tidal carbon dioxide levels within the in-hospital setting and consider the effect of hypopcapnoea on functional outcome of patients with severe traumatic brain injury. However it should be noted that is important parameter is not measured in the pre-hospital setting and results may not accurately
reflect the efficacy of paramedic management of persons with severe traumatic head injury. However the measurement of end-tidal carbon dioxide may be a critical intervention appropriate for inclusion into the pre-hospital phase of management to inhibit the onset of hypocapnoea through prolonged pre-hospital hyperventilation.

2.6.4 Airway Adjuncts – Oropharyngeal Airway, Nasopharyngeal Airway and Endotracheal Intubation

Three airway adjuncts are used by the Ambulance Service of NSW: oropharyngeal airway, nasopharyngeal airway and endotracheal intubation. Only the intensive care paramedic (level 5) is authorised in the use of endotracheal intubation. All levels of clinical skill, levels 1 through 5, are authorised to use oropharyngeal and nasopharyngeal airways (Ambulance Service of New South Wales, 1997, 2007). A literature search located several studies relating to the efficacy of pre-hospital intubation and patient outcome, however no studies relating to the use of oropharyngeal or nasopharyngeal airway adjuncts and patient outcome following severe head or brain injury were found.

Winchell and Hoyt (1997) reviewed 1,092 cases of severe head trauma in a retrospective case-control study. Severe head injury was classified as an abbreviated injury score of 4 or greater. The inclusion criteria were met by 1,092 patients with head injuries, with 671 patients having multi-trauma with severe head injury, and 351 with isolated severe head injury. Of the 1,092 head injured patients, 565 received endotracheal intubation in the field and 527 were not intubated in the field. The paramedic response included both ground crews and aeromedical crews. No comparison was made between patients
transported via ground versus those transported by aeromedical systems. Analysis was controlled for age, head or neck AIS, ISS and scene GCS.

Results demonstrated that field intubation was associated with significant decreases in mortality from 36% to 26% in the full study group, from 57% to 36% in severe head injury plus multi-trauma group and from 50% to 23% in the isolated severe head injury group. Chi-square analysis revealed significance to the level of $p < 0.05$ for mortality in all groups. However, no significant difference was found in the rate of discharge to home for all groups. While survival increased in the endotracheal intubation group, it was found that the number of patients with severe disability also increased. Those with severe disability were discharged to other medical institutions for ongoing care. Further, it was found that mortality rates were higher amongst the cohort transported via aeromedical services; and moreover, survival in this group was better amongst patients who did not receive endotracheal intubation (21% versus 35% respectively). Therefore, while endotracheal intubation in the field was associated with increased survival, the study was not able to demonstrate that field intubation had a beneficial effect on functional outcome for those who survived (Winchell & Hoyt, 1997).

Rainer et al. (1997) studied the efficacy of advanced pre-hospital life support in the management of trauma. The study was aimed at identifying the effect of advanced life support interventions including intravenous access, fluid resuscitation, and endotracheal intubation on outcome in trauma patients. The study was a prospective cohort study...
undertaken over a two-year period. Advanced life support paramedic interventions were compared with basic life support ambulance technician interventions.

The sample included 1,090 patients transported to hospital by ambulance and who met the criteria for the Scottish Trauma Audit Group. Chi-square analysis showed that on-scene time was significantly longer for paramedics than technicians regardless of whether an advanced life support intervention was undertaken or not (p < 0.01). No significant difference was found between paramedic and technician intervention for mortality, total days in the intensive care unit (ICU), total in-patient time, expected deaths or unexpected deaths. Results demonstrated that advanced life support interventions in trauma do not significantly reduce mortality or length of stay in ICU (Rainer et al., 1997).

To investigate the efficacy of pre-hospital endotracheal intubation, Bochicchio, Ilahi, Joshi, Bochicchio and Scalea (2003) considered the outcome in persons with severe traumatic injury, but without acutely lethal traumatic brain injury. The study was a prospective study with a sample size of 191 patients with a Glasgow Coma score of less than or equal to 8 and a head Abbreviated Injury Scale of less than or equal to 3 who were either intubated in the field or immediately on arrival at the R Adams Cowley Shock Trauma Center. Multiple logistic regression, chi-square and t-test were used to analyse data and p-values were reported in the results, however correlation coefficients were not reported. There was no significant differences in the age, ISS and pre-hospital and initial emergency department GCS. Those patients who died within 48 hours of admission were excluded from the study. It was found that patients who were intubated in
the field had significantly higher morbidity compared with those intubated on arrival at hospital, with ventilator time 14.7 days versus 10.4 days, increased in-patient time (20.2 versus 16.7 days, p < 0.04) and increased ICU stay (15.2 days versus 11.7 days, p < 0.005). Field intubations were associated with double the mortality (23% versus 12.4%, p = 0.05). Further, pre-hospital intubation was associated with a 1.5% greater risk of nosocomial pneumonia infection and increased risk of death due to respiratory failure-related complication (61% versus 29%, p < 0.05) (Bochicchio et al., 2003).

The study findings demonstrated a significant correlation between pre-hospital intubation and an increase in morbidity and mortality in trauma patients with traumatic brain injury but without an acutely lethal injury, although correlation coefficients were not provided. Descriptive results suggest a plausibly strong relationship between pre-hospital intubation and an increase in morbidity and mortality (Bochicchio et al., 2003). The results of this study should be considered in conjunction with other studies to determine the probable efficacy of pre-hospital endotracheal intubation.

Endotracheal intubation in the pre-hospital setting is quite contentious and the focus of much debate throughout the world. Murray et al. (2000) considered pre-hospital intubation of patients with severe traumatic brain injury and mortality. A total of 852 patients were enrolled into a retrospective registry-based cohort study. The sample included 3 subsets of airway control: not intubated, intubated and unsuccessful intubation. Mortality was considered for each group. Paramedics in the Los Angeles County are not authorised to administer paralysing agents. Mortality in the intubated
group was 84% (Students t-test, p = 0.07) compared with 70% in the non-intubated group (Students t-test, p = 0.37). When pre-hospital intubation was adjusted for other significant factors, the adjusted relative risk was found to be 1.74 (95% confidence interval = 1.41-2.00, p < 0.001). The relative risk associated with unsuccessful intubation was 1.53 (95% confidence interval = 1.31–5.34, p = 0.008). The results of the study support the contention that pre-hospital intubation does not improve patient outcome following severe traumatic brain injury.

Stockinger and McSwain (2004) undertook a retrospective cohort study for a 34 month period from 1 December 1999, to September 30, 2002 to compare mortality amongst trauma patients who received pre-hospital endotracheal intubation (n = 316) and those who received bag-valve-mask ventilation only (n = 217). Results revealed that those who received endotracheal intubation were more likely to die (88.9% vs 30.9%, p < 0.001). Patients receiving pre-hospital endotracheal intubation were found to have higher ISS than those who received bag-valve-mask resuscitation (p = 0.001). Analysis was adjusted to control for ISS, Revised Trauma Score, age and mechanism of injury. Pre-hospital endotracheal intubation was found to have a similar mortality rate to bag-valve-mask resuscitation. The benefit of pre-hospital endotracheal intubation over bag-valve-mask ventilation was not established. Further, Stockinger and McSwain found pre-hospital endotracheal intubation to be associated with longer time in the pre-hospital setting (22.0 minutes vs 20.1 minutes, p = 0.024).
Gausche et al. (2000) investigated the relationship between pre-hospital endotracheal intubation on survival and neurological outcome in the paediatric population. A controlled clinical trial was undertaken and 830 paediatric patients were enrolled into the study. The sample included both trauma and non-trauma patients. Bag-valve-mask ventilation was used in both the intubation and non-intubation groups. No significant differences in rates of survival or in improved neurological outcome were found between groups. Results failed to demonstrate an improved outcome with endotracheal intubation in the field in paediatric patients in the urban setting.

Further study of the efficacy of pre-hospital intubation was undertaken by Davis et al. (2005b). The study was a retrospective, registry-based analysis from the San Diego Traumatic Brain Injury Database. The study sample included 13,625 patients from five trauma centres. The overall mortality for patients undergoing pre-hospital intubation was 55% as opposed to only 15% for non-intubated patients (odds ratio [OR], 0.14; 95% confidence interval [CI] 0.13 – 0.16; p < 0.001). When adjusting for age, AIS score, ISS, pre-admission hypotension, pre-hospital intubation was still associated with higher mortality (OR, 0.36; 95% CI, 0.32 – 0.42; p < 0.001). The study supported the assertion that pre-hospital intubation of patients with moderate to severe traumatic brain injury was not associated with improved outcome. Pre-hospital intubation was associated with higher mortality risk and the benefit of pre-hospital intubation remains unproven.

The preponderance of evidence has not established improved outcomes with pre-hospital endotracheal intubation. Studies by Winchell and Hoyt (1997), Rainer et al (1997),
Bochicchio et al. (2003), Murray et al. (2000) and Gausche et al. (2000) all demonstrated similar findings: pre-hospital endotracheal intubation did not improve outcome following head or brain trauma. The studies, while retrospective in design, all featured large sample sizes which decrease the likelihood of sampling bias. Davis et al. (2005b) in this more recent study further corroborates the finds presented by previous authors. The current study will further investigate the efficacy of pre-hospital endotracheal intubation on patients with severe traumatic head injury through multiple regression analysis.

2.6.5 Cervical Collar and Spinal Immobilisation

Application of rigid cervical collar is routine in cases of severe head trauma. According to the Ambulance Service of NSW protocols, the paramedic should consider the possible presence of spinal injury and treat spinal injury if indicated (Ambulance Service of New South Wales, 2007). The justification for the routine application of a rigid cervical collar in trauma is that the collar may attenuate significant movement of the cervical spine and may also act as a marker indicating that the cervical spine has not been cleared of injury (Kolb, Summers & Galli, 1999). One study found that approximately 33% of 188 patients with spinal injuries studied had concurrent moderate to severe brain injury (GCS < 13). Brain injury was more commonly associated with high cervical injury (Iida et al., 1999).

Kolb, Summers and Galli (1999) studied 20 subjects to determine if the application of a rigid cervical collar increased ICP. The cerebrospinal fluid pressure (CSFP) was measured in adult patients undergoing lumbar puncture. Measurements were taken both pre- and post-application of a cervical collar. Mean CSFP pre-collar was 176.8 mm of
water (mmH2O) which increased to 201.5 mmH2O post-collar application. The mean difference was 24.8 mmH2O (Standard Deviation 28.4) which achieved statistical significance (p = 0.001) in a paired t-test. The clinical importance of this finding could not be verified in this study. However, Kolb et al. (1999) suggest that in patients with already elevated ICP, further increasing ICP through the application of a cervical collar may be detrimental to patient outcome and therefore the negative effects may outweigh the benefits of a cervical collar in brain trauma patients.

Bunn, Kwan, Roberts and Wentz (2001) reviewed existing research on spinal immobilisation of trauma patients in the pre-hospital setting. The review was undertaken on behalf of the World Health Organization to investigate the effectiveness of pre-hospital trauma care. The aim of pre-hospital spinal immobilisation is to restrict the mobility of the spine to prevent secondary injury during extrication, resuscitation and transport. Bunn et al. (2001) suggest that most trauma patients do not have spinal instability and will not benefit from spinal immobilisation. Primary spinal cord injury occurs at the time of impact and subsequent movement is not generally significant enough to cause further damage. Spinal immobilisation is undertaken largely in response to fear of litigation and the current protocol for pre-hospital spinal immobilisation has a strong historical precedent, rather than being based on scientific research.

Adverse effects associated with spinal immobilisation may lead to an increase in morbidity and mortality in trauma patients. The adverse effects of rigid cervical collar application in pre-hospital spinal immobilisation include the development of airway
obstruction, increased intracranial pressure, increased risk of aspiration, restricted respiration, dysphagia and skin ulceration (Bunn et al., 2001). Such adverse conditions can result in unnecessary radiation exposure from medical imaging, longer hospital stay and increased medical costs. Further, the potential risk of aspiration and respiratory compromise can lead to increased incidence of preventable death from asphyxiation. Therefore, as airway obstruction is a major cause of preventable death in trauma patients, spinal immobilisation may increase mortality and morbidity. The value of pre-hospital spinal immobilisation remains uncertain (Bunn et al., 2001).

2.6.6 Airway Adjunct and Respiratory Tract Infection

Various studies have been undertaken to determine the incidence of nosocomial infection, or infection acquired after admission to hospital; however, little research has been done into the incidence of pre-hospital acquired infection. Erbay et al. (2003) studied the incidence and risk factors associated with nosocomial infection in a Turkish university hospital during 2000 and 2001 and found that 26% patients acquired 225 different types of nosocomial infections. The major types of infections were pneumonia (40.9%), bacteraemia (30.2%) and urinary tract infections (32.6%). Logistic regression analysis was undertaken to establish the major risk factors for the development of nosocomial infections. Erbay et al. (2003) found that mechanical ventilation (OR = 16.35, p < 0.001), coma (OR = 15.04, p < 0.001), traumatic injury (OR = 10.27, p = 0.002) and presence of nasogastric tube (OR = 2.94, p = 0.002) or tracheostomy tube (OR = 5.77, p = 0.04) were associated with increased risk of infection and the rates of infection increased as the
number of risk factors increased. Mortality rates amongst infected patients were 3 times higher than those of non-infected patients (60.9% vs 22.1%).

Helling, Evans, Fowler, Hays and Kennedy (1988) reviewed 82 patients with head injuries admitted to a level one trauma centre in Kansas, USA for rates and cause of respiratory infection. As a result of depressed respiratory function, prolonged ventilator support with endotracheal intubation and post-traumatic immunosuppression, patients with head injuries may be particularly vulnerable to respiratory sepsis. Of the 82 patients reviewed, 9 (11%) died during hospitalisation and 41 (50%) experienced at least one infectious complication, with 34 of the 41 (83%) developing pulmonary infections. Three died from septic events. The average onset of pulmonary infection was 3.2 days following admission. Eighteen patients (22%) experienced the onset of pneumonia within two days of admission (Helling et al., 1988). The study assumed the infections were nosocomial in origin; the possibility of pre-hospital acquired infection was not considered.

Bochicchio et al. (2003) undertook a randomised prospective study of 191 patients with severe traumatic brain injury. The aim of the study was to determine the outcome of trauma patients intubated in the field compared with patients who were intubated on arrival at hospital. Seventy-eight (41%) of the 191 were intubated in the field verses 113 (59%) who were intubated in the hospital setting. No significant differences were found with age, GCS, head Abbreviated Injury Scale score or Injury Severity Score between the two groups.
Pre-hospital intubation was associated with poorer outcomes in several areas. Patients who were intubated in the field had significantly higher morbidity with mean ventilator days being 14.7 days versus 10.4 days for hospital intubated patients. Chi-square analysis revealed that patients who were intubated in the field had longer intensive care unit and in-hospital stays (15.2 vs 11.7, p < 0.005 and 20.2 vs 16.7, p < 0.04 respectively). Pre-hospital intubated patients had a significantly higher incidence of pneumonia (49% versus 32%) resulting in a relative risk of 1.53 greater in the pre-hospital intubation group. The pre-hospital group also had a significantly greater mortality rate (23% vs 12.4%, p = 0.05), a greater risk of mortality of 1.85 times that of the hospital intubated group. The pre-hospital intubation group was also found to have a greater risk of mortality from respiratory failure-related complications (Bochicchio et al., 2003). Results suggest strongly that pre-hospital intubation was associated with higher rates of nosocomial infection.

Karch, Lewis, Young and Ho (1996) also found that pre-hospital intubation was associated with the development of nosocomial pneumonia in a retrospective study of 94 severely injured patients. Of those who survived 50% (13 of 26) were intubated in the pre-hospital setting compared with 67% (37 of 71) non-survivors. Patients who had pre-hospital intubation were three times more likely to develop nosocomial pneumonia than those having hospital intubation. The small sample size and retrospective methodology may limit the power of the study; however, when considered in conjunction with other studies, the results do reflect the consensus of findings.
Further, Eckert et al. (2004) found in a retrospective study of 571 patients that several risk factors were associated with the development of nosocomial pneumonia after trauma. The risk factors included pre-hospital intubation, severe head injury, shock, blunt force trauma and high Injury Severity Score. Pre-hospital intubation was associated with significantly higher incidence of pneumonia (35% versus 23%, p = 0.048). Pneumonia was directly related to increased length of intensive care unit stay and total hospital stay (p < 0.001) and pre-hospital intubation was an independent risk factor for the development of pneumonia. Moreover, the study did not find emergency department intubation to be a risk factor for the development of pneumonia. Davis, Stern, Ochs, Sise and Hoyt (2005c) compared 352 patients who received rapid sequence induction and intubation in the field versus 704 matched controls. Aspiration pneumonia was more common in the RSI group (5.6% vs 2.8%, p = 0.045). Results suggest pre-hospital RSI and intubation does not prevent aspiration pneumonia. Both the Eckert et al. and Davis et al. studies support the assertion that pre-hospital intubation is associated with higher incidence of pneumonia and subsequent morbidity.

The current study will further explore the development of nosocomial infection in relation to the use of pre-hospital airway adjuncts. This study will investigate the insertion of an airway adjunct in the pre-hospital setting and the onset of respiratory infection in those patients. The outcome of those patients with respiratory infection will be analysed as well as time in ICU and total in-hospital days.
2.6.7 Maintenance of Blood Pressure

It is recommended that systolic blood pressure be maintained above 90 mmHg and mean arterial blood pressure be maintained at 70 mmHg or greater in adult patients with traumatic brain injury (Brain Trauma Foundation (2000a). Greaves, Porter and Revell (2002) advocate that due to modulation of the inflammatory response, critically ill patients exhibit increased capillary permeability. This results in the movement of molecules such as albumin and water across the capillary membrane and into the interstitium and therefore exacerbates cerebral oedema and further impedes oxygen transfer to cells. To avoid over-infusion of fluid and exacerbation of oedema Greaves et al. (2002) recommend that fluids be titrated in small boluses of 250 ml until a radial pulse is restored. Exceptions to the administration of 250 ml boluses include penetrating torso injury, isolated head injury and infants.

The Ambulance Service of NSW (1997) asserts there are four key signs of severe shock related to hypovolaemia:

1. Poor brain perfusion (restlessness and altered level of consciousness),
2. Poor skin perfusion (cold, pale, sweaty, capillary refill > 2 seconds)
3. Tachycardia (adult > 100, school child > 100, preschooler > 130, infant > 170 beats per minute)
4. Hypotension (blood pressure: adult < 90 mmHg systolic, school child < 80 mmHg systolic, preschooler < 70 mmHg systolic, infant < 60 mmHg systolic) (Ambulance Service of New South Wales, 1997, protocol 42).
Fluid resuscitation should be commenced and maintained while 2 or more key signs of shock are present. An optimal or upper limit of systolic blood pressure for the termination of fluid resuscitation is not stipulated in either the hypovolaemia or head injury protocols. The recommended bolus dose of fluid is 10 ml/kg (Ambulance Service of New South Wales, 1997). Paramedics manipulate intravascular volume through the infusion of crystalloid (Hartmann’s) and/or colloid (Haemaccel) fluids (Ambulance Service of New South Wales, 1997). Myburgh et al. (2008) in their study of outcomes following traumatic brain injury in Australia and New Zealand during the 2000 to 2001 period found 35.5% of patients received crystalloids and 22.7% of patients received colloids ($n = 635$).

Notably, Shafi and Gentilello (2005) found pre-hospital endotracheal intubation to be an independent predictor of hypotension upon arrival at the emergency department (pre-hospital ETI 54%, $n = 871$ vs emergency department ETI 33%, $n = 6581$, $p < 0.001$) in a retrospective study of pre-hospital intubated trauma patients from the National Trauma Data Bank in the USA from 1994 to 2002. The authors postulated that positive pressure ventilation causes hypotension in severely injured hypovolaemic patients.

This study will explore this issue further by analysing the outcomes of patients with severe traumatic head injury who receive pre-hospital fluid resuscitation and endotracheal intubation in the Sydney and metropolitan areas.
2.6.8 Time in the Pre-Hospital Setting and Advanced Pre-Hospital Interventions

Time in the field for trauma patients has been a controversial issue. Debate has taken place regarding the efficacy of ‘stabilise on scene’ versus ‘scoop and run’ to minimise pre-hospital time. The introduction of pre-hospital advanced life support was aimed at reducing mortality rates following trauma. Numerous studies since the early 1990s have failed to establish improved outcomes of pre-hospital advanced life support in trauma. Rainer et al. (1997) used a prospective study to compare outcomes of trauma patients admitted to an accident and emergency department in an Edinburgh hospital over a 2-year period (n = 1,090). Comparisons were made between patients transported by paramedic crews and patients transported by technician crews. Paramedic crews were trained in advanced life support (ALS) procedures including peripheral venous cannulation, fluid administration and tracheal intubation, whereas technician crews did not have ALS training. Chi-square analysis found no differences in injury severity between paramedic and technician groups (x², p < 0.0001).

The study found that on-scene times for the paramedic group were significantly longer when ALS skills were undertaken, including intravenous cannulation, fluid administration and/or tracheal intubation (x², p < 0.001). For patients who received no ALS interventions, scene time was significantly shorter (18 minutes for paramedics vs 15 minutes for technicians; x², p < 0.01). Moreover, improved outcome following ALS interventions was not established. There was no significant reduction in mortality or
length of stay in the intensive care unit for those patients who received ALS interventions in the pre-hospital setting (Rainer et al., 1997).

Harvey, Gerard, Rice and Finch (1998) reviewed actual versus patient perceived pre-hospital times in an emergency department which serviced a large urban centre in the USA (n = 137). The study found actual mean pre-hospital times included a response time of 9.1 minutes, on-scene time of 12.4 minutes and time to definitive care (hospital) 34.9 minutes versus patient perceived times: response time 12.4 minutes, on-scene time 9.1 minutes and time to definitive care 29.3 minutes. Multivariate analysis found the difference between actual and perceived times to be significant (r = -0.46, p < 0.001).

Demetriades et al. (1996) considered the mode of transportation of trauma victims: paramedic transportation or private means of transportation (friends, relatives, bystanders). The two groups were similar in terms of mechanism of injury, the need for surgical intervention and admission to an intensive care unit. The outcome measure used was mortality rate. The sample size was 5,782 with 4,856 in the paramedic group and 926 in the private group. The crude mortality rate was 9.3% in the paramedic group and 4.0% in the private group (relative risk = 2.23, p < 0.001). Linear regression was used to control for confounding variables such as, age, gender, mechanism of injury, cause of injury, Injury Severity Score, and severity of head injury. These variables are not affected by mode of transportation. Further subgroup analysis following adjustment for ISS was undertaken. Subgroup analysis of severe injury (ISS greater than 15) found that paramedic transportation patients had twice the mortality rate when compared with
private transportation patients (28.8% vs 14.1%). When confounding variables were controlled, the adjusted mortality rate for patients with an ISS greater than 15 was 28.2% in the paramedic group versus 17.9% in the private group (p < 0.001) (Demetriades et al., 1996). The sub-group of patients with critical injuries (ISS greater than 30) was skewed with 6.0% of the total population transported by paramedics as compared to 2.3% of the private group. Following adjustment for this variable the relative risk of mortality was still higher in the paramedic transportation group at 1.60 (95% confidence interval, 1.18 to 2.15; p = 0.002) (Dimetriades et al., 1996).

Possible explanations for the findings were proposed. Time in the pre-hospital setting for those transported by paramedics was assumed to be longer as compared to the private group. Paramedic response times averaged 8 minutes, time on scene averaged 22.5 minutes and average transport time was 7 minutes. The total mean pre-hospital time was 37 minutes. It was postulated that persons transported by private mode reached hospital 30 minutes earlier than those transported by paramedics. The non-paramedic group therefore received earlier definitive interventions ahead of the paramedic transportation group. Estimates suggest that for every 10 minutes delay in receiving definitive treatment, survival drops by 10% (Demetriades et al., 1996).

Harvey et al. (1998) and Demetriades et al. (1996) found mean times in the pre-hospital setting for paramedics in large urban areas in the USA to be similar for response time and time to definitive care: response time of 9.12 minutes, on-scene time of 12.39 minutes and time to definitive care 34.99 minutes (Harvey et al., 1998) versus response time of 8
minutes, on-scene time of 22.5 minutes and time to definitive care 37 minutes (Demetriades et al., 1996). The most variance is seen in on-scene time with Demetriades et al., (1996) finding on-scene time to be almost double that of Harvey et al. (12.39 vs 22.5 minutes). Neither study considered the effect of on-scene time alone on outcome following traumatic injury.

Baez, Lane, Sorondo and Giraldez (2006) studied the predictive effect of out-of-hospital time in outcomes of severely injured young adults and elderly patients. The study was a cross-sectional observational study of all injured patients transported by paramedics within the State of Pennsylvania during 1997. Multivariate logistic analysis was used to control for gender and injury severity based on ISS and Revised Trauma Scores (RTS). Amongst the young adult group out-of-hospital time correlated significantly with length of hospital stay (p = 0.001) and frequency of complications (p = 0.016), but not with mortality (p = 0.264). In the elderly group however, pre-hospital time was not found to be an effective predictor of length of hospital stay (p = 0.512), frequency of complications (p = 0.512) or mortality (p = 0.954). Correlation coefficients were not reported.

Delay preceding evacuation of an intracranial haematoma has been found to significantly increase the rate of mortality in patients with head injuries. Rose, Valtonen and Jennett (1977) reviewed 116 patients with head injuries who were known to have talked before they deteriorated and died. Post-mortem examination revealed that delay to neurosurgical evacuation of an intracranial haematoma was the most common avoidable factor causing death in 63 patients (54%). Other avoidable causes were hypoxia and hypotension.
Bickell et al. (1994) first observed the positive effects of delayed fluid resuscitation on persons with penetrating torso injuries with a systolic blood pressure less than 90 mmHg in the pre-hospital setting. The prospective study comprised a sample of 598 patients with penetrating torso injuries and low (< 90 mmHg) systolic blood pressure. The aim was to determine the effects of immediate (pre-hospital and emergency department) resuscitation and delayed (operating theatre) resuscitation. Delayed fluid resuscitation was received by 298 patients, while 309 patients received immediate resuscitation. The delayed resuscitation group had a lower mortality rate with 70% surviving to discharge as compared with the immediate resuscitation group with only 62% surviving to discharge ($x^2, p = 0.04$).

Among the delayed fluid resuscitation group it was noted that there was a lower incidence of adult respiratory distress syndrome, sepsis syndrome, acute renal failure, coagulopathy, wound infection and pneumonia. In the delayed fluid resuscitation group, 23% experienced one or more of these complications; as compared with 30% in the immediate fluid resuscitation group ($x^2, p = 0.08$). The benefit of pre-hospital fluid resuscitation was not demonstrated in individuals with penetrating torso injuries. However, because of the different nature of the primary injury, care should be exercised when extrapolating the findings to other types of trauma, including severe head trauma (Bickell et al., 1994).
Regal et al. (1997) retrospectively studied 1,223 multi-trauma patients and analysed the effect of pre-hospital care in respect to delayed complications. It was found that there was a significantly higher rate of multiple organ failure (MOF) in patients with a rescue time in excess of 30 minutes. The average rescue time for the entire sample was 45 minutes. Hypothermia and coagulation disorders were also found to be prevalent in this sub-group. Winter, Adamides, Lewis and Rosenfeld (2005) recommend that if the estimated time of arrival from the field to the hospital is less than 10 minutes, then pre-hospital interventions should be aimed at correcting hypoxia and hypotension only and that scene time should be kept to a minimum. If time to hospital is less than 10 minutes and the patient has a patent airway and a systolic blood pressure of 90 mmHg or more, advanced life support interventions are not recommended. Advanced life support interventions include intravenous access, fluid administration and endotracheal intubation.

Studies reviewed advocate minimising time to definitive interventions (hospital) as delays in the pre-hospital setting result in poorer outcome following severe injury (Rainer et al., 1997; Harvey et al., 1998; Demitriades et al., 1996; Baez et al., 2006 & Rose et al., 1977). The bulk of evidence suggest pre-hospital ALS interventions do not improve outcome following severe injury (Rainer et al., 1997; Bickell et al., 1994; Regal et al., 1997 & Winter et al., 2005); however, the correction of hypotension is nevertheless recommended in the pre-hospital setting (Brain Trauma Foundation 2000a; Greaves et al., 2002 & Chi et al., 2006). The current study investigates the issues of time in the pre-hospital setting and advanced life support interventions by analysing the impact of these variables on the functional outcome of the persons with severe traumatic injury.
2.6.9 Receiving Hospitals

Paramedics use a trauma triage system to determine the destination or receiving hospital for the trauma patient. The pre-hospital triage of trauma considers three parameters: physiology, injury and mechanism of injury (Ambulance Service of New South Wales, 1997; Ma, MacKenzie, Alcorta & Kelen, 1999).

The physiological parameter includes any significant physiological derangement, such as:

- respiratory distress with rate < 10 or > 30 or cyanosis
- systolic blood pressure < 90 mmHg or absence of radial pulse in children
- GCS < 14, or requires at least ‘shake and shout’ to arouse, or decreasing level of consciousness, or any depression of consciousness in children
- Burns > 20% in adults and > 10% in children

(Ambulance Service of New South Wales, 1997; Ma et al., 1999).

Persons with significant and specific injury are considered to have major trauma. Injury criteria indicative of significant injury include:

- severe single system injury
- multiple system injury
- central nervous system injury
- penetrating injury to the head, neck, torso or extremities
- head injury with 1 or 2 dilated pupils or severe facial injury or open head injury
- abdominal distension or rigidity

(Ambulance Service of New South Wales, 1997; Ma et al., 1999).
Mechanism of injury alone may be used as a criterion for transport to a trauma centre without the presence of additional physiological or injury criteria. Significant mechanism is considered as:

- vehicular crash > 60 Km/hour
- major deformation of vehicle
- fatal injury in the vehicle
- person ejected from within or on the vehicle
- fall from height > 5 metres
- cyclist or pedestrian hit by vehicle travelling > 30 Km/hour

(Ambulance Service of New South Wales, 1997; Ma et al., 1999).

Compliance by paramedics with trauma triage criteria is crucial to ensure trauma patients receive definitive care at trauma centres. Patients displaying any of the trauma triage criteria should be transported directly to a trauma centre. District or non-trauma centre hospitals should be bypassed (Ambulance Service of New South Wales, 1997). In many cases, however, the trauma centre is the closest hospital and the conscious adherence to trauma triage criteria by the paramedic is assumed.

Ma et al. (1999) investigated the compliance rates of paramedics in adhering to trauma triage criteria. A total of 7,652 patients met one or more of the trauma triage criteria in the state of Maryland, USA in 1995. In particular, 6,055 met one criterion only with 1,764 meeting the physiology criteria, 844 meeting the injury criteria and 3,447 meeting the mechanism criteria. Two criteria were met by 1,597 and 354 met all three criteria.
Overall compliance was highest for those who met the injury criteria with or without other criteria (86.0 – 94.0%), followed by patients who met both mechanism and physiology criteria (68.7%) and those who met only the mechanism criteria (45.8%). Compliance for those meeting the physiology criterion alone was the lowest (34.0%) (Ma et al., 1999). The patient not receiving timely appropriate level of hospital care is the consequence of paramedic failure to adhere to trauma triage criteria.

A statistically significant downward trend in the level of compliance with patient age was noted. For those aged between 0 to 54 years compliance levels were 47.0% compared with 39.7% for those 55 years or greater (x², p = 0.002). The lowest level of compliance was observed in patients who met the physiology criteria. Within this group, patients who were aged 15 to 54 years were two times more likely to be transported to a trauma centre than those older than 55 years. The odds of being transported to a trauma centre was poorer for those over 75 years: 2.71 (2.43 – 3.02) as compared with 0 to 54 years, 1.55 (1.43 – 1.68) and 55 to 74 years, 1.07 (0.96 – 1.19). This trend has significant implications for the management and outcome of those aged 55 years or more (Ma et al., 1999).

Intracranial haematoma following head trauma in some cases will require surgical craniotomy and evacuation of the haematoma. Early, rapid surgical intervention has been shown to provide better functional outcomes. Time to surgical intervention is therefore a determinate of functional outcome in patients with large intracranial haematoma. Such
patients require direct transport to a trauma centre. Secondary transport from a non-trauma facility will result in delay to surgical intervention and increase the risk of secondary insult to the brain (Wright, Knowles, Coats & Sutcliffe, 1997).

2.6.10 Time to Craniotomy

The evacuation of intracranial haematoma should be performed in an efficient and timely manner. To achieve this, persons with severe traumatic brain injury should be transported directly from the site of injury to an area trauma hospital or specialist centre with facilities and staff available to perform the craniotomy (Wright et al., 1996). A secondary inter-hospital transfer of persons with head injury is known to increase the time to neurosurgical intervention and also increases the risk of secondary insult to the brain. An inverse relationship has been established between time to craniotomy and functional outcome following brain injury. A reduction in time to craniotomy has been shown to result in a better neurological outcome. Therefore, persons with severe traumatic brain injury should be transported directly to a neurosurgical facility (Wright et al., 1996).

Delay in definitive neurosurgical intervention and adverse outcome was noted by Rose, Valtonen and Jennet (1977). Rose et al. reviewed 116 persons with traumatic brain injury who were known to have talked before dying from their injuries. In 63 (54%) of the sample, delay in the evacuation of intracranial haematoma was the most common avoidable factor contributing to death. In a further 22 cases (19%), delay in treatment was thought to be a possible factor contributing to death. A delay was recorded when an unreasonable time had elapsed from the time of clinical deterioration and positive
diagnostic or therapeutic action. Clinical deterioration included a significant alteration in level of consciousness or the occurrence of focal neurological abnormality of the limbs or in the pupils. It is believed that early diagnosis and urgent surgical intervention are the only measures that will reduce high mortality rates.

For patients requiring neurosurgical intervention, it is recommended that a maximum time from the onset of injury to definitive surgical intervention not exceed 4 hours. In order to achieve improved outcomes, all persons with severe traumatic brain injury should be transported directly to a neurosurgical facility where possible (Lind, Heppner, Robins & Mee, 2005). Thirty eight intubated patients who were transferred from secondary and tertiary hospitals to a trauma centre in New Zealand were reviewed by Lind et al. (2005). The median time from injury to arrival at the trauma centre was 6.5 hours. The majority of patients did not arrive within the recommended 4 hours from the time of injury. Outcome measures were not considered in this study.

This study will further investigate the adherence of paramedics to trauma triage principles and investigate the outcomes of those patients who required craniotomy and were transported to a non-trauma facility as compared with those transported directly to a trauma centre.
2.6.11 Summary

Severe traumatic head injury is associated with significant mortality, morbidity and economic cost in Australia. The economic burden to Australia during the 2004 and 2005 period was $184 million (Australian Institute of Health and Welfare, 2011). Lyle et al. (1990) found that the majority of persons who survived traumatic brain injuries would have some degree of residual disability following hospitalisation. To date, little research has been undertaken to investigate the pre-hospital management of persons with severe traumatic head injury in Australia, and the effect of paramedic interventions on mortality rates and functional outcome.

The Glasgow Coma Scale is used to assess the depth and duration of impaired consciousness or coma (Teasdale & Jennett, 1974) and was used an inclusion criteria for this study. The GCS has been used since the 1970s as a robust measure of impaired consciousness and also a robust predictor of functional outcome following trauma (Jennett, 2002; Pal et al., 1989; Ross et al., 1998). The strong inter-rater reliability of the GCS has been demonstrated in several studies (Menegazzi et al., 1993; Bazarian et al., 2003; Rowley & Fielding, 1991; Davis et al., 2006c; Kerby et al., 2007).

Several studies have demonstrated the efficacy of direct transportation to a trauma facility for those suffering severe traumatic head injury. Studies by Shackford et al, 1986, Samplis et al., 1995 and Harlt et al., 2006 found direct transportation of persons suffering traumatic injury to a trauma centre to be associated with reduced mortality and morbidity. In the Australian context little is known of the adherence of paramedics to trauma
transport protocols and the impact of transport decisions on patient outcome following severe traumatic head injury. This study will investigate this issue further.

The Glasgow Outcome Scale is a five-category scale used to estimate functional outcome following neurotrauma (Hall et al., 1985). The GOS has been a widely used scale for assessing outcome following severe traumatic head injury since 1975 (Teasdale et al., 1998). The strong validity and inter-rater reliability have been demonstrated in the studies of Teasdale et al. (1998), Satz et al., (1998) and Wilson et al. (2000). The GOS was used as the measure of outcome in this study.

The ability of paramedics to effectively triage trauma patients is uncertain. Shaban, Wyatt Smith and Cumming (2004) suggest that research into human clinical judgement in medicine and health is mainly limited to the medical and nursing professions. Much of ambulance practice including paramedic judgement and decision making have not undergone systematic and sustained research and therefore there is a paucity of evidence that examining the mechanics of human error in paramedic practice. The relationship between knowledge, judgement and decision making in paramedic practice has not been established. In Australian ambulance services paramedics operate under the auspices of guidelines or protocols and may elect to withhold advanced life support interventions if the paramedics consider that undertaking the interventions will not benefit patient outcome and/or will increase time to definitive treatment within the in-hospital setting. In New South Wales paramedics may utilise the Urgent Transport protocol in order to expedite transportation of persons with severe traumatic head injury (Ambulance Service
of New South Wales, 1997, 2007). Little published research exists in the area of paramedic adherence to protocol or the clinical decision of paramedics to withhold advanced life support interventions in order to minimise time at scene and time to definitive treatment. This study will investigate paramedic adherence to treatment and transportation protocol and investigate time in the pre-hospital setting and the functional outcome of persons with severe traumatic head injury.

Intensive care paramedics are authorised to undertake interventions such as endotracheal intubation and administer fluid volume replacement (Ambulance Service of New South Wales, 1997, 2007). Studies into the efficacy of pre-hospital endotracheal intubation have resulted in some conjecture. Winchell and Hoyt (1997) found pre-hospital endotracheal intubation to be associated with a decrease in mortality from 57% to 36% in a cohort of severe head injured patients. Regal et al. (1997) found pre-hospital endotracheal intubation to be associated with a lower rate of multiple organ failure in patients with an ISS between 20 and 39. However the studies of Rainer et al. (1997), Murray et al. (2000), Bochicchio et al. (2003) and Davis et al. (2005b) failed to demonstrate improved outcome with pre-hospital endotracheal intubation. While the preponderance of evidence has not established improved outcomes with pre-hospital endotracheal intubation, the intervention continues to be carried out by intensive care paramedics and requires further investigation. This study will further investigate the efficacy of pre-hospital intubation on patients with severe traumatic head injury in the Sydney and metropolitan areas.
Cerebral perfusion pressure is directly related to mean arterial blood pressure. Episodes of hypotension have been linked to deleterious outcomes following severe traumatic brain injury (Brain Trauma Foundations, 2000a). In the pre-hospital setting, blood pressure is maintained through the manipulation of intravascular volume and fluid volume replacement. McGraw (1998) found CCP of 80 mmHg or greater to be associated with improved outcomes. Marmarou et al. (1991) demonstrated a relationship between intracranial pressure and systolic blood pressure and recommended an ICP of 20 mmHg and a systolic BP of 80 mmHg were critical levels. An ICP greater than 20 mmHg and a systolic BP of less than 80 mmHg were predictors of poorer outcomes. The deleterious effect of hypotension following trauma were also demonstrated in the studies of Eisenberg et al. (1988, Marshall et al. (1979), Saul and Ducker (1982), Chesnut (1997), Shackford (1992) and Pietropaoli et al. (1992). However cerebral perfusion pressure is possibly more important than systolic blood pressure, as systolic blood pressure can be an unreliable as the relationship between CPP and systolic blood pressure is not consistent (Brain Trauma Foundation, 2007). The relationship between blood pressure, mean arterial pressure, cerebral perfusion pressure and intracranial pressure is complex. The efficacy of the manipulation of a single parameter, in this case blood pressure, therefore remains unclear. This study investigates the relationship between fluid volume replacement and outcome following severe traumatic head injury.

The maintenance of a patent airway and oxygen saturations greater than 90% is imperative in reducing the risk of secondary insult following severe traumatic brain injury (Brain Trauma Foundation, 2000a). Several studies have demonstrated that
hypoxaemia is associated with poor outcome following severe traumatic head injury (Atkinson et al., 1998; Warner et al., 2007; Davis et al., 2006b & White et al., 1994); however, there is scant published research which investigates the maintenance of a patent airway and oxygenation of persons with severe traumatic head injury within the Australian context. This study investigates this issue further by analysing the effect of airway maintenance, oxygenation and ventilation of patients with severe traumatic head injury by paramedics of the Ambulance Service of NSW.

The results of several studies recommend minimising time to definitive treatment (hospital) as delays in the pre-hospital setting result in poorer outcomes following severe traumatic injury (Rainer et al., 1997; Harvey et al., 1998; Demetriades et al., 1996; Baez et al., 2006 & Rose et al., 1997). The preponderance of evidence suggests advanced life support interventions do not improve outcome following severe traumatic injury (Rainer et al., 1997; Bickell et al., 1994; Regal et al., 1997 & Winter et al., 2005). This study will investigate the issues of time in the pre-hospital setting and advanced life support interventions by analysing the effect of these variables on functional outcome following severe traumatic head injury in the Sydney and metropolitan areas.
CHAPTER THREE

METHODOLOGY

3.1 Introduction

The early assessment and treatment provided by ambulance paramedics is the first crucial step in the care of individuals with severe traumatic head injury. Post-injury cognitive and functional recovery relies on effective treatment throughout the entire care timeframe. Little is known however about the efficacy of paramedic interventions during pre-hospital care.

The conceptual model (Figure 1) specifies the field of interest that were important in this study of the relationship between pre-hospital management and functional outcome of persons with severe traumatic head injury \((n = 326)\).

In this chapter, the research methods used for the study are described. The chapter begins with an outline of the study, followed by the steps taken to develop, pre-test and pilot test the study instruments, and the steps taken to analyse the quantitative data.

3.2 Outline of the Study

This section presents a brief overview of the study including the study aims, research questions, the study objective and the sample frame.
3.2.1 The Study Aim and Research Hypotheses

The aim of the study was to determine the relationship between the pre-hospital management and patient functional outcome in severe traumatic head injury.

It was hypothesised that:

1. Persons with severe traumatic head injury will benefit from the attendance of intensive care paramedics and pre-hospital endotracheal intubation provided by intensive care paramedics.
2. Maintenance of oxygen saturation of greater than or equal to 95% and maintenance of a clear and patent airway will benefit patients with severe traumatic head injury.
3. Patients with severe head injuries and hypotension will benefit from early intravenous fluid volume replacement to maintain a systolic blood pressure of greater than 90 mmHg.
4. Functional outcome of patients with head injuries is inversely related to pre-hospital response time, time at scene, the total time in the pre-hospital setting and time to neurosurgical intervention (craniotomy): as pre-hospital time and time to craniotomy increases, functional outcome decreases.

3.2.2 Study Design

A retrospective cohort study was used to investigate the relationship between pre-hospital management and functional outcome of persons with severe traumatic head injury. A prospective cohort study was not appropriate as it was not possible for one researcher to attend 10 area trauma hospitals to collect data as it became available. A retrospective
design was determined to be the most appropriate methodology. Retrospective cohort studies based on reliable data from databases or registries, case reports or clinical series are considered by the Brain Trauma Foundation (2000a) to yield Class III evidence.

3.2.3 Inclusion Criteria

The inclusion criteria for the study were as follows:

- Traumatic head injury
- Glasgow coma score of 8 or less in the pre-hospital setting
- Injury occurring in the Sydney and metropolitan areas
- Treatment and/or transport provided by the Ambulance Service of NSW
- Injury occurring between 1 January 1999 and 31 December 2001
- Patients were transported directly from the scene to a hospital within the Sydney and metropolitan areas.

3.2.4 Exclusion Criteria

The exclusion criteria for the study were as follows:

- Patients with normal intracranial findings on the computed tomography (CT)
- Patients with prior severe brain trauma or significant disability
- Persons with a Glasgow Coma Score greater than 8 in the pre-hospital setting
- Injuries occurring in rural areas or inter-hospital transfers from rural areas
- Patients injured prior to 1 January 1999 or post-31 December 2001. At the time of data collection, for those patients injured post-31 December 2001, many medical
records were incomplete due to on-going patient treatment within the hospital setting.

3.3 Area Trauma Hospitals

3.3.1 Area Trauma Hospitals in the Sydney and Metropolitan Areas

The area trauma hospitals were selected as locations for data collection as patients meeting the inclusion criteria for the study should be transported directly to an area trauma hospital (Ambulance Service of NSW, 1997). District hospitals should be bypassed unless the patient is likely to undergo cardiac arrest during transportation and the district hospital is the closest hospital destination (Ambulance Service of NSW, 1997). Ambulance Service of NSW (1997) specify that all patients with serious trauma to any body region, respiratory distress, systolic blood pressure less than 90 mmHg, requires at least ‘shake and shout’ to arouse response, or falling level of consciousness in adults, and any cause of depressed level of consciousness in children should be transported directly to an area trauma hospital. For those patients not transported directly to an area trauma hospital, the majority would be transferred via medical retrieval from a district hospital to an area trauma hospital following stabilisation. Transfer is undertaken by the Medical Retrieval Unit and copies of the pre-hospital and receiving hospital patient care documents were transported with the patient during medical retrieval. Both ambulance and medical data were accessed through the medical record within the medical records department at each area trauma hospital. Area trauma hospitals and the cases reviewed, cases excluded and cases included into the study are presented in Table 8.
Table 8  Area Trauma Hospitals, Cases Reviewed, Cases Excluded and Cases Included into the Study

<table>
<thead>
<tr>
<th>Hospital</th>
<th>Reviewed</th>
<th>Excluded</th>
<th>Included</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prince of Wales</td>
<td>53</td>
<td>37</td>
<td>16</td>
</tr>
<tr>
<td>Westmead</td>
<td>161</td>
<td>99</td>
<td>62</td>
</tr>
<tr>
<td>Royal Prince Alfred</td>
<td>117</td>
<td>63</td>
<td>54</td>
</tr>
<tr>
<td>St Vincent's</td>
<td>109</td>
<td>83</td>
<td>26</td>
</tr>
<tr>
<td>Liverpool</td>
<td>123</td>
<td>62</td>
<td>61</td>
</tr>
<tr>
<td>St George</td>
<td>108</td>
<td>61</td>
<td>47</td>
</tr>
<tr>
<td>Nepean</td>
<td>48</td>
<td>28</td>
<td>20</td>
</tr>
<tr>
<td>Sydney Children’s</td>
<td>22</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>Westmead Children’s</td>
<td>26</td>
<td>14</td>
<td>12</td>
</tr>
<tr>
<td>John Hunter Children’s</td>
<td>22</td>
<td>5</td>
<td>17</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>789</strong></td>
<td><strong>463</strong></td>
<td><strong>326</strong></td>
</tr>
</tbody>
</table>

There are 12 hospitals within the Sydney and metropolitan areas designated as an area trauma hospital or Trauma Service Provider (New South Wales Health, 2009). A total of 10 area trauma hospitals were included into the study with the exception of Royal North Shore Hospital and John Hunter Hospital. For ethics approval to be gained at each hospital, the sponsorship of a senior medical officer member of the hospital staff to act as an associate researcher was required. Sponsorship at Royal North Shore and John Hunter hospitals was not obtained with the reason given for the decline as an inability of senior members of staff to commit to the study due to time restraints.
3.3.2 Powering the Study and Profiles of Area Trauma Hospitals

The pilot study identified 52 cases of severe head trauma which met the inclusion criteria from the total cases transported by the ASNSW state-wide during 1998 \((n = 35,973)\). Literature searches failed to identify data depicting the total population of severe head trauma cases managed within the in-hospital setting in the Sydney and metropolitan area during the period from 1998 to 2003. As a rule small differences between the sample statistic and the population parameter can be statistically significant if the sample size is large and the likelihood of sampling error is reduced (Urdan, 2010). Analysis of the Brain Trauma Foundation (2000b) evidentiary tables demonstrated that the majority of studies cited had sample sizes of less than 200 persons with severe traumatic head injury. In studies relating to oxygenation and blood pressure 14 of 17 studies had samples of less than 200 persons. In studies of airway, ventilation and oxygenation, all five studies cited had samples of less than 200 persons while four of the six studies in fluid resuscitation had sample sizes of less than 200 persons (Brain Trauma Foundation, 2000b). Therefore, the number of cases required to be considered representative of the population of severely head injured persons, to provide sample validity, control for sampling bias and provide a large cohort for parametric analysis was considered to be 300. It was concluded that the required number of cases could be attained from 10 area trauma hospitals with all cases of severe traumatic head injury which met the inclusion criteria to be included into the study.

A profile of each area trauma hospital is provided in an overview of emergency department attendances for all area trauma hospitals included into this study during 1999
to 2000 period and total trauma admissions with an ISS greater than or equal to 16 during 2002 is provided below (New South Wales Health, 2001; New South Wales Health, 2002). Royal North Shore Hospital and John Hunter Hospital have been included to identify and compare the case load of these two hospitals that did not participate in the study with those that did participate.

- Prince of Wales Hospital, Randwick (emergency department attendances 1999/2000 = 38,141; total admissions 2002 trauma cases with an ISS ≥ 16 = 122).
- Liverpool Hospital, Liverpool (emergency department attendances 1999/2000 = 42,533; total admissions 2002 trauma cases with an ISS ≥ 16 = 251).
- Nepean Hospital, Penrith (emergency department attendances 1999/2000 = 36,985; Total admissions 2002 trauma cases with an ISS ≥ 16 = 107).
- Royal Prince Alfred Hospital, Camperdown (emergency department attendances 1999/2000 = 44 203; total admissions 2002 trauma cases with an ISS ≥ 16 = 194).
- St George Hospital, Kogarah (emergency department attendances 1999/2000 = 43 202; total admissions 2002 trauma cases with an ISS ≥ 16 = 162).
- St Vincent’s Hospital, Darlinghurst (emergency department attendances 1999/2000 = 32 901; Total admissions 2002 trauma cases with an ISS ≥ 16 = 123).
- Westmead Hospital, Westmead (emergency department attendances 1999/2000 = 37 856; total admissions 2002 trauma cases with an ISS ≥ 16 = 212).
- Sydney Children’s Hospital, Randwick (emergency department attendances 1999/2000 = 27 290; total admissions 2002 trauma cases with an ISS ≥ 16 = 31).
Profiles of two hospitals not included into the study:

- John Hunter Hospital, Newcastle (emergency department attendances 1999/2000 = 45,853; total admissions 2002 trauma cases with an ISS ≥ 16 = 256).
- Royal North Shore Hospital, North Sydney (emergency department attendances 1999/2000 = 40,888; total admissions 2002 trauma cases with an ISS ≥ 16 = 230).

The John Hunter Hospital and Royal North Shore Hospital accounted for 27% of all area trauma hospital emergency department admissions during 2002. The number of persons with traumatic head injury in the 27% is unknown and could not be found in the literature. However during this time the pre-hospital management of severe traumatic head injury remained the same throughout the Sydney and metropolitan areas.

The total trauma cases in with an Injury Severity Score ≥ 16 for all area trauma hospitals (n = 12) (inclusive of John Hunter Hospital and Royal North Shore Hospital) in 2002 was 1,809. This number reflects the State-wide total and includes all direct admissions and also patients transferred in from another hospital in NSW. Trauma cases with an Injury Severity Score ≥ 16 totalled 1,323 in 2002 amongst the 10 area trauma hospitals who participated in this study which represents 73% of total trauma cases in the Sydney and
Metropolitan area. However it should be noted that the total represents all trauma types and all body regions. The breakdown of numbers within each trauma type or number of severe brain trauma cases was not presented in the literature (New South Wales Health, 2001; New South Wales Health, 2002). The Victorian Government Department of Human Services (2004) estimates severe head injury to be 25.6% of all major trauma cases in 2002 to 2003. Extrapolation of this percentage to NSW statistics suggests of the 1,323 trauma cases approximately 338 may likely be cases of severe brain trauma. It should be noted that this figure is speculative as the exact number of severe head injury cases was not found in the literature.

Nine of the area trauma hospitals included in this study are located within the Greater Sydney region and one within the Newcastle region. Two maps showing the location of the area trauma hospitals within the State of NSW and the location of the hospitals within the Greater Sydney region are presented in Figure 2 and Figure 3. The location of Prince of Wales Hospital is not included in Figure 3. The Prince of Wales Hospital is located immediately next to Sydney Children’s Hospital in the suburb of Randwick (refer to hospital number 5 in Figure 3).
Figure 2 Location of Area Trauma Hospitals in NSW

(New South Wales Government, 2012)
Figure 3  Location of Area Trauma Hospitals within the Greater Sydney Region

Metropolitan Major Trauma Services:
1. Children's Hospital at Westmead
2. Westmead Hospital
3. Royal North Shore Hospital
4. St Vincent's Hospital
5. Sydney Children's Hospital
6. Royal Prince Alfred Hospital
7. St George Hospital
8. Liverpool Hospital

Statewide Speciality Services:
Severe Burn Injury Service -
Royal North Shore Hospital
Concord Hospital
Children's Hospital at Westmead

Spinal Cord Injury Service -
Royal North Shore Hospital
Prince of Wales Hospital
Children's Hospital at Westmead
Sydney Children's Hospital

(New South Wales Government, 2012)
3.3.3 Sample Frame for the Study

Analysis of Ambulance Service of NSW data over the 5-year period from 1995 to 2000 indicated that a three year span was required to enlist an adequate number of patients with severe traumatic head injuries into the study. Ambulance Service of NSW data were used to calculate the number of patients with severe traumatic head injuries that were treated and transported each year from 1995 to 2000. A total of 3,658 trauma cases transported by the Ambulance Service of NSW in the Sydney and metropolitan area were scrutinised, of which 417 met the inclusion criteria during the five year period.

To obtain a sample of over 300 persons with severe traumatic head injury, it was calculated that access to data from the Ambulance Service of NSW and at least nine area trauma hospitals would be required. John Hunter Children’s Hospital was included in the study to increase the number of paediatric patients within the cohort. A total of 10 area trauma hospitals were enlisted in the study.

The enlistment time frame set for the study was from 1 January 1999 to 31 December 2001 to ensure a full three year enlistment period. Subjects suitable for inclusion were identified from the Ambulance Service of NSW patient database (GCS < 9, protocol 34 Head Injury, trauma triage record) and hospital trauma registry using Glasgow Coma Score, mode of arrival to hospital, head injury and ICD 10 codes as identifying criteria. Following identification of suitable subjects, the medical records of those patients were reviewed. Patient identification was required for the purpose of matching Ambulance Service of NSW documentation and hospital records. Following matching, a numerical
identifier was allocated to each case and all reference to patient identity was destroyed by shredding. All persons were de-identified and their identity remained hidden.

All times for pre-hospital and in-hospital interventions, observations and administration of pharmacological agents were recorded. The first and last set of vital signs were recorded in each setting or department during the pre-hospital and in-hospital phase of patient care (pre-hospital, emergency department, surgery, intensive care unit, general ward) until discharge from the hospital. Data collection was undertaken from April 2003 to December 2005.

A total of 789 medical records were reviewed to verify appropriateness for inclusion into the study. Records were reviewed at a total of 10 area trauma hospitals within the Sydney and metropolitan areas. Area trauma hospitals were selected for data collection as any persons with severe traumatic head injury requiring surgical intervention or time in an intensive care unit would be transported directly to an area trauma hospital or later transferred from a district hospital to an area trauma hospital. The final sample size consisted of 326 patients with severe traumatic head injury who meet the inclusion criteria of the study. The sample included all age groups (age range 3 months to 99 years) and both genders, with males making up a larger proportion of the sample (68.7%) than females (31.3%).

An average of 39,431 trauma related cases were transported by the Ambulance Service of NSW per annum during the 1995 to 2000 period. Of those 784 were severe cases that
were transported to an area trauma centre (New South Wales Department of Health, 2004). It is estimated that 7.1% of all injury related hospital separations in Australia during 1997 and 1998 were attributable to survivors of severe head trauma (O’Connor, 2002). Overall mortality associated with severe traumatic head injury in Australia during 2004 to 2005 was estimated at 4.3% (Helps, Henley & Harrison, 2008). The overall rate of severe traumatic head injury is therefore estimated at 11.4% of all trauma cases. Thus the incidence of traumatic head injury in the Sydney and metropolitan area during 1995 to 2004 could be estimated at 90 cases per annum (270 cases per three year period). It should be noted that these are estimates and no direct measure of severity of brain injury was recorded in hospital separations data (O’Connor, 2002). Khan, Baguley and Cameron (2003) warn that actual cases of severe head trauma may be considerably under reported due to errors in data collection, diagnostic errors, errors in classification, or traumatic head injury as an additional rather than a principle diagnosis. The sample in the current study ($n = 326$) could therefore be considered representative of the total population of severely head-injured patients in the Sydney and metropolitan areas during 1999 to 2001.

3.4 Quantitative Data Collection and Analysis

3.4.1 Development of the Data Collection Form

A data collection form was developed with reference to previous research in the area of traumatic brain injury (see Chapter Two), the Ambulance Service of NSW (1997) patient care records, and medical records of the NSW hospital system. The scope of data collected for each patient included all times, treatments/interventions and pharmacology
administered from the pre-hospital phase of management to discharge from the area trauma hospital. Outcome data was also collected including final Glasgow Coma Scale and data relating to Glasgow Outcome Scale, Westmead Post-Traumatic Amnesia Scale and activities of daily living. The data collection form incorporated the following categories (refer to Appendix 1, Data Collection Form):

- **Pre-hospital information**
  - Patient information
  - Factors relating to the circumstances of the injury
  - Mechanism of injury
  - Location and/or place of injury
  - Pre-hospital patient assessment and observation information
  - Pre-hospital patient management
  - Pre-hospital pharmacological interventions
  - Pre-hospital care factors

- **Emergency department assessment and interventions**
  - Emergency department patient assessment and observations
  - Diagnostic testing

- **Operating theatres**
  - Surgeon’s report
  - Patient notes and observations

- **Intensive care unit and general ward**
  - ICU and general ward notes and observations
  - Significant interventions
- Other interventions
- Hospital pharmacological interventions
- Outcome details

• Other
  - Mortality information
  - Adverse events
  - Activities of daily living
  - Glasgow Coma Score and outcome details (Glasgow Outcome Score, Disability Rating Scale and Westmead Post-Traumatic Amnesia Scale)

Data was retrospectively collected from Ambulance Service of NSW patient care records and medical records of patients with severe traumatic head injury. All data collection was undertaken within the medical records departments of the area trauma hospitals in the Sydney and metropolitan regions. Data was transcribed from the medical record onto the data collection forms. Patients were de-identified and no personal information was transcribed onto the data collection forms. Data collected onto the data collection forms was entered into a Statistical Packages for the Social Sciences (SPSS) data base.

3.4.2 Pre-Test and Resolution

A pre-test of the data collection tool was undertaken and it was found that medical records were not complete or were still in use within the hospital for many persons injured during 2002, therefore preventing access to these patients. It was decided to set the data collection period from 1 January 1999 to 31 December 2001 for the researcher to
have access to the complete medical record and to be able to access all data from the pre-hospital phase to discharge from the area trauma hospital.

Data collection was undertaken on a hospital by hospital basis in the following order: Prince of Wales Hospital, Sydney Children’s Hospital, Royal Prince Alfred Hospital, St George Hospital, St Vincent’s Hospital, Westmead Hospital, Westmead Children’s Hospital, Liverpool Hospital, John Hunter Children’s Hospital and Nepean Hospital. Data collection commenced September 2002 and was completed in 2005. Data analysis commenced in 2006 was completed in 2009. Ambulance Service of NSW treatment protocols for persons with severe traumatic head injury have remained unchanged during the time of data collection and analysis.

3.4.3 Pilot Study of the Data Collection Form

Fifty-two (52) cases of severe traumatic head injury were reviewed for the pilot study. The Ambulance Service of NSW data base contained all cases treated and transported during 1998 (35,973 cases). Trauma cases identified totalled 3,658 and included all forms of trauma and all injury severity. Only a total of 58 patients meet the study inclusion criteria within the Sydney and metropolitan area. It was therefore concluded that a cohort of 300 patients over a three year period would be reflective of the total population of severe brain trauma patients and therefore reduce selection bias. It would also augment population validity.
A pilot study was undertaken to validate the data collection tool content and length. Pilot test analysis disclosed valuable data items that were not incorporated into the pilot data collection form. These data items included Injury Severity Score, Westmead Post-Traumatic Scale, Activities of Daily Living Assessment and airway status on arrival at the emergency department. Data collection relating to co-morbid conditions was also expanded as a result of pilot study analysis. The data collection tool was subsequently modified and expanded post-pilot study analysis to include additional data to be collected during the main study (refer to Appendix 1, Data Collection Form). Preliminary analysis using Statistical Packages for the Social Sciences (SPSS) was undertaken to validate the use of descriptive, cross tabulation, correlation and stepwise liner regression analysis methods used in the study.

3.4.4 Statistical Analysis

The statistical analysis software utilised in this study was Statistical Packages for Social Sciences (SPSS) versions 14 to 18. Descriptive analysis utilised cross tabulation, frequency, range, percent, mean and standard deviation. Descriptive analysis was used to report measures of central tendency and variability and to interpret to results of correlation and regression analysis. Correlation analysis applied included bivariate analysis using 2-tailed Spearman’s rho for categorical variables and 2-tailed Pearson’s correlation for continuous variables. Independent samples t-test was utilised to compare means between groups (2-group analysis). All p-values were set at the p < 0.05 level to be considered statistically significant.
In order to test Research Hypotheses 1 to 4, data were analysed using stepwise linear regression analysis with confidence interval (CI) set at 95% for the regression coefficient. The outcome measure of interest in this analysis was the Glasgow Outcome Scale (GOS). Each patient was assigned a GOS category at discharge from the area trauma hospital. The GOS category was assigned by the researcher from review of medical record entries at the time of discharge. Parameters gained from medical records used to determine the GOS category were Glasgow Coma Scale, activities of daily living, residual disability and discharge destination (for example home, other hospital, brain injury unit, nursing home/high dependency care facility). Following review of medical records, each patient was categorised into 1 of 5 categories: dead, persistent vegetative state, severe disability, moderate disability or good recovery.

Figure 1 provides a conceptual model of the dependent and independent variables in this study. Glasgow Outcome Scale was used as the dependent variable in stepwise linear regression analysis to determine the regression coefficient for neurological outcome. The independent variables were airway maintenance and oxygenation, fluid resuscitation and correction of hypotension, time in the pre-hospital setting, hospital destination and paramedic level of clinical skill.

Regression and correlation coefficients of less than 0.1 were not considered to be representative of a significant relationship. Regression and correlation coefficients of between 0.1 and 0.2 with a p-value of less than or equal to 0.05 were considered weak but significant linear relationship. Regression and correlation coefficients of 0.2 or greater
with a p-value of less than or equal to 0.05 were considered to represent a small to very strong linear relationship, with 0.2 representing a small relationship, 0.3 to 0.5 a moderate relationship, 0.6 to 0.7 representing a strong relationship and > 0.8 representing an extremely strong relationship (Billittier et al., 1998; Francis, 2007; Urdan, 2010). The importance of the regression coefficient was investigated through descriptive analysis using cross tabulation, mean and standard deviation, frequency, range and percent. The findings were compared with current and previous literature to establish content validity.

Stepwise regression was selected in preference to standard or hierarchical regression due to the method of entry of independent variables being forwards, backwards and a combination of both. The stepwise procedure permits entry of predictors at one time in the forward method. The acceptance of a predictor is determined by the F-test and critical alpha level. Backward selection then deletes weak variables based on F-value. Stepwise procedure also allows for the later removal of variables previously entered (Francis, 2007, Coakes & Ong, 2011; Urdan, 2010).

3.5 Ethics Considerations

This study received approval from the following human research ethics committees with approval numbers listed:

- Charles Sturt University Human Research and Ethics Committee (98/108)
- South East Sydney Area Health Service Research Ethics Committee (00/079)
- Central Sydney Area Health Service Ethics Review Committee (X01-0153)
• Western Sydney Area Health Service Human Research Ethics Committee HREC2001/10/4.5(1318)

• South Western Sydney Area Health Service Research Ethics Committee (01-84 Webb)

• Wentworth Area Health Service Human Ethics and Research Committee (2001/066)

• Hunter Area Research Ethics Committee (01/11/14/3.13)

• Health and Research Employee’s Association (MW:PM:VV/A026462).

A master list of patient names and medical record numbers was kept in a separate locked cabinet for the duration of data collection. At no time was the master list stored with data collection forms. The master list was shredded at the completion of the data analysis phase. Patients were de-identified and a unique four digit identifier was assigned to each patient. All records were viewed within the Medical Records Departments at each hospital. Data was transcribed onto the data collection forms. Medical records were not photocopied or reproduced in any way. Patient names and personal details were not recorded onto the data collection forms.
4.1 Characteristics of the Cohort

The final cohort sample size consisted of 326 patients with severe traumatic head injury who met the inclusion criteria of the study. All patients were treated and/or transported to hospital by paramedics employed by the Ambulance Service of NSW. The sample included all age groups and both genders. Age and gender distributions of the sample are presented in Table 9 and Table 10.

Table 9  
Baseline Characteristics of the Patients

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Subgroup</th>
<th>Number and Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>Male</td>
<td>224 (68.7%)</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>102 (31.3%)</td>
</tr>
<tr>
<td>Age</td>
<td>&lt; 15 years</td>
<td>49 (15%)</td>
</tr>
<tr>
<td></td>
<td>16 – 35 years</td>
<td>150 (46%)</td>
</tr>
<tr>
<td></td>
<td>36 – 55 years</td>
<td>60 (18.4%)</td>
</tr>
<tr>
<td></td>
<td>&gt; 55 years</td>
<td>67 (20.6%)</td>
</tr>
<tr>
<td>Age Range</td>
<td>3 months – 99 years</td>
<td>326 (100%)</td>
</tr>
</tbody>
</table>
Mean age was found to be 35 years, however the greater number of cases were distributed between the age range from 17 years to 27 years (35.8%). The highest percentages of patients were in the age brackets of 18 years (5.5%) and 19 years (4.9%).

Cause of injury was distributed into 8 categories. The distributions by cause of injury are presented in Table 11.
### Table 11  
**Cause of Injury**

<table>
<thead>
<tr>
<th>Cause of Injury</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor Vehicle</td>
<td>81 (24.9%)</td>
</tr>
<tr>
<td>Motor Cyclist</td>
<td>17 (5.2%)</td>
</tr>
<tr>
<td>Cyclist</td>
<td>6 (1.8%)</td>
</tr>
<tr>
<td>Train</td>
<td>6 (1.8%)</td>
</tr>
<tr>
<td>Horse/Animal</td>
<td>2 (0.6%)</td>
</tr>
<tr>
<td>Other Transport</td>
<td>7 (2.2%)</td>
</tr>
<tr>
<td>Pedestrian</td>
<td>85 (26.0%)</td>
</tr>
<tr>
<td>Non-Transport</td>
<td>40 (12.3%)</td>
</tr>
<tr>
<td>Fall</td>
<td>82 (25.2%)</td>
</tr>
<tr>
<td>Total</td>
<td>326 (100%)</td>
</tr>
</tbody>
</table>

Types of transportation in the category of ‘other transport’ included skate-board, push scooter, jet-ski and power boat. Non-transport incidents included assaults, deliberate injury and suicide. Falls were the causation of injury in 25.2% of the total sample. Landing surfaces in falls included concrete ($n = 50, 61.0\%$ of falls), floor of building/house ($n = 13, 15.9\%$ of falls), post/guard rail ($n = 2, 2.4\%$ of falls), ground (grass, dirt) ($n = 5, 6.1\%$ of falls), water ($n = 1, 1.2\%$ of falls) and other surfaces ($n = 11, ...
13.4% of falls). In 1 of the 11 ‘other surfaces’ cases, the patient received their injuries from a tree branch falling 20 metres from a tree and striking the patient on the head. Distance of falls and frequency are presented in Table 12.

Table 12  
Falls – Distance and Frequency of Falls

<table>
<thead>
<tr>
<th>Distance of Fall</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3 metres</td>
<td>51 (62.2%)</td>
</tr>
<tr>
<td>3-10 metres</td>
<td>28 (34.1%)</td>
</tr>
<tr>
<td>&gt; 10 metres</td>
<td>3 (3.7%)</td>
</tr>
<tr>
<td>Total</td>
<td>82 (100%)</td>
</tr>
</tbody>
</table>

A total of 7 cases of entrapment reported the patient was trapped by a compressive force (trapped with compression). Following review of medical records it was found that no patient had received injuries associated compressive force. The valid number of entrapment with compression was zero and the cases of entrapment by confinement were 47.

4.2 Representativeness of the Cohort

The cohort was representative of the population in age and gender when compared with other Australian studies (Tate et al., 1998; Lyle et al., 1990). The majority of patients were young, previously healthy adults, with males out-numbering females by a ratio of
2:1; however, the reasons for this gender difference have not been explored in this study. The majority of patients were between the ages of 17 years and 27 years (35.5%) and the highest percentages of severe traumatic brain injury were found in the 18 and 19-year-old groups, 5.5% and 4.9% respectively. Mean age of the cohort was 35 years of age.

Causation of injury was also found to compare with the study by Hillier, Hillier and Metzer (1997). Details of the Hillier et al. study and the current study are outlined in Table 13.

### Table 13  Cause of Injury – Comparison of Hillier and Webb Studies

<table>
<thead>
<tr>
<th>Cause of Injury</th>
<th>Hillier et al. (1997)</th>
<th>Webb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transport: Motor Vehicle</td>
<td>46 (26.0%)</td>
<td>81 (24.9%)</td>
</tr>
<tr>
<td>Transport: Motorcyclist</td>
<td>12 (6.8%)</td>
<td>17 (5.2%)</td>
</tr>
<tr>
<td>Transport: Cyclist, Train, Horse/Animal, Other</td>
<td>Not Specified</td>
<td>21 (6.4%)</td>
</tr>
<tr>
<td>Non-Transport: Assault, Other</td>
<td>25 (14.2%)</td>
<td>40 (12.3%)</td>
</tr>
<tr>
<td>Pedestrian</td>
<td>12 (6.8%)</td>
<td>85 (26.0%)</td>
</tr>
<tr>
<td>Falls</td>
<td>52 (29.3%)</td>
<td>82 (25.2%)</td>
</tr>
<tr>
<td>Other</td>
<td>30 (16.9%)</td>
<td>N/A</td>
</tr>
<tr>
<td>Total</td>
<td>177 (100%)</td>
<td>326 (100%)</td>
</tr>
</tbody>
</table>
The greatest variation between the two studies was seen in the pedestrian category with almost 4 times as many pedestrians noted in the current (Webb) study. The study locations may be contributing factors in this discrepancy with the Hillier study undertaken in Adelaide, South Australia and the Webb study in Sydney, New South Wales. However, verification of this assumption is not within the scope of this study.

4.3 Glasgow Coma Scale and Inter-Rater Reliability – Findings from the Study

A statistically significant correlation was found between the final pre-hospital GCS and the first emergency department GCS (Spearman’s rho 0.802, p < 0.001). Significant correlations were found between all components of the final pre-hospital GCS and first emergency department GCS; eye opening (Spearman’s rho = 0.432, p < 0.001), verbal response (Spearman’s rho = 0.530, p < 0.001) and motor score (Spearman’s rho = 0.746, p < 0.001). The significant correlation between pre-hospital and emergency department GCS demonstrates good inter-rater reliability between assignment of the GCS by pre-hospital and emergency department raters in the Sydney and metropolitan areas. Results suggest paramedics were able to assign a GCS with a high degree of reliability.
4.4 Outcome of the Cohort – Glasgow Outcome Scale

The final Glasgow Outcome Score for 323 patients is presented in Table 14. The final outcome is uncertain for three patients due to incomplete documentation.

Table 14  Final Outcome for 323 Patients – Glasgow Outcome Scale

<table>
<thead>
<tr>
<th>GOS</th>
<th>Death</th>
<th>Vegetative State</th>
<th>Severe Disability</th>
<th>Moderate Disability</th>
<th>Good Recovery</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>137</td>
<td>15</td>
<td>62</td>
<td>29</td>
<td>80</td>
<td>323</td>
</tr>
<tr>
<td>Percent</td>
<td>42.4%</td>
<td>4.6%</td>
<td>19.2%</td>
<td>9.0%</td>
<td>24.8%</td>
<td>100%</td>
</tr>
</tbody>
</table>
CHAPTER FIVE

THE ATTENDANCE OF INTENSIVE CARE PARAMEDICS AND ENDOTRACHEAL INTUBATION

5.1 Overview of the Chapter

Chapter Five will present the findings for research question 1; persons with severe traumatic head injury will benefit from the attendance of intensive care paramedics and pre-hospital endotracheal intubation provided by intensive care paramedics. This chapter will present results and discussion relating to the attendance of intensive care paramedics and the results and discussion specifically relating to the intervention of endotracheal intubation. The above listed variables and their relationship to the depended variable (GOS) will be discussed (refer to Figure 1).

5.2 Intensive Care Paramedics

5.2.1 Results – Intensive Care Paramedics

The majority of cases had at least one intensive care paramedic (level 5) responder in attendance (75.2%, n = 245). It is unknown if intensive care paramedics were in attendance in 8.6% of cases (n = 28). Non-intensive care paramedic attendance only was documented in 16.3% of cases (n = 53). Two-crew (dual) response was noted in 91.4% of cases (n = 298 cases). Level 5 paramedics were the first responders in 40.8% of cases (n = 133) and second responders in 37.4% of cases (n = 122). In 3.4% of cases (n = 11) there was a dual intensive care paramedic response with level 5 as both first and second responders. A medical officer was present at the scene in 8.6% (n = 28) of cases.
Correlation and regression analysis were used to determine if level of clinical skill, intensive care paramedic or a medical officer at the scene were predictors of outcome (GOS). Results of correlation analysis are presented in Table 15.

Table 15  Correlation Analysis - Level of Clinical Skill and GOS

<table>
<thead>
<tr>
<th>Level of Clinical Skill</th>
<th>Spearman Correlation</th>
<th>p Value (2-Tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intensive Care Paramedic Level 5</td>
<td>0.031</td>
<td>0.600</td>
</tr>
<tr>
<td>Level of 1st Responder</td>
<td>−0.016</td>
<td>0.790</td>
</tr>
<tr>
<td>Level of 2nd Responder</td>
<td>0.008</td>
<td>0.888</td>
</tr>
<tr>
<td>Medical Officer at Scene</td>
<td>−0.038</td>
<td>0.502</td>
</tr>
</tbody>
</table>

Stepwise linear regression showed no significant correlations for the level of clinical response ($r = 0.003$, $p = 0.952$), the attendance of intensive care paramedics ($r = −0.005$, $p = 0.931$) or medical officers ($r = −0.073$, $p = 0.187$) at the scene and the Glasgow Outcome score of the patients.

Correlation analysis found that GCS was not a significant predictor of the level first response (Spearman’s rho $r = −0.081$, $p = 0.164$, 2-tailed). However, GCS was a significant predictor of the level of second response (Spearman’s rho $r = 0.158$, $p = 0.006$, 2-tailed). Intensive care paramedics were dispatched as second responders in 70.1% of cases where the level of second response could be determined ($n = 122$ of 174). The presence of intensive care paramedics was also associated with the undertaking of intravenous cannulation (Spearman’s rho $r = 0.494$, $p < 0.001$, 2-tailed) and the
administration of fluid resuscitation (Spearman’s rho r = 0.338, p < 0.001, 2-tailed). However fluid resuscitation was not a significant predictor of improved outcome (GOS) (Spearman’s rho r = 0.062, p = 0.267, 2-tailed) and nor was the attendance of intensive care paramedics (Spearman’s rho r = 0.031, p = 0.600, 2-tailed). It should be noted that the response time of the second responders (from the time of the incident to the arrival to the second response crew) was not available in the medical records and therefore could not be determined.

Time at scene was significantly increased with the attendance of intensive care paramedics (Spearman’s rho = −0.228, p < 0.001, 2-tailed). The presence or absence of intensive care paramedics and time at scene could be determined in 293 of the 326 cases (89.9%). Time at scene for intensive care paramedics and non-intensive care paramedics are outlined in Table 16.

### Table 16  Time at Scene – Intensive Care and Non-Intensive Care Paramedics

<table>
<thead>
<tr>
<th>Level of Clinical Skill</th>
<th>Number</th>
<th>Minimum time (mins)</th>
<th>Maximum time (mins)</th>
<th>Mean Time at Scene (mins)</th>
<th>Standard Deviation (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intensive Care Paramedic at Scene</td>
<td>242</td>
<td>3</td>
<td>77</td>
<td>22.11</td>
<td>11.891</td>
</tr>
<tr>
<td>Non-Intensive Care Paramedic at Scene</td>
<td>51</td>
<td>3</td>
<td>58</td>
<td>15.90</td>
<td>9.345</td>
</tr>
</tbody>
</table>

Mean scene times were greater when intensive care paramedics were in attendance: 22.1 minutes versus 15.9 minutes for non-intensive care paramedics (independent samples t-test, p < 0.001). Intensive care paramedics had a mean scene time of 6.2 minutes greater
than non-intensive care paramedics. Cases of entrapment \( (n = 42) \) were then removed from the analysis. In the absence of cases of entrapment, intensive care paramedics had a mean scene time of 4.8 minutes greater than non-intensive care paramedics \( (p = 0.001) \). Time at scene in cases without entrapment is presented in Table 17.

<table>
<thead>
<tr>
<th>Level of Clinical Skill</th>
<th>Number</th>
<th>Minimum time (mins)</th>
<th>Maximum time (mins)</th>
<th>Mean Time at Scene (mins)</th>
<th>Standard Deviation (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intensive Care Paramedic at Scene Nil Entrapment</td>
<td>202</td>
<td>3</td>
<td>59</td>
<td>20.50</td>
<td>10.932</td>
</tr>
<tr>
<td>Non-Intensive Care Paramedic at Scene Nil Entrapment</td>
<td>49</td>
<td>3</td>
<td>58</td>
<td>15.69</td>
<td>9.470</td>
</tr>
</tbody>
</table>

Mean scene time for all cases was 21.3 minutes (Std Deviation = 12.0 minutes). In two cases, time at scene exceeded 60 minutes. In both cases, the patient was stated to be trapped within a motor vehicle. For cases where entrapment was not a factor that delayed transportation, both intensive care paramedics and non-intensive care paramedics had a very similar minimum and maximum time on scene. The minimum scene time for both intensive care paramedics and non-intensive care paramedics was three minutes. The maximum time at scene was very similar for both groups; 59 minutes for intensive care paramedics and 58 minutes for non-intensive care paramedics. However, mean time at scene was significantly higher for intensive care paramedics \( (t\text{-test}, p = 0.001) \) (refer to Table 17).
Scene times of either greater than 10 minutes (> 10 mins) or less than or equal to 10 minutes (≤ 10 mins) were analysed for 293 cases. Intensive care paramedics were in attendance at the scene in 242 of the 293 cases (82.6%) and achieved a scene time of less than or equal to 10 minutes in 35 of the 242 cases (14.5%). In the remaining 85.5% of cases where intensive care paramedics were in attendance, time at scene was greater than 10 minutes. Comparatively, non-intensive care paramedics achieved scene times less than or equal to 10 minutes in 34.0% of cases as opposed to only 14.5% for intensive care paramedics. Scene times less than or equal to 10 minutes and scene times greater than 10 minutes for intensive care paramedic response and non-intensive care paramedic response are presented in Table 18.

**Table 18** Time at Scene Less Than or Equal to 10 Minutes and Greater Than 10 Minutes

<table>
<thead>
<tr>
<th>Time at Scene</th>
<th>Intensive Care Paramedic</th>
<th>Non-Intensive Care Paramedic</th>
<th>Total</th>
<th>t-test p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 10 mins</td>
<td>208 (71.0%)</td>
<td>33 (11.3%)</td>
<td>241 (82.3%)</td>
<td>p = 0.001</td>
</tr>
<tr>
<td>≤ 10 mins</td>
<td>35 (12.0%)</td>
<td>17 (5.8%)</td>
<td>52 (17.7%)</td>
<td>p = 0.001</td>
</tr>
<tr>
<td>Total</td>
<td>243 (82.9%)</td>
<td>50 (17.1%)</td>
<td>293 (100%)</td>
<td></td>
</tr>
</tbody>
</table>

Stepwise linear regression showed the presence of intensive care paramedics at the scene was a significant predictor of scene time greater than or equal to 10 minutes (r = 0.198,
p = 0.001, 95% CI = 0.087 to 0.319). The attendance of intensive care paramedics and scene time greater than 10 minutes was analysed using correlation analysis and was found to be significant (Spearman’s rho = 0.198, p = 0.001, 2-tailed).

5.2.2 Discussion – Intensive Care Paramedics

The ability of paramedics to effectively triage trauma patients is thought to be a complex relationship between clinical experience, repeated clinical assessment of trauma patients and feedback from trauma experts. Therefore it was hypothesised that paramedics with more experience and a higher level of clinical skill would be more adept at recognising serious traumatic injury which may require surgical intervention. It was postulated that more experienced paramedics would therefore limit scene time to less than 10 minutes, maintain a patent airway and oxygen saturations of greater than or equal to 95% and transport the patients directly to an area trauma centre. Such management and clinical decision making would improve patient outcome by maintaining oxygen saturation and reducing time to definitive medical intervention.

Studies by Rainer et al. (1997), Dimetriades et al. (1996) and Regal et al. (1997) found that advanced life support interventions such as intravenous access, fluid administration and endotracheal intubation were not significantly associated with improved outcome in trauma patients. Regal et al. also suggest that advanced pre-hospital interventions were not recommended in patients where time to hospital was less than 10 minutes, the patient had a patent airway and systolic blood pressure was 90 mmHg or greater. In all studies reviewed, advanced life support interventions were positively associated with increased
time at scene. The current study found that in 62% of cases, transportation time to hospital was less than or equal to 10 minutes. No relationship between the undertaking or withholding of advanced life support interventions and proximity to hospital was established in this study.

5.2.3 Summary – Intensive Care Paramedics

Analysis did not establish that persons with severe traumatic head injury demonstrated improved outcome (GOS 4 or 5) following pre-hospital endotracheal intubation provided by intensive care paramedics. A weak but significant correlation was found between the presence of intensive care paramedics and increased time at scene. Increased scene time, and scene time greater than 10 minutes was a significant predictor of poor outcome (Spearman’s rho = −0.140, p = 0.016, 2-tailed). As the correlation between the presence of intensive care paramedics and increased scene time was significant but weak, further investigation is required. The efficacy of advanced pre-hospital interventions, the presence of intensive care paramedics, dual crew response and non-intensive care paramedics waiting at the scene for intensive care paramedics to take control of patient management remain unclear. However as the findings of this research and the findings of Rainer et al. (1997), Dimetriades et al. (1996) and Regal et al. (1997) failed to demonstrate improved outcomes with pre-hospital advanced life support interventions, consideration should therefore be given to not delaying time at scene for endotracheal intubation if the airway can be adequately managed without endotracheal intubation.
5.3 Endotracheal Intubation and Patient Outcome Following Severe Traumatic Head Injury

5.3.1 Results – Pre-Hospital Endotracheal Intubation

Analysis demonstrated a significant correlation between pre-hospital endotracheal intubation and a poorer GOS (Spearman’s rho = −0.444, p < 0.001, 2-tailed) (refer to Table 17 for percentage analysis for intubated and non-intubated patients). Stepwise linear regression revealed pre-hospital endotracheal intubation as a significant predictor of poorer outcome (r = −0.433, p < 0.001, 95% CI = 1.011 to 1.858). The majority of the 65 patients who received endotracheal intubation in the pre-hospital setting had a GCS of 3 to 5 (90.1%, n = 59). Regression analysis found a low pre-hospital GCS to be a predictor of endotracheal intubation (r = 0.292, p < 0.001, 95% CI = 1.186 to 2.523), indicating that the lower the GCS, the more likely a patient will receive endotracheal intubation. A significant correlation was also found between high Injury Severity Score and low pre-hospital GCS (r = −0.226, p < 0.001, 95% CI = −1.674 to −0.546). High Injury Severity Score was found to be a significant predictor of pre-hospital endotracheal intubation (r = −0.182, p = 0.001, 95% CI = −9.604 to −2.349). Analysis also failed to demonstrate improved outcome for age range and pre-hospital intubation (Spearman’s rho r = 0.018, p = 0.749, 2-tailed) and gender and pre-hospital intubation (Spearman’s rho r = 0.037, p = 0.511, 2-tailed).

For those patients who received pre-hospital intubation, 95.3% had a poor outcome (GOS 1, 2 or 3) versus 59.1% for those who were intubated in hospital (p < 0.001). Improved outcomes were noted in 4.6% of those who were intubated in the pre-hospital setting (GOS 4 or 5) and 40.9% of those intubated within the in-hospital setting (p < 0.001). A
high mortality rate was also noted in patients who received pre-hospital endotracheal intubation (75.4%) compared with a much lower mortality rate in those who were intubated in the emergency department (34.2%) (Spearman’s rho r = 0.338, p < 0.001, 2-tailed). A very weak correlation was established between ISS and pre-hospital intubation (Spearman’s rho = -0.173, p = 0.002) indicating that the more severely injured the patient the more likely they will receive endotracheal intubation in the pre-hospital setting. This effect is likely due to the correlation between low initial pre-hospital GCS and high injury severity scores (Spearman’s rho r = -0.242, p < 0.001). However the weak correlation between ISS and pre-hospital endotracheal intubation suggests that injury severity alone is not a strong determinant of poorer outcome when associated with pre-hospital endotracheal intubation. The Glasgow Outcome Score of patients intubated in the pre-hospital setting is presented in Table 19.

Table 19  Endotracheal Intubation and Glasgow Outcome Scale

<table>
<thead>
<tr>
<th></th>
<th>Death</th>
<th>Vegetative State</th>
<th>Severe Disability</th>
<th>Moderate Disability</th>
<th>Good Recovery</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pre-hospital ETI - Yes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>49 (75.4%)</td>
<td>2 (3.0%)</td>
<td>11 (16.9%)</td>
<td>1 (1.5%)</td>
<td>2 (3.1%)</td>
<td>65 (100.0%)</td>
</tr>
<tr>
<td><strong>Pre-hospital ETI - No</strong></td>
<td>88 (34.2%)</td>
<td>13 (5.1%)</td>
<td>51 (19.8%)</td>
<td>28 (10.9%)</td>
<td>77 (30.0%)</td>
<td>257 (100.0%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>137 (42.6%)</td>
<td>15 (4.7%)</td>
<td>62 (19.3%)</td>
<td>29 (9.0%)</td>
<td>79 (24.5%)</td>
<td>322 (100%)</td>
</tr>
</tbody>
</table>

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Pre-hospital endotracheal intubation was also found to correlate with increased scene time (Spearman’s rho = −0.186, p = 0.001, 2-tailed). Stepwise linear regression found pre-hospital intubation was a significant predictor of increased time at scene (r = −0.192, p = 0.001, 95% CI = −0.010 to −0.003). Further, time to endotracheal intubation, including both pre-hospital and in-hospital intubation, was not significant in the prediction of Glasgow Outcome score in persons with severe traumatic head injury (r = 0.059, p = 0.319).

No significant correlation was found between pre-hospital endotracheal intubation and the first pulse oximetry reading on arrival at the emergency department (r = −0.065, p = 0.276) (refer to Table 18 for percentage analysis). A total of 18.8% of patients intubated in the pre-hospital setting were hypoxic on arrival at the emergency department (n = 9) (SpO2 < 95%) compared with 21.2% of those who were not intubated in the pre-hospital setting (n = 49) however these results were not statistically significant (Spearman’s rho r = 0.019, p = 0.752, 2-tailed). Those patients who were intubated in the pre-hospital setting were found to have higher incidence of moderate to severe hypoxic episodes (SpO2 < 90% & SpO2 < 85%); 14.6% (n = 7) for those receiving pre-hospital intubation and 10.8% (n = 25) for those not receiving pre-hospital intubation (Spearman’s rho r = 0.054, p = 0.373, 2-tailed). Fewer incidences of mild hypoxaemia (SpO2 90% to 94%) were found in the pre-hospital intubation group (4.2%) versus those not intubated in the pre-hospital setting (10.4%) but analysis failed to demonstrate a significant correlation (Spearman’s rho r = −0.078, p = 0.225, 2-tailed). The differences between those receiving pre-hospital intubation and those not receiving pre-hospital intubation on
the first emergency department SpO2 were not significant (Spearman’s rho $r = -0.12$, $p = 0.937$, 2-tailed). The total percentage of patients found to be hypoxic on arrival at the emergency department was 20.8% ($n = 58$). The first emergency department pulse oximetry reading and pre-hospital intubation results are presented in Table 20.

**Table 20**  
Pre-Hospital Endotracheal Intubation (ETI) and First Emergency Department Pulse Oximetry Reading (ED SpO2)

<table>
<thead>
<tr>
<th></th>
<th>ED SpO2 &lt; 85%</th>
<th>ED SpO2 85-89%</th>
<th>ED SpO2 90-94%</th>
<th>ED SpO2 95-100%</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pre-hospital ETI -</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Yes</strong></td>
<td>5 (10.2%)</td>
<td>2 (4.2%)</td>
<td>2 (4.2%)</td>
<td>39 (81.4%)</td>
<td>48 (100%)</td>
</tr>
<tr>
<td><strong>No</strong></td>
<td>17 (7.3%)</td>
<td>8 (3.5%)</td>
<td>24 (10.4%)</td>
<td>182 (78.8%)</td>
<td>231 (100%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>22 (7.9%)</td>
<td>10 (3.6%)</td>
<td>26 (9.3%)</td>
<td>221 (79.2%)</td>
<td>279 (100%)</td>
</tr>
</tbody>
</table>

Pulmonary injury was present in 44.8% patients ($n = 146$) and not present in 55.2% of patients ($n = 180$). A very weak correlation was found between the presence of pulmonary injury and hypoxaemia (SpO2 < 95%) on arrival at the emergency department (Spearman’s rho $r = -0.182$, $p = 0.02$, 2-tailed). The effectiveness of pre-hospital manual ventilation may be a significant contributing factor to oxygen saturation levels during the pre-hospital phase of management of persons with severe traumatic head injury; however,
it is beyond the scope of this study to determine the efficacy of pre-hospital manual ventilation.

5.3.2 Discussion – Pre-Hospital Endotracheal Intubation

Chi et al. (2005) in their study found no significant correlation between endotracheal intubation and improved oxygen saturation levels. No significant difference in the episodes of hypoxia between intubated and non-intubated patients in the pre-hospital setting. Among the 99 patients who were intubated in the field, 28% (n =30) experienced episodes of hypoxia, compared with 30% (n =15) of the 51 non-intubated patients.

The current study also found no significant correlation between pre-hospital endotracheal intubation and improved oxygen saturation levels at the emergency department (r = −0.065, p = 0.276). Analysis found the maintenance of oxygen saturations of greater than 95% were more crucial in improving patient outcome than endotracheal intubation. Therefore endotracheal intubation did not assure adequate ventilation or maintenance of oxygen saturations. Mechanical ventilation is not a routine procedure for paramedics assigned to road ambulances and no documentation of the use of mechanical ventilation was identified in the Ambulance Service patient care records. Analysis also failed to demonstrate a correlation between time to endotracheal intubation and improved outcome. Time to endotracheal intubation, including both pre-hospital and in-hospital intubations, was not a significant predictor of Glasgow Outcome Score in persons with severe traumatic brain injury (r = 0.059, p = 0.319).
Studies by Bochicchio et al. (2003), Murray et al. (2000), Davis et al. (2004), Rainer et al. (1997), Dimetriades et al. (1996) and Regal et al. (1997), all concluded that pre-hospital intubation of patients with severe traumatic brain injury was not associated with improved outcomes. Pre-hospital intubation was associated with higher mortality risk and the efficacy of pre-hospital intubation remains unproven. In all studies the rates of mortality and morbidity were increased in patients receiving pre-hospital endotracheal intubation as opposed to those who did not receive intubation until arrival at the emergency department. Bochicchio et al. (2003), found that field intubations were associated with double the mortality (23% versus 12.4%, p = 0.05). Murray et al. (2000) found mortality in the intubated group was 84% (p = 0.07) compared with 70% in the non-intubated group (p = 0.37).

Similarly the current study found pre-hospital endotracheal intubation to be associated with almost double the rate of mortality: a mortality rate of 75.4% for intubated patients versus 34.2% for non-intubated patients. Significant morbidity (vegetative state or severe disability) was noted in 19.9% of patients intubated in the pre-hospital setting versus 24.9% intubated in the in-hospital environment. The most notable difference was observed in the improved outcome categories of moderate disability and good recovery. Only 4.6% of patients intubated in the pre-hospital setting demonstrated an improved outcome (GOS 4 or 5) as compared with 40.9% for those not intubated in the pre-hospital setting.
The use of endotracheal intubation may not be the single variable that is associated with improved outcomes. Rather the consistency of ventilation rate and tidal volume during resuscitation may be the critical variable associated with improved neurological outcome. The ability of paramedics to maintain consistency of ventilatory rate and tidal volume during patient management at the scene, and during extrication and transportation may be limited without the aid of mechanical ventilation. Further studies considering not only endotracheal intubation, but manual ventilatory technique, SpO2, end-tidal carbon dioxide level and mechanical ventilation would be required to establish the efficacy of advanced pre-hospital airway management and resuscitation in severe traumatic head injury.

Operationally, paramedics work in teams of two and regularly wait at scene for an additional crew to provide assistance for patient management and extrication. Rarely are more than four paramedics responded to a single case. The first crew on scene must consider danger to themselves and the patient, and must control the scene before administering treatment. The first crew on scene then manages the patient until a further support crew arrives. The patient is subsequently extricated on a lifting/carrying device such as a spine board. Where possible, additional assistance from bystanders is sought to lift and carry the patient. If additional assistance is not available during extrication of the patient, then all four paramedics must carry the patient; therefore, ventilation of the patient may not be possible at that time. The process of managing and extricating a patient from a changeable and difficult pre-hospital scene, as opposed to managing the patient in the more stable and more predictable in-hospital setting, may adversely affect
the final outcome of the patient. However, until the magnitude and size of effect are investigated, the efficacy of pre-hospital endotracheal intubation may not be fully understood. Endotracheal intubation should therefore not be perceived as a solitary skill, but as a component of a sequence of airway and ventilation interventions that potentially may improve patient functional outcome following brain trauma.

It should be noted that all studies reviewed (Bochicchio et al., 2003; Murray et al., 2000; Davis et al., 2004; Rainer et al., 1997; Dimetriades et al., 1996; Regal et al., 1997) were undertaken in large metropolitan areas. The efficacy of endotracheal intubation in cases of severe traumatic brain injury in rural and remote areas is still largely unknown. Patients from rural and remote areas were excluded from the current study.

5.3.3 Summary - Pre-Hospital Endotracheal Intubation

A total of 20.2% \((n = 65)\) of the sample received endotracheal intubation in the pre-hospital setting. Results failed to support the hypothesis that persons with severe traumatic head injury will benefit from pre-hospital endotracheal intubation. However, this study did not investigate the efficacy of the concurrent use of end-tidal carbon dioxide monitoring, rapid sequence induction, mechanical ventilation, or the use of positive end expiratory pressure and endotracheal intubation on persons with severe traumatic head injury. Further research is required to investigate the efficacy of early endotracheal intubation versus expedited transport to a trauma facility.
CHAPTER SIX

MAINTENANCE OF OXYGEN SATURATION GREATER THAN OR EQUAL TO 95% AND MAINTENANCE OF A CLEAR AND PATENT AIRWAY IN PATIENTS WITH SEVERE TRAUMATIC HEAD INJURY

6.1 Overview of the Chapter

Chapter Six will present the findings for research question 2: maintenance of clear and patient airway and maintenance of oxygen saturation of greater than 95% will benefit patients with severe traumatic head injury. This chapter will present results and discussion relating to the pre-hospital maintenance of oxygen saturation levels, airway maintenance, the effect of unrelieved pre-hospital airway obstruction, the relationship between cervical collar application and airway maintenance, intermittent positive pressure ventilation in the pre-hospital setting and pre-hospital airway adjunct insertion and the development of respiratory tract infection. The above listed variables and their relationship to the depended variable (GOS) will be discussed (refer to Figure 1).

6.2 Maintenance of Oxygen Saturations Greater than or Equal to 95%

6.2.1 Results - Oxygen Saturation: Initial Pulse Oximetry Reading in the Emergency Department

Pre-hospital pulse oximetry readings were documented in only 13.5% of the total cases ($n = 44$). Two pre-hospital pulse oximetry readings were documented in 70.5% ($n = 31$) of the 44 cases. In 29.5% ($n = 13$) cases, only one reading was documented: therefore
analysis using pre-hospital pulse oximetry was considered unreliable. The first emergency department (ED) oxygen saturation levels (SpO2) were obtained rapidly after arrival of the patient to the ED for 85.6% of patients \((n = 279)\) (mean time from triage time to time of first emergency department observations = 1.9 minutes). The first ED SpO2 level was considered to be an indicator of the oxygenation and ventilation received by the patient in the pre-hospital setting due to the diminutive difference in time from the last pre-hospital to the first ED observations.

Oxygen saturation ranges were divided into the following categories: < 85%; 85 to 89%; 90 to 94%; and 95 to 100%. Of the 279 patients, 20.8% \((n = 58)\) were documented as having a SpO2 of less than 95%. Oxygen saturation levels were found to be 95% or greater in 79.2% \((n = 221)\) of the 279 patients. Initial ED SpO2 was not documented in 14.4% \((n = 47)\) of the 326 cases. Oxygen saturation ranges on arrival at the ED are presented in Table 21.
Table 21  Oxygen Saturation Range on Arrival at the Emergency Department

<table>
<thead>
<tr>
<th>Oxygen Saturation Range – ED</th>
<th>Number (n)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 85%</td>
<td>22</td>
<td>7.9%</td>
</tr>
<tr>
<td>85 – 89%</td>
<td>10</td>
<td>3.6%</td>
</tr>
<tr>
<td>90 – 94%</td>
<td>26</td>
<td>9.3%</td>
</tr>
<tr>
<td>95 –100%</td>
<td>221</td>
<td>79.2%</td>
</tr>
<tr>
<td>Total</td>
<td>279</td>
<td>100.0%</td>
</tr>
</tbody>
</table>

Initial emergency department SpO2 and GOS was documented in 276 cases. Stepwise linear regression revealed that initial emergency department SpO2 was a significant predictor of GOS (r = 0.208, p = 0.001, 95% CI = 0.169 to 0.599). Higher oxygen saturation range was associated with improved outcome with 39.0% of patients with a SpO2 between 95% to 100% (n = 85 of 218 patients) showing improved outcome (GOS 4 or 5). Whereas for those patients with a SpO2 < 95%, only 24.1% of patients demonstrated an improved outcome (n = 14 of 58 patients) (Spearman’s rho = 0.157, p = 0.009, 2-tailed).

Poor outcomes (GOS 1, 2 or 3) were similar amongst the 85% to 89%, 90% to 94% and 95% to 100% SpO2 ranges. Poor outcomes were confirmed in 60.0% (n = 6) in the 85% to 89% range, 61.0% (n = 17) in the 90% to 94% range and 61.0% (n = 133) in the 95% to 100% range. Most notably, 95.5% of those with a SpO2 < 85% had a poor outcome (n
= 21). Overall, for those patients experiencing an episode of pre-hospital hypoxaemia (SpO2 < 95%) 75.9% (n = 44 of 58) had a poor outcome versus 61.0% (n = 133 of 218) in the 95% to 100% SpO2 range. Oxygen saturation of < 85% was associated with mortality rate of 77.3% (n = 17 of 22 patients). Oxygen saturation ranges and GOS were cross-tabulated and results are presented in Table 22.

Table 22  Initial Emergency Department SpO2 Range and GOS

<table>
<thead>
<tr>
<th>GOS</th>
<th>&lt; 85%</th>
<th>85–89%</th>
<th>90–94%</th>
<th>95–100%</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>17 (77.3%)</td>
<td>4 (40.0%)</td>
<td>7 (26.9%)</td>
<td>79 (36.2%)</td>
<td>107 (38.8%)</td>
</tr>
<tr>
<td>Vegetative State</td>
<td>2 (9.1%)</td>
<td>0 (0%)</td>
<td>2 (7.7%)</td>
<td>10 (4.6%)</td>
<td>14 (5.1%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>2 (9.1%)</td>
<td>2 (20.0%)</td>
<td>8 (30.8%)</td>
<td>44 (20.2%)</td>
<td>56 (20.3%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>0 (0%)</td>
<td>2 (20.0%)</td>
<td>3 (11.5%)</td>
<td>22 (10.1%)</td>
<td>27 (10.0%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>1 (4.5%)</td>
<td>2 (20.0%)</td>
<td>6 (23.1%)</td>
<td>63 (28.9%)</td>
<td>72 (26.1%)</td>
</tr>
<tr>
<td>Total</td>
<td>22 (100%)</td>
<td>10 (100%)</td>
<td>26 (100%)</td>
<td>218 (100%)</td>
<td>276 (100%)</td>
</tr>
</tbody>
</table>

The first emergency department oxygen saturation readings are presented in Table 23. A total of 8.0% (n = 22) of the total cohort had de-saturated with SpO2 less than 85%. Three patients recorded SpO2 of less than 60%. The lowest SpO2 recorded was 52%. 

153
Table 23  Oxygen Saturation Reading on Arrival at the Emergency Department

<table>
<thead>
<tr>
<th>Oxygen Saturation ED (%)</th>
<th>Number (n)</th>
<th>Percent (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>52</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>58</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>59</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>60</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>63</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>66</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>71</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>72</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>75</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>77</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>80</td>
<td>5</td>
<td>1.5</td>
</tr>
<tr>
<td>82</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>83</td>
<td>2</td>
<td>0.6</td>
</tr>
<tr>
<td>84</td>
<td>3</td>
<td>0.9</td>
</tr>
<tr>
<td>85</td>
<td>2</td>
<td>0.6</td>
</tr>
<tr>
<td>86</td>
<td>3</td>
<td>0.9</td>
</tr>
<tr>
<td>87</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>88</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>89</td>
<td>3</td>
<td>0.9</td>
</tr>
<tr>
<td>90</td>
<td>4</td>
<td>1.2</td>
</tr>
<tr>
<td>91</td>
<td>2</td>
<td>0.6</td>
</tr>
<tr>
<td>92</td>
<td>9</td>
<td>2.8</td>
</tr>
<tr>
<td>93</td>
<td>4</td>
<td>1.2</td>
</tr>
<tr>
<td>94</td>
<td>6</td>
<td>1.8</td>
</tr>
<tr>
<td>95</td>
<td>9</td>
<td>2.8</td>
</tr>
<tr>
<td>96</td>
<td>22</td>
<td>6.7</td>
</tr>
<tr>
<td>97</td>
<td>15</td>
<td>4.6</td>
</tr>
<tr>
<td>98</td>
<td>27</td>
<td>8.3</td>
</tr>
<tr>
<td>99</td>
<td>52</td>
<td>16.0</td>
</tr>
<tr>
<td>100</td>
<td>98</td>
<td>30.1</td>
</tr>
<tr>
<td>Total</td>
<td>279</td>
<td>85.6</td>
</tr>
<tr>
<td>Missing</td>
<td>47</td>
<td>14.4</td>
</tr>
</tbody>
</table>
As discussed in Chapter Five, the presence of intensive care paramedics did not result in improved oxygenation in patients with severe traumatic head injury. Two-tailed correlation analysis failed to demonstrate a significant difference in Injury Severity Score in cases attended by intensive care paramedics and non-intensive care paramedics (Spearman’s rho = -0.065, p = 0.271). A total of 12.7% of patients cared for by intensive care paramedics (n = 27) were found to have SpO2 readings less than 90% on arrival at the emergency department versus 8.7% (n = 4) for those cared for by non-intensive care paramedics. Oxygen saturation readings on arrival at the emergency department for 79.1% (n = 258) of patients cared for by intensive care and non-intensive care paramedics are presented in Table 24.

<table>
<thead>
<tr>
<th>Oxygen Saturation Range – ED</th>
<th>Intensive Care Paramedic</th>
<th>Non-Intensive Care Paramedic</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 85%</td>
<td>18 (8.5%)</td>
<td>3 (6.5%)</td>
</tr>
<tr>
<td>85–89%</td>
<td>9 (4.2%)</td>
<td>1 (2.2%)</td>
</tr>
<tr>
<td>90–94%</td>
<td>19 (9.0%)</td>
<td>6 (13.0%)</td>
</tr>
<tr>
<td>95–100%</td>
<td>166 (78.3%)</td>
<td>36 (78.3%)</td>
</tr>
<tr>
<td>Total</td>
<td>212 (100%)</td>
<td>46 (100%)</td>
</tr>
</tbody>
</table>
6.2.2 Discussion – Oxygen Saturation and Maintenance of SpO2 95-100%

Analysis in the current study demonstrated the importance of maintaining oxygen saturations between 95-100% following severe traumatic head injury. This would suggest that adequate ventilation and oxygenation is a critical intervention that may afford maximal opportunity for improved patient outcome and inhibit the deleterious effects of hypoxaemia in persons with severe traumatic brain injury. Analysis supported the hypothesis that maintenance of oxygen saturation of greater than or equal to 95% will benefit persons with severe traumatic head injury.

Data in the current study determined that 20.8% of patients suffered hypoxaemia in the pre-hospital setting. Similarly the Brain Trauma Foundation (2007) found in a prospectively collected data set from the Traumatic Coma Data Bank found that hypoxaemia occurred in 22.4% of patients with severe traumatic brain injuries and was significantly associated with increased morbidity and mortality.

Chi et al. (2006), in a study of 150 trauma patients, found that 37 patients (24.7%) had hypoxic episodes only, 14 (9.3%) had hypotensive episodes only and 6 (4.0%) had both hypoxia and hypotension. Mortality rates for patients experiencing hypoxia alone was 37%, hypotension alone 8% and both hypoxia and hypotension was 33%. Overall mortality was 23.3%. Hypoxaemia in the pre-hospital setting was concluded to be a significant determinant of poor outcome. Similarly studies by Stocchetti et al., (1996), Cooke et al., (1995) and Jones et al., (1994) found that episodes of hypoxaemia were significantly associated with poorer outcome.
The variance in patients experiencing episodes of hypoxaemia in the pre-hospital setting ranged from 24.7% (Chi et al., 2006), 27% (Cooke et al., 1995) and 50% (Stocchetti et al., 1996), compared with 20.8% in the current study. Manley et al. (2001) found 20.5% of patients experienced episodes of hypoxaemia in the pre-hospital setting but hypoxaemia was not shown to be a significant predictor of outcome. The Manley et al. cohort, however, included patients with moderate brain injuries in a sample size of 117, as opposed to patients with severe brain injuries only in the current study with a sample size of 326. It is unknown if the disparity in injury severity and sample size between the two studies may have contributed to the disparity in the final results. The mean percentage of pre-hospital hypoxaemia for all five studies (Chi et al., 2006; Cooke et al., 1995; Stocchetti et al., 1996; Manly et al., 2001 & Webb, 2010) is 28.6%.

6.2.3 Summary – Oxygen Saturation and Maintenance of SpO2 95-100%

The results of the current study and the studies reviewed are suggestive of a trend in the occurrence of pre-hospital hypoxaemia in persons with severe traumatic head injury, with the majority of studies revealing a pre-hospital hypoxaemia rate of 20% to 27%. This trend has remained unevaluated and unresolved since 1995. Pre-hospital hypoxaemia is associated with poorer outcome as indicated by high morbidity and mortality rates in all studies. Further study is required to identify causation of hypoxaemia and identify any paramedic procedures and interventions either contributing to the development of hypoxaemia or failing to correct hypoxaemia. Further investigation of paramedic education and training in the management of persons with severe traumatic head injury is also required to identify any deficits in paramedic education and training. Failure to
improve the incidence of pre-hospital hypoxaemia may result in unnecessarily high rates morbidity and mortality following severe traumatic head injury. It is recommended that further research investigating airway maintenance and manual ventilation by paramedics of persons with severe traumatic head injury be undertaken to identify opportunities for improvement in airway maintenance, manual ventilation and oxygenation. It is also recommended that the investigation of the efficacy of cervical collar application analysis of the influence of cervical collar application on airway maintenance and ventilation in persons with severe traumatic head injury in the pre-hospital setting be undertaken. Moreover, persons with severe traumatic head injury should have oxygen saturations maintained at 95% or greater during pre-hospital management.

6.3 Maintenance of a Clear and Patent Airway

6.3.1 Results - Maintenance of a Clear and Patent Airway

A clear and patent airway on arrival at the emergency department was documented in 73.6% of cases ($n = 240$). Airway patency status was not documented in 1.2% of cases ($n = 4$). The presence of an airway obstruction was documented in 25.2% ($n = 82$) of patients on arrival at the emergency department. A very weak correlation was demonstrated between a clear and patent airway on arrival at the emergency department and improved outcome (Spearman’s rho = −0.114, $p = 0.042$, 2-tailed).
6.3.2 Results - Maintenance of a Clear Airway versus Unrelieved Airway Obstruction in the Pre-Hospital Setting

In those patients with an airway obstruction on arrival at the emergency department (25.2%, \( n = 82 \)) blood was the most common cause of airway obstruction. Blood was found in the airway of 61.0%, of patients with airway obstruction (\( n = 50 \) of 82 patients). Causes of airway obstruction are outlined in Table 25.

Table 25 Cause of Airway Obstruction in 82 Patients

<table>
<thead>
<tr>
<th>Cause of Airway Obstruction</th>
<th>Number (n)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluid</td>
<td>6</td>
<td>7.3%</td>
</tr>
<tr>
<td>Blood</td>
<td>50</td>
<td>61.0%</td>
</tr>
<tr>
<td>Foreign Body</td>
<td>1</td>
<td>1.2%</td>
</tr>
<tr>
<td>Vomitus</td>
<td>10</td>
<td>12.2%</td>
</tr>
<tr>
<td>Other</td>
<td>15</td>
<td>18.3%</td>
</tr>
<tr>
<td>Total</td>
<td>82</td>
<td>100.0%</td>
</tr>
</tbody>
</table>

Chewing gum was the foreign body discovered in the airway of one patient. Other causes of airway obstruction included ineffective positioning of the head to allow for adequate air movement during respiration.
Data for both airway status and SpO2 on arrival at the emergency department was documented for 278 patients. Stepwise linear regression revealed that airway obstruction was a significant predictor of SpO2 range (< 85%; 85-89%; 90-94%; 95-100%) on arrival at the emergency department (r = −0.247, p < 0.001, 95% CI = −0.728 to −0.266). Cross-tabulation of airway status and SpO2 on arrival at the emergency department is presented in Table 26.

Table 26  Airway Status and SpO2 Range on Arrival at the Emergency Department

<table>
<thead>
<tr>
<th>Oxygen Saturation Range – ED</th>
<th>Airway Obstruction Yes</th>
<th>Airway Obstruction No</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 85%</td>
<td>13 (18.1%)</td>
<td>9 (4.4%)</td>
</tr>
<tr>
<td>85 – 89%</td>
<td>2 (2.8%)</td>
<td>7 (3.4%)</td>
</tr>
<tr>
<td>90 – 94%</td>
<td>12 (16.6%)</td>
<td>14 (6.8%)</td>
</tr>
<tr>
<td>95 –100%</td>
<td>45 (62.5%)</td>
<td>176 (85.4%)</td>
</tr>
<tr>
<td>Total</td>
<td>72 (100%)</td>
<td>206 (100%)</td>
</tr>
</tbody>
</table>

Hypoxaemia (SpO2 < 95%) was more common in patients with an airway obstruction on arrival at the emergency department than those patients where airways were clear (37.5% vs 14.6% respectively; p < 0.001).

A correlation was found between GOS and oxygen saturation range on arrival at the emergency department (Spearman’s rho = 0.254, p < 0.001, 2-tailed). Linear regression
found both initial ED SpO2 range and airway obstruction on arrival at the emergency department to be significant predictors of outcome (GOS); however, the correlation between airway obstruction and GOS is considered very weak. Data relating to ED SpO2 range, airway obstruction and GOS is presented in Table 27.

Table 27  Emergency Department (ED) Oxygen Saturation Range, Airway Obstruction and GOS

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Correlation Coefficient (r)</th>
<th>p Value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>ED SpO2 Range</td>
<td>0.202</td>
<td>p = 0.001</td>
<td>0.159 to 0.591</td>
</tr>
<tr>
<td>Airway Obstruction on Arrival at ED</td>
<td>−0.116</td>
<td>p = 0.038</td>
<td>−0.847 to −0.017</td>
</tr>
</tbody>
</table>

Stepwise Linear Regression – Dependent variable – GOS

Poor outcome (GOS 1, 2 or 3) was demonstrated in 78.1% of patients with an airway obstruction on arrival at the emergency department (n = 64) versus 62.4% of patients with a clear airway (n = 148). Improved outcomes (GOS 4 or 5) were observed in 21.9% of patients with an airway obstruction (n = 18) versus 37.6% of patients with a clear airway (n = 89). An unrelieved airway obstruction in the pre-hospital setting was associated with higher morbidity (33.0% vs 20.7%) and higher mortality (45.1% vs 41.8%); however, these results were not significant at the 0.05 level. Cross tabulation of airway status on arrival at the emergency department and GOS is presented in Table 28.
Table 28  Airway Status and GOS – Cross Tabulation

<table>
<thead>
<tr>
<th>GOS</th>
<th>Airway Clear on Arrival at ED</th>
<th>Airway Obstructed on Arrival at ED</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>99 (41.8%)</td>
<td>37 (45.1%)</td>
<td>136 (42.6%)</td>
</tr>
<tr>
<td>Vegetative State</td>
<td>6 (2.5%)</td>
<td>8 (9.8%)</td>
<td>14 (4.4%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>43 (18.1%)</td>
<td>19 (23.2%)</td>
<td>62 (19.5%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>22 (9.3%)</td>
<td>6 (7.3%)</td>
<td>28 (8.8%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>67 (28.3%)</td>
<td>12 (14.6%)</td>
<td>79 (24.8%)</td>
</tr>
<tr>
<td>Total</td>
<td>237 (100%)</td>
<td>82 (100%)</td>
<td>319 (100%)</td>
</tr>
</tbody>
</table>

6.3.3 Discussion - Maintenance of a Clear Airway versus Unrelieved Airway Obstruction in the Pre-Hospital Setting

The current study revealed that unrelieved airway obstruction in the pre-hospital setting to be common. One quarter of all patients (25.2%) were found to have an airway obstruction on arrival at the emergency department. This study also demonstrated the existence of a significant correlation between airway obstruction and oxygen saturation range on arrival at the emergency department (Spearman’s rho = −0.254, p < 0.001, 2-tailed). Obstructions varied from partial obstruction to almost complete obstruction of the airway. A point worthy of note is that a correlation was also found between oxygen
saturation level on arrival at the emergency department and Glasgow Outcome Scale (Spearman’s rho = 0.254, p < 0.001, 2-tailed).

6.3.4 Summary - Maintenance of a Clear Airway versus Unrelieved Airway Obstruction in the Pre-Hospital Setting

Analysis revealed the importance of airway patency in improved outcome following severe traumatic head injury. This would suggest that prompt clearance of any airway obstruction and maintenance of airway patency are critical interventions that may afford maximal opportunity for improved patient outcome and inhibit the deleterious effects of airway occlusion and hypoxaemia in persons with severe traumatic brain injury. Analysis supported the hypothesis that patients with severe traumatic head injury would benefit from the maintenance of a clear and patent airway during the pre-hospital phase of management.

6.3.5 Results - Maintaining a Patent Airway and the Use of Suctioning

Suctioning of the airway was documented as being performed in only 33.4% of the 326 cases (n = 109). Suctioning of the airway was documented as not undertaken in 56.5% of cases (n = 184). In 10.1% of cases (n = 33) it was not documented if suctioning of the airway was performed or not performed in the pre-hospital phase of management. Airway suctioning is either recorded as undertaken or not undertaken; the frequency of airway suctioning in the pre-hospital setting is not recorded on Ambulance Service of NSW patient care records. It is unspecified if suctioning was undertaken once or on multiple occasions during the pre-hospital phase of management. The use of suctioning in the pre-
hospital setting and the incidence of airway obstruction on arrival at the emergency
department is presented in Table 29.

Table 29  Suctioning of the Airway in the Pre-Hospital Setting and Airway
Status on Arrival at the Emergency Department

<table>
<thead>
<tr>
<th>Airway Status</th>
<th>Airway Suctioning Performed</th>
<th>Airway Suctioning Not Performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway Clear</td>
<td>75 (68.8%)</td>
<td>142 (77.2%)</td>
</tr>
<tr>
<td>Airway Obstructed</td>
<td>34 (31.2%)</td>
<td>42 (22.8%)</td>
</tr>
<tr>
<td>Total</td>
<td>109 (100%)</td>
<td>184 (100%)</td>
</tr>
</tbody>
</table>

Despite airway suctioning being performed in the pre-hospital setting, 31.2% of patients
were discovered to have an unrelieved airway obstruction on arrival at the ED. For those
patients who did not receive suctioning, 22.8% presented to the ED with an undetected
and unrelieved airway obstruction (refer to Table 29).

Airway obstruction was associated with increased rates of hypoxaemia: 37.5% of patients
with an airway of obstruction on arrival at the ED were also hypoxic (SpO2 < 95%) \((n = 27)\), whereas only 14.6% of patients with a clear airway were hypoxic on arrival at the
ED \((n = 30)\). Correlation analysis demonstrated a significant relationship between a clear
airway and improved oxygen saturation \((\text{SpO2} \text{ 95 to100\%})\) (Spearman’s \(\rho = -0.254\), \(p < 0.001\), 2-tailed).
Of those patients with an airway obstruction and hypoxaemia on arrival in the emergency department 53.5% had received suctioning in the pre-hospital setting \((n = 15)\): of those 35.7% \((n = 10)\) presented with moderate to severe hypoxaemia \((\text{SpO}2 < 90\%)\). The majority of airway obstruction was due to blood, fluid and vomitus in the posterior hypopharynx of the respiratory tract. An overview of documented occurrences of airway suctioning in the pre-hospital environment, airway obstruction remaining in-situ on arrival at ED and emergency department oxygen saturation readings is provided in Table 30.

Table 30  
Suctioning of the Airway in the Pre-Hospital Setting, Airway Obstruction Remaining on Arrival at the Emergency Department and Emergency Department SpO2 Range

<table>
<thead>
<tr>
<th>Oxygen Saturation Range – ED</th>
<th>Airway Suctioning Performed – Total Number</th>
<th>Airway Suctioning Performed and Obstruction Remaining In-situ at ED</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 85%</td>
<td>16 (17.8%)</td>
<td>9 (32.1%)</td>
</tr>
<tr>
<td>85-89%</td>
<td>2 (2.2%)</td>
<td>1 (3.6%)</td>
</tr>
<tr>
<td>90-94%</td>
<td>11 (12.2%)</td>
<td>5 (17.9%)</td>
</tr>
<tr>
<td>95-100%</td>
<td>61 (67.8%)</td>
<td>13 (46.4%)</td>
</tr>
<tr>
<td>Total</td>
<td>90 (100%)</td>
<td>28 (100%)</td>
</tr>
</tbody>
</table>

Results suggest that suction is not performed as often as required. While suction is periodically performed in the pre-hospital setting the frequency of suctioning may be
insufficient with 31.2% of patients receiving suctioning were still found to have an airway obstruction on arrival at the emergency department ($n = 28$ of 90, refer to Table 30).

### 6.3.6 Discussion - Maintaining a Patent Airway and the Use of Suctioning

Patency of the airway is a factor that is thought to influence oxygenation and ventilation, and ultimately outcome, following severe traumatic brain injury. The Brain Trauma Foundation (2000a) recommends that the correction of airway compromise and ventilatory insufficiency should be a priority in the management of the patient with brain injury. Obstructed airway and subsequent hypoxaemia may result in secondary insult and poor outcome. Birnbaum and Pollack (2002) recommend that maintenance of a patent airway is essential for good outcome and failure to achieve and maintain a patent airway can lead to hypoxaemia and neurological injury within minutes. While it is acknowledged that airway obstruction potentially may result in an adverse outcome, no related research was found that determined the frequency of airway obstruction in the pre-hospital setting or considered the affects of pre-hospital airway obstruction on neurological outcome.

Results of the current study indicate that paramedics are failing to perform airway suctioning and clear an obstruction in 22.8% of patients, and are failing to clear detected obstructions in 31.2% of patients. Airway obstruction is either not being performed when required or is not performed frequently enough. The factors relating to airway obstruction not being cleared by paramedics was beyond the scope of this research. However, possibilities giving rise to the non-clearance of airway obstruction were postulated as being: the application of a rigid cervical collar and the inability of paramedics to open the
mouth and visualise the obstruction. Direct laryngoscopy using a laryngoscope is not an approved intervention for non-intensive care paramedics. Therefore airway obstruction may not be easily visualised by ambulance paramedics and paramedics may be hesitant to loosen or remove the cervical collar because of the possibility of cervical spine injury. Further research is required to explore these suppositions.

6.3.7 Summary - Maintaining a Patent Airway and the Use of Suctioning

Results suggest that uninterrupted patency of the airway during and post-injury is a significant predictor of improved oxygenation and oxygenation saturation is a predictor of neurological outcome. Therefore any airway occlusion may be deleterious to patient outcome. Analysis supported the hypothesis that persons with severe traumatic head would benefit from the maintenance of a clear and patent airway. Therefore more emphasis should be given to basic airway techniques such as regular inspection and clearance of the airway, basic positioning of the patient to maximise airway patency and minimising scene time in order to decrease time to definitive airway control within the in-hospital setting.

6.3.8 Results - Airway Status, Oxygenation and the Application of a Rigid Cervical Collar

Data was available for airway status on arrival at the emergency department and the application of a cervical collar in the pre-hospital setting in 322 cases, which is presented in Table 31.
Table 31  Airway Status on Arrival at ED and Cervical Collar Application in the Pre-Hospital Setting for 322 Patients

<table>
<thead>
<tr>
<th>Airway Status on Arrival at ED</th>
<th>Cervical Collar In Situ</th>
<th>Nil Cervical Collar</th>
<th>Not Documented</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway Clear</td>
<td>186 (57.8%)</td>
<td>2 (0.6%)</td>
<td>52 (16.1%)</td>
<td>240 (74.5%)</td>
</tr>
<tr>
<td>Airway Obstructed</td>
<td>69 (21.4%)</td>
<td>1 (0.3%)</td>
<td>12 (3.7%)</td>
<td>82 (25.5%)</td>
</tr>
<tr>
<td>Total</td>
<td>255 (79.2%)</td>
<td>3 (0.9%)</td>
<td>64 (19.9%)</td>
<td>322 (100%)</td>
</tr>
</tbody>
</table>

Rigid cervical collars are applied in the pre-hospital setting for possible or suspected cervical spine injury. In only three cases was it documented that a cervical collar was not in-situ on arrival at the emergency department and none of those three patients were found to have a spinal injury. The one patient who did not receive a cervical collar, but did have an airway obstruction, was a 68-year-old obese male who weighed 139 kilograms. The patient fell seven metres onto concrete and underwent two episodes of seizure activity with each lasting 60 seconds. The patient was described as a grade 4 intubation. All pre-hospital and in-hospital attempts at endotracheal intubation failed. On arrival at the emergency department, the patient was reported to be receiving ineffective ventilations and as such oxygen saturation level was less than 85%. The patient was stated as having an airway obstruction and cause of the airway obstruction was stated as ineffective positioning of the airway. The attending paramedics documented that they encountered immense difficulty in maintaining the airway while a cervical collar was in-situ and elected to remove the cervical collar. The time of removal was not documented.
Spinal injury occurred in 9.9% of the total cohort \((n = 32)\) of 322 patients) and 56.3% of those patients with a spinal injury had a cervical spine injury \((n = 18)\). Cervical spine injury occurred in 5.6% of the total cohort \((n = 18)\) with 3.1% being stable injuries \((n = 10)\) and 2.5% being unstable injuries \((n = 8)\). Data relating to spinal injury and type of injury in 322 cases are presented in Table 32.

### Table 32 Region of Spinal Injury and Injury Stability in 322 Patients

<table>
<thead>
<tr>
<th>Spinal Injury Region</th>
<th>Frequency</th>
<th>Stable Injury</th>
<th>Unstable Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nil</td>
<td>292 (90.7%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Cervical</td>
<td>18 (5.6%)</td>
<td>10 (3.1%)</td>
<td>8 (2.5%)</td>
</tr>
<tr>
<td>Thoracic</td>
<td>12 (3.7%)</td>
<td>9 (2.8%)</td>
<td>3 (0.9%)</td>
</tr>
<tr>
<td>Lumbar</td>
<td>2 (0.6%)</td>
<td>2 (0.6%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Total</td>
<td>322 (100%)</td>
<td>21 (6.5%)</td>
<td>11 (3.4%)</td>
</tr>
</tbody>
</table>

Data describing spinal injury status, the stability of injury and Glasgow Outcome score are presented in Table 33. It should be emphasised that no permanent spinal cord injury with neurological deficit was documented in any patient who survived.
Regression analysis failed to find the presence of a spinal injury to be a significant predictor of outcome when using the Glasgow Outcome Scale as the dependant variable \(r = 0.053, p = 0.341\). However the presence of an airway obstruction on arrival at the emergency department was found to be a weak but significant predictor of poorer outcome when analysed using stepwise linear regression \(r = -0.116, p = 0.038, 95\% CI = -0.854\) to \(-0.024\). Cross-tabulation data detailing airway status on arrival at the emergency department, spinal injury and GOS are presented in Tables 34 and 35.
<table>
<thead>
<tr>
<th>GOS</th>
<th>Nil Spinal Injury</th>
<th>Cervical Spine Injury</th>
<th>Thoracic Spine Injury</th>
<th>Lumbar Spine Injury</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>88 (40.9%)</td>
<td>5 (45.5%)</td>
<td>5 (62.5%)</td>
<td>0 (0%)</td>
<td>98 (41.6%)</td>
</tr>
<tr>
<td>Vegetative State</td>
<td>4 (1.7%)</td>
<td>0 (0%)</td>
<td>1 (12.5%)</td>
<td>1 (50.0%)</td>
<td>6 (2.5%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>40 (18.6%)</td>
<td>2 (18.2%)</td>
<td>1 (12.5%)</td>
<td>0 (0%)</td>
<td>43 (18.2%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>21 (9.8%)</td>
<td>1 (9.0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>22 (9.3%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>62 (28.9%)</td>
<td>3 (27.3%)</td>
<td>1 (12.5%)</td>
<td>1 (50.0%)</td>
<td>67 (28.4%)</td>
</tr>
<tr>
<td>Total</td>
<td>215 (100%)</td>
<td>11 (100%)</td>
<td>8 (100%)</td>
<td>2 (100%)</td>
<td>236 (100%)</td>
</tr>
</tbody>
</table>
Table 35  Obstructed Airway on Arrival at ED, Spinal Injury and GOS

<table>
<thead>
<tr>
<th>GOS</th>
<th>Nil Spinal Injury</th>
<th>Cervical Spine Injury</th>
<th>Thoracic Spine Injury</th>
<th>Lumbar Spine Injury</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Death</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>31 (43.1%)</td>
<td>5 (83.3%)</td>
<td>1 (25.0%)</td>
<td>0 (0%)</td>
<td>37 (45.1%)</td>
</tr>
<tr>
<td><strong>Vegetative State</strong></td>
<td>7 (9.7%)</td>
<td>1 (16.7%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>8 (9.8%)</td>
</tr>
<tr>
<td><strong>Severe Disability</strong></td>
<td>17 (23.6%)</td>
<td>0 (0%)</td>
<td>2 (50.0%)</td>
<td>0 (0%)</td>
<td>19 (23.2%)</td>
</tr>
<tr>
<td><strong>Moderate Disability</strong></td>
<td>6 (8.3%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>6 (7.3%)</td>
</tr>
<tr>
<td><strong>Good Recovery</strong></td>
<td>11 (15.3%)</td>
<td>0 (0%)</td>
<td>1 (25.0%)</td>
<td>0 (0%)</td>
<td>12 (14.6%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>72 (100%)</td>
<td>6 (100%)</td>
<td>4 (100%)</td>
<td>0 (0%)</td>
<td>82 (100%)</td>
</tr>
</tbody>
</table>

Cervical spine injury with concurrent airway obstruction was associated with high mortality and morbidity. For those patients with a cervical spine injury, 45.5% with a clear airway died versus 83.3% of those with an obstructed airway. Poor outcomes (GOS 1, 2 or 3) were confirmed in 63.6% of patients with a cervical spine injury and a clear airway ($n = 7$) versus 100% in those with a cervical spine injury and concurrent obstructed airway ($n = 6$). Improved outcomes (GOS 4 or 5) were noted in 36.4% of patients with a cervical spine injury and a clear airway ($n = 4$) as compared with no improved outcomes in the cervical spine injury with concurrent airway obstruction group.
Eight patients were found to have unstable cervical spine injury and 75.0% of those \( (n = 6) \) were found to have airway obstruction on arrival at the emergency department. Hypoxaemia was confirmed in 37.5% of patients with unstable cervical spine injury and concurrent airway obstruction \( (n = 3) \) with two patients having a SpO2 of less than 85% and one patient having a SpO2 between 90-94%. The two patients with a SpO2 < 85% died and the patient with a SpO2 between 90-94% suffered severe disability without permanent spinal cord injury. The mortality rate for patients with an unstable cervical spine injury was 75.0% \( (n = 6) \) with death being attributed to extensive brain injury and not to spinal cord injury.

**6.3.9 Discussion – Airway Maintenance, the Occurrence of Spinal Injury and Pre-Hospital Cervical Collar Application**

The application of a rigid cervical collar is routine in cases of severe traumatic head injury as outlined the Ambulance Service of NSW Protocols, Pharmacology and Procedures (1997, 2007). The rationale for the application of a rigid cervical collar in trauma cases with a decreased level of consciousness is based on potential spinal injury due to mechanism of injury and high index of suspicion. Cervical collars are applied prophylactically until the cervical spine is cleared of injury through x-ray and other medical imaging and physical assessment by a physician. Kolb et al. (1999) suggest justification for the routine application of a rigid cervical collar in trauma is that the collar may attenuate significant movement of the cervical spine and may also act as a marker indicating that the cervical spine has not been cleared of injury. However Kolb et al. found the application of a rigid cervical collar to increase cerebrospinal fluid pressure in 20 patients during lumbar puncture and found mean cerebrospinal fluid pressure
(CSFP) pre-collar was 176.8 mm of water (mm H2O) which increased to 201.5 mm H2O post-collar application. The mean difference was 24.8 mm H2O (Standard Deviation 28.4) which achieved statistical significance (p = 0.001). They concluded that in patients with already elevated ICP, further increasing ICP through the application of a cervical collar may be detrimental to patient outcome and therefore the negative effects may outweigh the benefits of a cervical collar in brain trauma patients.

Bunn et al. (2001) reviewed existing research on spinal immobilisation of trauma patients in the pre-hospital setting and proposed that spinal immobilisation in trauma is undertaken largely in response to fear of litigation and that the current protocol for pre-hospital spinal immobilisation has a strong historical precedent, rather than being based on scientific research. Bunn et al. (2001) further suggested that the adverse effects of rigid cervical collar application in pre-hospital spinal immobilisation, such as the development of airway obstruction, increased intracranial pressure, increased risk of aspiration and restricted respiration, may have deleterious effects on patient outcome. Airway obstruction caused by spinal immobilisation may increase mortality and morbidity.

In the current study it was noted that the substantial majority of patients (79.2%, n = 255) had a rigid cervical collar applied during the pre-hospital phase of management. In only 0.9% of cases (n = 3) a rigid collar was documented as not being applied, and application of a cervical collar was unknown in 19.9% of cases (n = 64). As application of a cervical collar is considered by the Ambulance Service of NSW to be an obligatory intervention
in the management of the patient with head injuries, then it is probable that a collar was applied in the majority of the 64 unknown cases, however this cannot be verified. While bivariate correlation and stepwise linear regression found a significant relationship between cervical collar application and emergency department SpO2, no consequential or meaningful interpretation and conclusion relating to the efficacy of the application of a rigid cervical collar was possible due to the large disparity in group size between those patients with a collar and those patients without a collar (Spearman’s rho 0.145, p = 0.015, 2-tailed; r = 0.127, p = 0.033, 95% CI = 0.011 to 0.276). The extrapolation of these results to the clinical context is therefore unclear and further investigation is necessary.

Airway suctioning, direct laryngoscopy, manual intermittent positive pressure ventilation via bag-valve-mask, visualisation of the oropharynx and maintenance of a patent airway may perhaps be made more difficult with a rigid cervical collar in-situ, particularly in a moving ambulance vehicle. Urgent transportation to hospital and extraneous movement of the vehicle further increases the difficulty of maintaining a patent airway and adequate ventilation, thus predisposing the patient to increased risk of airway occlusion. It should also be noted that the cause of airway obstruction in 18.3% of patients (n = 15) was documented as ineffective positioning of the head to allow for adequate air movement during respiration and 80.5% (n = 66) due to blood, fluid and vomitus. Further investigation is required to determine the level of association between cervical collar and airway occlusion or obstruction.
Rigid cervical collars were applied to the greater majority of patients; however, spinal injury occurred in only 9.8% of cases \((n = 32)\), with 5.5% being cervical injuries \((n = 18)\). Thoracic spinal injury occurred in 3.7% of cases \((n = 12)\) and lumbar spinal injury in 0.6% of cases \((n = 2)\). Unstable spinal injury was observed in very few cases: unstable cervical spine injury occurred in 2.5% of cases \((n = 8)\), the thoracic spine in 0.9% of cases \((n = 3)\) and no unstable injury was found in the lumbar spine. In those patients with unstable cervical spine injury, six died, one patient remained in a vegetative state and one patient experienced severe disability.

Overall, spinal injury was associated with a poor outcome, where 50% of spinally injured patients died \((n = 16)\), 9.4% remained in a vegetative state \((n = 3)\) and 18.8% experienced severe disability \((n = 6)\). The resultant outcome for 3.1% was moderate disability \((n = 1)\) and 18.8% \((n = 6)\) had a good recovery. All patients who had moderate disability or a good recovery were found to have stable spinal injuries. The benefit of cervical collar application was not established in patients with unstable cervical spine injury as the associated mortality was 75% and profound morbidity was noted in the remaining 25%. Moreover, no patient who survived was shown to have a spinal cord injury with permanent neurological deficit.

Stepwise linear regression failed to find the presence of a spinal injury as a significant predictor of outcome \((r = 0.053, p = 0.341)\). The result may be subject to type II error due to under-representation of spinally injured patients in the complete cohort. Descriptive
analysis does point to spinal injury being associated with poorer outcome, especially unstable injury; however, the magnitude of the relationship remains unidentified.

The presence of an airway obstruction on arrival at the emergency department was, however, found to be a significant predictor of outcome when analysed using stepwise linear regression ($r = -0.116$, $p = 0.038$, 95% CI = $-0.854$ to $-0.024$). A total of 82 patients experienced an airway obstruction in the pre-hospital setting that remained unrelieved on arrival at the emergency department. Results suggest that airway patency and adequacy of ventilation are important interventions that may potentially improve neurological outcome following severe traumatic brain injury. However, care should be exercised when generalising the results in the clinical context due to the diminutive magnitude of the correlation coefficient. Results may have been confounded by the type and extent of airway obstruction in the cohort of patients with an unrelieved airway obstruction on arrival at the emergency department.

6.3.10 Summary – Airway Maintenance, the Occurrence of Spinal Injury and Pre-Hospital Cervical Collar Application

Cervical spine injury was found to occur in 18 patients (5.6%); unstable cervical injury was found in merely 8 patients (2.5%); and 6 of the 8 patients subsequently died from brain injury rather as a result of the spinal injury. In the resounding majority of cases cervical spine injury was not found (94.5%, $n = 308$) and the application of a cervical collar would therefore provide no benefit. Moreover, it is reasonable to postulate that the application of a rigid cervical collar may be deleterious to patient outcome by way of
associated airway obstruction, reduced ventilation and increased intracranial pressure. Consideration should be given to the modified or controlled use of rigid cervical collars in patients with brain injuries. Rigid cervical collars should not be applied or if present should be removed when airway management is compromised. Manual in-line traction or maintenance of the head in the neutral position by way of sandbags or head blocks may be preferable to the application of a rigid collar. Rigid cervical collars may be of most benefit during extrication of the patient from the scene only, then being released during transportation so as to afford maximal opportunity for effective airway management by attending paramedics. Consideration should be given to either withholding the application of a rigid cervical collar or removing a cervical collar if airway maintenance and ventilation are compromised during the pre-hospital phase of management of persons with severe traumatic head injury.

6.4 Pre-Hospital Ventilation, Oxygen Saturation and End-Tidal Carbon Dioxide Levels

6.4.1 Results – Pre-Hospital Intermittent Positive Pressure Ventilation and Oxygen Saturation

Intermittent positive pressure ventilation (IPPV) was performed in the pre-hospital setting in 38.9% of cases \( (n = 127) \). In 19.3% of cases \( (n = 63) \), it could not be determined if IPPV was performed or not performed due to incomplete documentation. Data relating to IPPV was documented in 80.6% of cases \( (n = 263 \) of the 326 cases). No correlation was found between pre-hospital IPPV and SpO2 range on arrival at the emergency department.
(Spearman’s rho = 0.093, p = 0.133). Pre-hospital IPPV and the first emergency department oxygen saturation range are presented in Table 36.

**Table 36  Oxygen Saturation Range on Arrival at the Emergency Department and Intermittent Positive Pressure Ventilation**

<table>
<thead>
<tr>
<th>Oxygen Saturation Range – ED</th>
<th>IPPV - Yes</th>
<th>IPPV - No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 85%</td>
<td>15 (5.7%)</td>
<td>6 (2.3%)</td>
<td>21 (8.0%)</td>
</tr>
<tr>
<td>85-89%</td>
<td>4 (1.5%)</td>
<td>6 (2.3%)</td>
<td>10 (3.8%)</td>
</tr>
<tr>
<td>90-94%</td>
<td>13 (4.9%)</td>
<td>13 (4.9%)</td>
<td>26 (9.9%)</td>
</tr>
<tr>
<td>95-100%</td>
<td>95 (36.1%)</td>
<td>111 (42.2%)</td>
<td>206 (78.3%)</td>
</tr>
<tr>
<td>Total</td>
<td>127 (10.3%)</td>
<td>136 (51.7%)</td>
<td>263 (100%)</td>
</tr>
</tbody>
</table>

Results reveal that 18.4% of patients ($n = 25$) who did not receive IPPV did require ventilatory support through IPPV and were subsequently hypoxic on arrival at the emergency department. A further 25.2% of patients ($n = 32$) who did receive IPPV in the pre-hospital setting remained hypoxic on arrival at the emergency department. These findings suggest there is an inability of some paramedics to accurately determine the presence of hypoventilation in patients with severe traumatic head injury and/or adequately ventilate a hypoventilating patient to correct the presence of hypoxaemia.
Paramedics are required to document the respiratory status of patients with categories including spontaneous breathing, shallow or retractive respiration, apnoea, hyperventilation or irregular respirations. Respiratory status, IPPV and emergency department oxygen saturation ranges are presented in Table 37.
### Table 37  Respiratory Status, Oxygen Saturation Range on Arrival at the Emergency Department and Intermittent Positive Pressure Ventilation

<table>
<thead>
<tr>
<th>Pre-hospital Respiratory Status</th>
<th>Oxygen Saturation Range – ED</th>
<th>IPPV - Yes</th>
<th>IPPV - No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Spontaneous</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 85%</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>85-89%</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>90-94%</td>
<td>3</td>
<td>9</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>95-100%</td>
<td>8</td>
<td>87</td>
<td>95</td>
<td>95</td>
</tr>
<tr>
<td>Total</td>
<td>11</td>
<td>100</td>
<td>111</td>
<td>111</td>
</tr>
<tr>
<td><strong>Shallow/Retractive</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 85%</td>
<td>10</td>
<td>5</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>85-89%</td>
<td>2</td>
<td>3</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>90-94%</td>
<td>7</td>
<td>3</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>95-100%</td>
<td>66</td>
<td>17</td>
<td>77</td>
<td>77</td>
</tr>
<tr>
<td>Total</td>
<td>79</td>
<td>28</td>
<td>107</td>
<td>107</td>
</tr>
<tr>
<td><strong>Hyperventilating</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 85%</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>85-89%</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>90-94%</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>95-100%</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>2</td>
<td>3</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td><strong>Irregular</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 85%</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>85-89%</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>90-94%</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>95-100%</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>Apnoeic</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 85%</td>
<td>4</td>
<td>0</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>85-89%</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>90-94%</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>95-100%</td>
<td>20</td>
<td>1</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
<td>1</td>
<td>27</td>
<td>27</td>
</tr>
<tr>
<td><strong>Not Documented</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 85%</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>85-89%</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>90-94%</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>95-100%</td>
<td>4</td>
<td>1</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>5</td>
<td>1</td>
<td>6</td>
<td>6</td>
</tr>
</tbody>
</table>
The category of shallow and/or retractive respirations demonstrated the greatest variance in SpO2 readings and the highest percentage of hypoxaemia: 26.2% of patients within this category did not receive IPPV \((n = 28)\) despite the respiratory status being assessed as shallow. Of those 28 patients 39.3% were hypoxic on arrival at the emergency department \((n = 11)\) (refer to Table 35). Further, 28.6% of the 28 patients had an initial ED SpO2 reading of <90% indicating the presence of moderate to severe hypoxaemia.

For those patients who did receive IPPV \((n = 79)\), 24.1% remained hypoxic on arrival at hospital \((\text{SpO}_2 < 95\%)\) \((n = 19)\) and of those 18.2% \((n = 12)\) had a SpO2 less than 90%. In addition to this, 13.0% of patients who were documented as having spontaneous respirations were also hypoxic on arrival at the emergency department \((n = 13)\) which suggests the respiratory status may have been inaccurately assessed and IPPV may have been required due to respiratory dysfunction and hypoventilation. All patients were documented as receiving supplemental oxygen via an oxygen therapy mask.

### 6.4.2 Discussion—Pre-Hospital Intermittent Positive Pressure Ventilation and Oxygen Saturation

The importance of maintaining SpO2 greater than 95% was established and discussed in sections 6.2.1 and 6.2.2 of this thesis. Hypoxaemia following severe traumatic head injury is associated with poor functional outcome and may lead to secondary brain injury. Hypoxaemia and hypercapnoea may concurrently exist due to respiratory dysfunction and hypoventilation (Brain Trauma Foundation, 2000a). Atkinson, Anderson and Murray (1998) found respiratory dysfunction to be common following severe brain trauma due to brain-stem injury in an animal model. Severe brain trauma was induced in a sample of six
rats. All animals displayed brain-stem mediated respiratory dysfunction as well as significantly elevated mean arterial blood pressure, intracranial pressure and increases in partial pressure of carbon dioxide (hypercapnoea). Increases in partial pressure of carbon dioxide could be managed with intermittent positive pressure ventilation via mechanical ventilation.

Several studies have demonstrated the deleterious effects of hypoventilation, hypoxaemia and hypercapnoea in persons with severe traumatic head injury (Warner et al., 2007; Davis et al., 2006b; Chi et al., 2006; Obrist et al., 1984; Davis et al., 2004). However no study was found that investigated the effect of manual ventilation of patients with severe traumatic head injury by ambulance paramedics.

6.4.3 Summary—Pre-Hospital Intermittent Positive Pressure Ventilation and Oxygen Saturation

Results of the current study point to the possibility of some inaccuracy by paramedics to assess respiratory status and adequately ventilate a patient to correct the presence of hypoxaemia. The ability of paramedics to assess and manage the ventilation status of patients with severe traumatic head injury was not a research question within this study. However, results would indicate that the ability of paramedics to accurately assess ventilation status is uncertain and further investigation is required. It is recommended that further research be undertaken into ability of paramedics to assess the respiratory and ventilation status of persons with severe traumatic head injury.
6.4.4 Results - Ventilation and End-Tidal Carbon Dioxide Readings within the In-Hospital Setting

End-tidal carbon dioxide monitoring was not an approved procedure in the pre-hospital setting. End-tidal carbon dioxide was determined during the first vital sign survey in only 4.6% \((n = 15)\) patients on arrival at the emergency department. In 47.2% of cases \((n = 154)\), end-tidal carbon dioxide levels were not determined for some time after arrival at the emergency department and subsequently an estimation of the efficacy pre-hospital ventilation could not be established. In a total of 51.8% of cases \((n = 169)\) end-tidal carbon dioxide was determined in the emergency department. In a further 48.2% of cases \((n = 157)\) end-tidal carbon dioxide levels were not determined until after the patient had been transferred the emergency department or the emergency department readings were not documented.

Emergency department end-tidal carbon dioxide levels were found to be a weak but significant predictor of outcome \((r = 0.192, p = 0.013, 95\% \text{ CI} = 0.008 \text{ to } 0.066)\). Forty-five percent \((45\%)\) of normocapnoeic patients \((\text{PaCO}_2 = 30 \text{ to } 45 \text{ mmHg})\) were observed to have improved outcomes \((n = 45)\) \((\text{GOS 4 or 5})\) versus 27.8\% \((n = 17)\) of hypocapnoeic patients \((\text{PaCO}_2 < 30 \text{ mmHg})\). End-tidal carbon dioxide readings of less than 30 mmHg were associated with poorer outcome. Of those patients experiencing hypocapnoeic episodes in the emergency department 72.1\% \((n = 44)\) had a poor outcome \((\text{GOS 1, 2 or 3})\) as compared with the normocapnoeic group with a poor outcome observed in 55\% \((n = 55)\). Emergency department hypocapnoea was associated with high mortality \((37.7\%, n = 23)\). In the hypercapnoeic group 50\% were observed to have a poor outcome and 50\% an improved outcome. It should be noted that this group had a very
small sample size \( (n = 8) \) and results may not be representative due to sample bias. A summary of emergency department end-tidal carbon dioxide levels and Glasgow Outcome Scale are provided in Table 38.

### Table 38 Emergency Department End-Tidal Carbon Dioxide and Glasgow Outcome Scale (GOS)

<table>
<thead>
<tr>
<th>GOS</th>
<th>ETCO2 &lt; 30 mmHg</th>
<th>ETCO2 30 to 45 mmHg</th>
<th>ETCO2 &gt; 45 mmHg</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>23 (37.8%)</td>
<td>30 (30.0%)</td>
<td>2 (25.0%)</td>
<td>55 (32.5%)</td>
</tr>
<tr>
<td>Vegetative State</td>
<td>3 (4.9%)</td>
<td>0 (0%)</td>
<td>1 (12.5%)</td>
<td>4 (2.4%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>18 (29.5%)</td>
<td>25 (25.0%)</td>
<td>1 (12.5%)</td>
<td>44 (26.1%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>4 (6.6%)</td>
<td>14 (14.0%)</td>
<td>1 (12.5%)</td>
<td>19 (11.2%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>13 (21.3%)</td>
<td>31 (13.0%)</td>
<td>3 (37.5%)</td>
<td>47 (27.8%)</td>
</tr>
<tr>
<td>Total</td>
<td>61 (100%)</td>
<td>100 (100%)</td>
<td>8 (100%)</td>
<td>169 (100%)</td>
</tr>
</tbody>
</table>

#### 6.4.5 Discussion – Ventilation and End-Tidal Carbon Dioxide Readings within the In-Hospital Setting

Several studies have demonstrated the deleterious effects of hyperventilation in patients with brain injuries (Muizelaar et al., 1991; Brain Trauma Foundation, 2000a; Fortune et al., 1995; Raichle, 1970; Diringer et al., 2002; Crockard et al., 1973; Oertel et al., 2002;
Nolan et al., 2003; Davis, Dunford, Poste et al., 2004). In these studies it was determined that hyperventilation markedly reduces cerebral blood flow; however, hyperventilation does not consistently reduce intracranial pressure. Substantial decreases in cerebral blood flow leads to cerebral ischaemia and secondary brain injury due to cellular changes and a loss of autoregulation.

Davis, Heister, Poste et al. (2005) explored the effects of pre-hospital ventilation on ETCO2 levels in 76 patients and found that hyperventilation occurred in 79% of the patients. Moreover, severe hyperventilation occurred in 59% of patients. The mean duration of hyperventilation for hypocapnoeic patients was 6.5 minutes and for severely hypocapnoeic patients was 8.0 minutes. Interestingly, all patients received rapid sequence induction, endotracheal intubation and ETCO2 monitoring. The authors could not account for the underlying reasons for the hyperventilation.

Manley et al. (2000) in their study on haemorrhaged swine found that hyperventilation resulted in a 56% mean decrease in brain tissue oxygenation, whereas hyperventilation resulted in a 166% increase in brain tissue oxygenation (p = 0.001). It was concluded that hyperventilation exacerbated brain injury by increasing brain tissue hypoxia through cerebral vascular constriction, whereas hypoventilation alleviated brain tissue hypoxia. The findings suggest that hyperventilation has a deleterious effect on brain oxygenation in patients with haemorrhagic shock and head trauma.
The effect of pre-hospital manual ventilation of patients with severe brain injuries requires more investigation. The effect of pre-hospital ventilation rate and tidal volume may be a critical variable that has profound effect on the neurological outcome of persons with brain injuries. The consistency of ventilation during the pre-hospital management of brain injured patients can be affected by several factors including, the clinical experience of the paramedic, distraction from the task of airway maintenance, the paramedic attempting to multi-task while ventilating the patient, and movement of the patient during transfer to the stretcher and the vehicle.

By tradition in Australian Ambulance Services, the paramedic who controls the airway is also in command of the overall resuscitation process. It is plausible to consider that paramedic multi-tasking and distraction and movement of the patient could be a major contributor to inadvertent hyperventilation. As such, consideration should be given to the adjustment of the operational practice where the airway paramedic commands the overall resuscitation process and interventions. Airway maintenance and consistency of ventilation requires total focus and concentration. The paramedic who controls the airway should not be in command of the overall resuscitation process and should not attempt to multi-task.

6.4.6 Summary – Ventilation and End-Tidal Carbon Dioxide Readings within the In-Hospital Setting

Episodes of hypocapnoea were significantly associated with poorer outcomes following severe traumatic brain injury. The frequency of inadvertent hyperventilation in the pre-
hospital setting could not be determined in this study. The relationship between the knowledge and experience of the paramedic in airway management and ventilation and the outcome of patients with brain injuries is for the most part, unknown. Further investigation in this area is required to provide meaningful and consequential data.

While emergency department end-tidal carbon dioxide readings may not accurately reflect the pre-hospital ventilation and management of the patient, the possibility of inadvertent hyperventilation of patients in the pre-hospital phase of management may exist. The determination of inadvertent pre-hospital hyperventilation was not within the scope of this research. Further study would be required to investigate this phenomenon. Due to the deleterious effects of hypocapnoea, the introduction of end-tidal carbon dioxide measurement in the pre-hospital should be considered.

6.5 Airway Adjunct and Respiratory Infection

6.5.1 Results - Pre-Hospital Airway Adjunct Insertion and the Development of Respiratory Infection

Within the pre-hospital setting, airway adjuncts and suction equipment are routinely removed from sterile packaging and stored in a non-sterile environment, such as within an oxygen carry case or bag. The adjuncts may remain in a non-sterile environment for extended periods of time including days to weeks. Analysis reveals that non-sterile airway adjuncts may be contributing to the onset of respiratory infection in persons with severe traumatic head injury.
Airway adjuncts were used in 59% of the 326 patients \((n = 193)\). Of those 193 who received an airway adjunct 41.7% of the total cohort \((n = 136)\) developed a respiratory infection. Therefore a total of 70.5% of patients who received an airway adjunct in the pre-hospital setting developed a respiratory tract infection. The frequency of use of airway adjuncts in the pre-hospital setting is outlined in Table 39. For those patients who did not receive an airway adjunct in the pre-hospital setting \((n = 133)\) 45% \((n = 60)\) developed a respiratory infection within the in-hospital setting.

### Table 39  Frequency of use of Airway Adjuncts in the Pre-Hospital Setting

<table>
<thead>
<tr>
<th>Airway Adjunct</th>
<th>Frequency</th>
<th>Percent (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nil</td>
<td>114</td>
<td>35.0%</td>
</tr>
<tr>
<td>Oral</td>
<td>114</td>
<td>35.0%</td>
</tr>
<tr>
<td>Nasal</td>
<td>28</td>
<td>8.6%</td>
</tr>
<tr>
<td>ETT</td>
<td>51</td>
<td>15.6%</td>
</tr>
<tr>
<td>Missing</td>
<td>19</td>
<td>5.8%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>326</strong></td>
<td><strong>100.0%</strong></td>
</tr>
</tbody>
</table>

Stepwise linear regression showed the onset of respiratory infection to be a weak but significant predictor of outcome using the GOS as the dependent variable \(r = -0.140, p = 0.012, 95\% CI = -0.816 to -0.103\); and the insertion of an airway adjunct in the pre-
hospital setting to be a weak but significant predictor of the onset of respiratory infection \((r = 0.147, p = 0.01, 95\% = 0.017 \text{ to } 0.123)\). Correlation analysis also demonstrated a significant relationship between the use of pre-hospital airway adjuncts and the onset of respiratory infection (Spearman’s rho = 0.131, \(p = 0.023\), 2-tailed).

Low GCS is thought to contribute to the onset of respiratory infection. Low GCS is associated with reduced capacity to maintain a clear airway and therefore secretions, blood or foreign materials may enter the airway of the unconscious or semi-conscious patient. Correlation analysis however failed to demonstrate a significant relationship between first pre-hospital GCS and the onset of respiratory infection (Spearman’s rho \(r = -0.036\), \(p = 0.517\), 2-tailed) and first pre-hospital GCS range (3-4, 5-7, 8-10,11-13,14-15) and the onset of respiratory infection (Spearman’s rho \(r = -0.60\), \(p = 0.283\), 2-tailed).

Further, correlation analysis also failed to demonstrate a significant relationship between airway obstruction on arrival at the emergency department and the onset of respiratory infection (Spearman’s rho \(r = -0.071\), \(p = 0.204\), 2-tailed).

The mean time of onset of respiratory infection was 3.56 days from injury (Standard Deviation 2.96 days), with minimum time being one day and maximum being 21 days from time of injury (data known for a subgroup of 131 patients). Identification of the onset of respiratory infection results are outlined in Table 40.
Table 40  Onset of Respiratory Infection (Days to Infection) and Pre-Hospital Airway Maintenance (Adjunct and Suction)

<table>
<thead>
<tr>
<th>Days to Infection</th>
<th>Frequency &amp; Percent</th>
<th>Pre-Hospital Airway Adjunct or Suction</th>
<th>Nil Pre-Hospital Airway Adjunct or Suction</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10 (7.6%)</td>
<td>8 (6.1%)</td>
<td>3 (2.3%)</td>
</tr>
<tr>
<td>2</td>
<td>71 (54.2%)</td>
<td>50 (38.2%)</td>
<td>21 (16.0%)</td>
</tr>
<tr>
<td>3</td>
<td>11 (8.4%)</td>
<td>7 (5.3%)</td>
<td>4 (3.1%)</td>
</tr>
<tr>
<td>4</td>
<td>8 (6.1%)</td>
<td>7 (5.3%)</td>
<td>1 (0.8%)</td>
</tr>
<tr>
<td>5</td>
<td>4 (3.1%)</td>
<td>3 (2.3%)</td>
<td>1 (0.8%)</td>
</tr>
<tr>
<td>6</td>
<td>5 (3.8%)</td>
<td>3 (2.3%)</td>
<td>2 (1.5%)</td>
</tr>
<tr>
<td>7</td>
<td>5 (3.8%)</td>
<td>3 (2.3%)</td>
<td>2 (1.5%)</td>
</tr>
<tr>
<td>8</td>
<td>7 (5.3%)</td>
<td>5 (3.8%)</td>
<td>2 (1.5%)</td>
</tr>
<tr>
<td>9</td>
<td>7 (5.3%)</td>
<td>6 (4.9%)</td>
<td>1 (0.8%)</td>
</tr>
<tr>
<td>10</td>
<td>1 (0.8%)</td>
<td>1 (0.8%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>14</td>
<td>1 (0.8%)</td>
<td>1 (0.8%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>21</td>
<td>1 (0.8%)</td>
<td>0 (0%)</td>
<td>1 (0.8%)</td>
</tr>
<tr>
<td>Total</td>
<td>131 (100%)</td>
<td>94 (71.8%)</td>
<td>37 (28.2%)</td>
</tr>
</tbody>
</table>

Airway interventions were undertaken in the pre-hospital setting in 71.8% of patients who developed respiratory infection. The vast majority of infections were identified within the first four days post-injury (76.3%). Of the 76.3%, 54.9% received a pre-
hospital airway intervention and 22.3% did not receive a pre-hospital airway intervention. The highest incidence of infection was detected on day two post-injury and 70.4% of those had received a pre-hospital airway intervention (refer to Table 40). It is possible that the incidence of infection was higher than recorded but death occurred prior to the onset or diagnosis of infection, during the incubation period. A total of 99 patients died within the first four days post-admission that had not been diagnosed with a respiratory infection. Death prior to infection onset or diagnosis may have potentially impacted on analysis and hence resulting in a weak correlation coefficient. The incidence of death within four days of injury is summarised in Table 41.

### Table 41 Incidence of Death for the Total Cohort and Incidence of Death Within 4 Days Post-Admission

<table>
<thead>
<tr>
<th>Death</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Died</td>
<td>136</td>
<td>41.7%</td>
</tr>
<tr>
<td>Total Died Within 4 Days of Admission</td>
<td>101</td>
<td>30.9%</td>
</tr>
<tr>
<td>Died Day 1</td>
<td>47</td>
<td>14.4%</td>
</tr>
<tr>
<td>Died Day 2</td>
<td>30</td>
<td>9.2%</td>
</tr>
<tr>
<td>Died Day 3</td>
<td>18</td>
<td>5.5%</td>
</tr>
<tr>
<td>Died Day 4</td>
<td>6</td>
<td>1.8%</td>
</tr>
</tbody>
</table>
Respiratory infection was associated with increased morbidity with 45.9% \( (n = 62) \) of patients with respiratory infection experiencing a poor outcome (vegetative state or severe disability) versus 7.6% of those who did not experience the onset of respiratory infection \( (n = 14) \). The mortality rate associated with respiratory infection was 21.5%. Cross-tabulation results of GOS and respiratory infection are presented in Table 42.

### Table 42 Respiratory Infection and Glasgow Outcome Scale

<table>
<thead>
<tr>
<th>GOS</th>
<th>Respiratory Infection</th>
<th>Nil Respiratory Infection</th>
<th>Not Documented</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>30 (22.1%)</td>
<td>107 (58.1%)</td>
<td>0 (0%)</td>
<td>137 (42.6%)</td>
</tr>
<tr>
<td>Vegetative State</td>
<td>14 (10.3%)</td>
<td>1 (0.5%)</td>
<td>0 (0%)</td>
<td>15 (4.7%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>48 (35.3%)</td>
<td>13 (7.1%)</td>
<td>1 (50.0%)</td>
<td>62 (19.3%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>18 (13.2%)</td>
<td>10 (5.4%)</td>
<td>0 (0%)</td>
<td>28 (8.7%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>26 (19.1%)</td>
<td>53 (28.8%)</td>
<td>1 (50.0%)</td>
<td>80 (24.8%)</td>
</tr>
<tr>
<td>Total</td>
<td>136 (100%)</td>
<td>184 (100%)</td>
<td>2 (100%)</td>
<td>322 (100%)</td>
</tr>
</tbody>
</table>

The relationship between respiratory infection, the use of pre-hospital airway interventions and outcome are presented in Table 43.
Table 43  Respiratory Infection, Pre-Hospital Airway Intervention and Glasgow Outcome Scale

<table>
<thead>
<tr>
<th>GOS</th>
<th>Respiratory Infection</th>
<th>Pre-Hospital Airway Adjunct or Suction</th>
<th>Nil Pre-Hospital Airway Adjunct or Suction</th>
<th>Pre-Hospital Airway Adjunct or Suction Unknown</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>30 (22.1%)</td>
<td>24 (17.6%)</td>
<td>6 (4.4%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Vegetative State</td>
<td>14 (10.3%)</td>
<td>10 (7.4%)</td>
<td>2 (1.5%)</td>
<td>2 (1.5%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>48 (35.3%)</td>
<td>28 (20.6%)</td>
<td>13 (9.6%)</td>
<td>7 (5.2%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>18 (13.2%)</td>
<td>9 (6.6%)</td>
<td>8 (5.9%)</td>
<td>1 (0.7%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>26 (19.1%)</td>
<td>14 (10.3%)</td>
<td>11 (8.1%)</td>
<td>1 (0.7%)</td>
</tr>
<tr>
<td>Total</td>
<td>136 (100.0%)</td>
<td>85 (62.5%)</td>
<td>40 (29.4%)</td>
<td>11 (8.1%)</td>
</tr>
</tbody>
</table>

Correlation analysis demonstrated a significant relationship between the onset of respiratory infection and Glasgow Outcome Scale (Spearman’s rho = −0.164, p = 0.003, 2-tailed). However, much stronger correlations were found between the onset of respiratory infection and length of intensive care unit stay, total in-patient days, and total days intubated as described in Table 44.
Table 44  Respiratory Infection and Total Intensive Care Unit (ICU) Stay, Total In-Patient Days and Total Time Intubated – Correlation Analysis 2-Tailed

<table>
<thead>
<tr>
<th>Days</th>
<th>Days Range</th>
<th>Days Mean</th>
<th>Standard Deviation (Days)</th>
<th>Spearman's rho</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total ICU Days</td>
<td>1 to 96</td>
<td>9.1</td>
<td>10.75</td>
<td>−0.593</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Total In-Patient Days</td>
<td>1 to 176</td>
<td>18.84</td>
<td>22.88</td>
<td>−0.573</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Total Days Intubated</td>
<td>0 to 25</td>
<td>4.31</td>
<td>4.52</td>
<td>−0.614</td>
<td>p &lt; 0.001</td>
</tr>
</tbody>
</table>

Dependant variable – onset of respiratory infection

Stepwise linear regression revealed significant results between the onset of respiratory infection and total ICU stay, total in-patient days and total days intubated as presented in Table 45.

Table 45  Stepwise Linear Regression – Respiratory Infection and Total ICU Stay, Total In-Patient Days, Total Days Intubated and Outcome

<table>
<thead>
<tr>
<th>Days</th>
<th>Correlation Coefficient (r)</th>
<th>p Value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total ICU Days</td>
<td>−0.444</td>
<td>p &lt; 0.001</td>
<td>−0.027 to −0.017</td>
</tr>
<tr>
<td>Total In-Patient Days</td>
<td>−0.396</td>
<td>p &lt; 0.001</td>
<td>−0.011 to −0.006</td>
</tr>
<tr>
<td>Total Days Intubated</td>
<td>−0.528</td>
<td>p &lt; 0.001</td>
<td>−0.070 to −0.048</td>
</tr>
</tbody>
</table>

Dependent variable – Onset of Respiratory Infection
Independent samples t-test was used to compare means for respiratory infection and total ICU stay, total in-patient days and total days intubated. Results are presented in Table 46.

Table 46  Respiratory Infection and Mean ICU Days, In-Patient Days and Days Intubated

<table>
<thead>
<tr>
<th>Days</th>
<th>Respiratory Infection Yes/No</th>
<th>Number</th>
<th>Mean Days</th>
<th>Std Deviation</th>
<th>Std Error Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICU Days</td>
<td>Yes</td>
<td>135</td>
<td>13.30</td>
<td>12.293</td>
<td>1.058</td>
</tr>
<tr>
<td>ICU Days</td>
<td>No</td>
<td>182</td>
<td>3.56</td>
<td>5.606</td>
<td>.416</td>
</tr>
<tr>
<td>In-Patient Days</td>
<td>Yes</td>
<td>134</td>
<td>29.95</td>
<td>22.287</td>
<td>1.925</td>
</tr>
<tr>
<td>In-Patient Days</td>
<td>No</td>
<td>185</td>
<td>10.62</td>
<td>19.776</td>
<td>1.454</td>
</tr>
<tr>
<td>Days Intubated</td>
<td>Yes</td>
<td>125</td>
<td>7.22</td>
<td>4.4684</td>
<td>0.3997</td>
</tr>
<tr>
<td>Days Intubated</td>
<td>No</td>
<td>165</td>
<td>2.05</td>
<td>3.0152</td>
<td>0.2347</td>
</tr>
</tbody>
</table>

Those with respiratory infection demonstrated significantly longer mean ICU days (p < 0.001), total in-patient days (p < 0.001) and days intubated (p < 0.001). The types of microbial bacteria associated with respiratory infection in 133 cases are presented in Table 47.
Table 47  Microbial Bacteria Type Associated with Respiratory Infection in 133 Cases

<table>
<thead>
<tr>
<th>Microbial Bacteria Type</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unknown</td>
<td>41</td>
<td>30.8%</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>24</td>
<td>18.0%</td>
</tr>
<tr>
<td>Serratia Marcescens</td>
<td>1</td>
<td>0.8%</td>
</tr>
<tr>
<td>Staphylococcus</td>
<td>38</td>
<td>28.6%</td>
</tr>
<tr>
<td>Streptococcus</td>
<td>4</td>
<td>3.0%</td>
</tr>
<tr>
<td>Haemophilus Influenzae</td>
<td>10</td>
<td>7.5%</td>
</tr>
<tr>
<td>E. Coli</td>
<td>1</td>
<td>0.8%</td>
</tr>
<tr>
<td>Pseudomonas</td>
<td>3</td>
<td>2.3%</td>
</tr>
<tr>
<td>Acinetobacter</td>
<td>1</td>
<td>0.8%</td>
</tr>
<tr>
<td>MRSA</td>
<td>4</td>
<td>3.0%</td>
</tr>
<tr>
<td>Aspiration Pneumonia</td>
<td>6</td>
<td>4.5%</td>
</tr>
<tr>
<td>Total</td>
<td>133</td>
<td>100.0%</td>
</tr>
</tbody>
</table>
6.5.2 Discussion—Pre-Hospital Airway Adjunct Insertion and the Development of Respiratory Infection

The incidence of nosocomial infection originating in the pre-hospital setting is not well documented. Incubation periods for common acute respiratory infections vary considerably. Incubation periods for influenza B and influenza A are 0.6 and 1.4 days respectively (Lessler et al., 2009). Some forms of pneumonia have incubation periods of up to 3 weeks (Lee & Bishop, 2002).

Data analysis suggests that certain practices may be contributing to the incidence of pre-hospital acquired nosocomial infection. Removal of airway adjuncts and suction equipment from sterile packaging may be a potential source of contamination. A number of studies have found nosocomial infection to be associated with increased mortality and morbidity (Erbay et al., 2003; Helling et al., 1988). Bochicchio et al. (2002) and Karch et al. (1996) also established an association between pre-hospital airway interventions and the onset of respiratory tract infection.

Erbay et al. (2003) studied the incidence and risk factors associated with nosocomial infection in a Turkish university hospital during 2000 and 2001 and found that 26% patients acquired 225 different types of nosocomial infections and pneumonia was found to be particularly common (40.9% of patients). Similarly the current study found pneumonia to be common at 18.0%.
Helling et al. (1988) reviewed 82 patients with head injuries admitted to a level one trauma hospital in Kansas, USA for rates and cause of respiratory infection. It was suggested that due to depressed respiratory function, prolonged ventilator support with endotracheal intubation, and post-traumatic immunosuppression, patients with head injuries may be particularly vulnerable to respiratory sepsis. Of the 82 patients reviewed 41 (50%) experienced at least one infectious complication, with 34 of the 41 (83%) developing pulmonary infections. The average onset of pulmonary infections was 3.2 days following admission and 18 (22%) experienced the onset of pneumonia within two days of admission. The study assumed the infections were nosocomial in origin; however, the possibility of pre-hospital acquired infection was not considered. The current study found the average time to the onset of respiratory infection to be 3.56 days.

Bochicchio et al. (2002) in their randomised prospective study of 191 patients found patients with severe traumatic brain injury who were intubated in the field had longer intensive care unit and hospital stays (15.2 versus 11.7, p < 0.005 and 20.2 versus 16.7, p < 0.04 respectively) and significantly higher incidence of pneumonia (49% versus 32%), resulting in a relative risk 1.53 greater in the pre-hospital intubation group. The pre-hospital group also had a significantly greater mortality rate (23% versus 12.4%, p = 0.05, a risk of mortality of 1.85 times that of the hospital intubated group). The pre-hospital intubation group was also found to have a greater risk of mortality from respiratory failure-related complications. It was concluded that pre-hospital intubation was associated with higher rates of nosocomial infection.
Further to this, Karch et al. (1996) their retrospective study of 94 trauma patients found that pre-hospital intubation was three times more likely to be associated with the development of nosocomial pneumonia than was in-hospital intubation. Eckert et al. (2004) found in a retrospective study of 571 patients that pre-hospital intubation, severe head injury, shock, blunt force trauma and high Injury Severity Score were associated with the development of nosocomial pneumonia after trauma. Pre-hospital intubation was associated with significantly higher incidence of pneumonia (35% versus 23%, p = 0.048), and pneumonia was directly related to increased length of intensive care unit stay and total hospital stay (p < 0.001).

Data analysis in the current study found pre-hospital airway adjuncts were used in 193 of the 326 patients (59%). Respiratory infection was confirmed in 71.8% of patients who received an airway intervention in the pre-hospital environment. Analysis demonstrated that the use of pre-hospital airway interventions significantly increased the risk of the onset of respiratory infection. Respiratory infection significantly correlated with increased morbidity (p < 0.001), increased ICU stay (p < 0.001), increased total in-patients days (p < 0.001) and increased total days intubated (p < 0.001).

6.5.3 Summary – Pre-Hospital Airway Adjunct Insertion and the Development of Respiratory Infection

The current study found the use of pre-hospital airway adjuncts to be associated with higher risk for the development of respiratory infection. Determining causation of respiratory infection was not within the scope of this study, however the possibility of
contamination originating from non-sterile airway equipment should be considered. Anecdotally, removal of airway adjuncts from the packaging reduces the time to definitive airway management and is therefore thought to decrease time in the hypoxic state and potentially improve outcome. No evidence to support this practice was found in the literature. The current study has taken into account all airway adjuncts including endotracheal intubation, oropharyngeal airway, nasopharyngeal airway and suction equipment. However it was beyond this scope of this study to determine if the causation of respiratory tract infection was associated with interventions undertaken in the pre-hospital or in-hospital setting; or as a result of decreased ability of the patient to maintain and protect their airway (low GCS). Moreover, a combination of factors may be associated with the development of respiratory tract infection. As a precaution it is recommended that the practice of removing airway adjuncts from sterile packaging prior to use should cease and a review of current practice in airway management of patients with traumatic head injuries by ambulance paramedics should be considered. Further investigation is required to substantiate these findings and extrapolate to the pre-hospital clinical context.
CHAPTER SEVEN

HYPOTENSION AND INTRAVENOUS FLUID REPLACEMENT TO MAINTAIN A SYSTOLIC BLOOD PRESSURE GREATER THAN 90 mmHg

7.1 Introduction to the Chapter

Chapter Seven will present findings for research question number 3: patients with severe head injuries and hypotension will benefit from early intravenous fluid volume replacement to maintain a systolic blood pressure of greater than 90 mmHg. This chapter will present results and discussion relating to crystalloid and colloid administration and fluid volume replacement in hypotensive and normotensive patients in the pre-hospital setting. The above listed variables and their relationship to the depended variable (GOS) will be discussed (refer to Figure 1).

7.2 Pre-Hospital Fluid Administration and Patient Outcome

7.2.1 Results - Administration of Crystalloid and Colloid Fluids in the Pre-Hospital Setting

Episodes of hypotension (systolic blood pressure less than 90 mmHg) were noted in 26.4% of patients \((n = 86)\). In 0.9% of cases \((n = 3)\) it was unknown whether hypotension had occurred and in 72.7% \((n = 237)\) there were no documented episodes of hypotension. Intravenous access was performed on 79.5% of patients and fluid volume replacement was administered to 44.5% of patients \((n = 145)\). Haemaccel was administered to 22.4% \((n = 73)\) and Hartmann’s solution was administered to 23.6% of patients \((n = 77)\). A total of 1.5% of patients \((n = 5)\) received both Haemaccel and Hartmann’s solution. The
administration of fluid resuscitation was unknown in 0.6% of cases ($n = 2$). Fluid resuscitation was not undertaken in 54.9% of cases ($n = 179$).

Fluid resuscitation was not administered in 38.4% ($n = 33$) of the 86 patients presenting with an episode of pre-hospital hypotension. Fluid administration in hypotensive and non-hypotensive patients is presented in Table 48, and the administration of Haemaccel and Hartmann’s are presented in Table 49 and Table 50.

### Table 48 Fluid Administration in the Pre-Hospital Setting

<table>
<thead>
<tr>
<th>Pre-Hospital BP</th>
<th>Fluid Administration</th>
<th>Nil Fluid Administration</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP &lt; 90</td>
<td>53 (61.6%)</td>
<td>33 (38.4%)</td>
<td>86 (100.0%)</td>
</tr>
<tr>
<td>Systolic BP &gt; 90</td>
<td>92 (39.1%)</td>
<td>143 (60.9%)</td>
<td>235 (100.0%)</td>
</tr>
</tbody>
</table>

### Table 49 Administration of Haemaccel in the Pre-Hospital Setting

<table>
<thead>
<tr>
<th>Pre-Hospital BP</th>
<th>Haemaccel</th>
<th>Nil Haemaccel</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Hospital Systolic BP &lt; 90</td>
<td>36 (41.9%)</td>
<td>50 (58.1%)</td>
<td>86 (100.0%)</td>
</tr>
<tr>
<td>Pre-Hospital Systolic BP &gt; 90</td>
<td>37 (15.7%)</td>
<td>198 (84.3%)</td>
<td>235 (100.0%)</td>
</tr>
</tbody>
</table>
Table 50  Administration of Hartmann’s in the Pre-Hospital Setting

<table>
<thead>
<tr>
<th>Pre-Hospital Systolic BP</th>
<th>Hartmann’s</th>
<th>Nil Hartmann’s</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Hospital Systolic BP &lt; 90</td>
<td>19 (22.1%)</td>
<td>67 (77.9%)</td>
<td>86 (100.0%)</td>
</tr>
<tr>
<td>Pre-Hospital Systolic BP &gt; 90</td>
<td>58 (24.7%)</td>
<td>177 (75.3%)</td>
<td>235 (100.0%)</td>
</tr>
</tbody>
</table>

Mean volume of crystalloid infused was 620 ml (Std Deviation ± 437 ml) and mean volume of colloid infused was 401 ml (Std Deviation ± 274 ml).

Ambulance Service of NSW Protocols, Pharmacology and Procedures (1997) state that Haemaccel should be administered if two or more of the key signs of hypovolaemic shock are present: systolic BP < 90 mmHg, tachycardia (relative to age group); poor brain perfusion and/or poor skin perfusion. Hartmann’s may be administered in cases of hypovolaemia without the key signs of shock. According to the Ambulance Service of NSW Protocols, Pharmacology and Procedures (1997), all of the hypotensive patients should have received fluid resuscitation if an appropriate level of Ambulance Paramedic was present at the scene of the incident.

Results were unclear as to the benefit of fluid volume replacement in the pre-hospital setting. Episodes of pre-hospital hypotension were demonstrated as a significant predictor of outcome and linear regression analysis also demonstrated that the presence of hypotension on arrival at the emergency department was a significant predictor of poor outcome following severe traumatic head injury (refer to Table 51). However, the
administration of fluid volume replacement in the pre-hospital setting was not found to be a significant predictor of outcome (p = 0.742). The results of analysis are presented in Table 51.

**Table 51  Regression Analysis – The Relationship between Pre-Hospital and Emergency Department Hypotension, Pre-Hospital Fluid Volume Replacement and Patient Outcome (GOS)**

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Correlation Coefficient (r)</th>
<th>p-Value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Hospital hypotension (BP &lt; 90 mmHg)</td>
<td>0.250</td>
<td>p &lt; 0.001</td>
<td>0.538 to 1.342</td>
</tr>
<tr>
<td>Emergency Department Hypotension (BP &lt; 90 mmHg)</td>
<td>0.241</td>
<td>p &lt; 0.001</td>
<td>0.759 to 1.992</td>
</tr>
<tr>
<td>Pre-Hospital Fluid Volume Replacement</td>
<td>0.019</td>
<td>p = 0.742</td>
<td>n/a</td>
</tr>
</tbody>
</table>

Stepwise Linear Regression – Dependent variable – Glasgow Outcome Scale

Fluid administration in patients experiencing an episode of hypotension in the pre-hospital setting was inconsistent. A total of 83 patients experienced an episode of hypotension (systolic BP < 90 mmHg), which represented 25.5% of the total cohort. Cross-tabulation results relating to the administration of crystalloid and colloid fluids to hypotensive patients (systolic BP < 90 mmHg) in the pre-hospital setting are presented in Tables 52, 53 and 54.
Table 52  Colloid Replacement in Hypotensive Patients in the Pre-Hospital Setting (Systolic BP < 90 mmHg)

<table>
<thead>
<tr>
<th>Glasgow Outcome Scale</th>
<th>Haemaccel</th>
<th>Nil Haemaccel</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>25 (71.2%)</td>
<td>30 (62.5%)</td>
<td>55 (66.3%)</td>
</tr>
<tr>
<td>Persistent Vegetative State</td>
<td>1 (2.9%)</td>
<td>1 (2.1%)</td>
<td>2 (2.4%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>4 (11.5%)</td>
<td>5 (10.4%)</td>
<td>9 (10.8%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>1 (2.9%)</td>
<td>5 (10.4%)</td>
<td>6 (7.2%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>4 (11.5%)</td>
<td>7 (14.6%)</td>
<td>11 (13.3%)</td>
</tr>
<tr>
<td>Total</td>
<td>35 (100.0%)</td>
<td>48 (100.0%)</td>
<td>83 (100.0%)</td>
</tr>
</tbody>
</table>

Cross-tabulation

Generally, patients who did not receive colloids in the pre-hospital setting demonstrated improved outcomes (GOS 4 or 5) compared to those who did receive colloids. Poorer outcomes (GOS 1, 2 or 3) were demonstrated in 85.6% of patients receiving colloids versus 75.0% in those not receiving colloids. Improved outcomes were confirmed in 14.4% in those receiving colloids as opposed to 25.0% in those not receiving colloids (refer to Table 52). However there was a significant correlation found between Injury Severity Score and colloid administration (Spearman’s rho = −0.307, p < 0.001, 2-tailed).
A total of 90.1% (n = 64 of 71 patients) with an Injury Severity Scores of ≥ 25 received colloids.

**Table 53  Crystalloid Replacement in Hypotensive Patients in the Pre-Hospital Setting (Systolic BP < 90 mmHg)**

<table>
<thead>
<tr>
<th>Glasgow Outcome Scale</th>
<th>Hartmann’s</th>
<th>Nil Hartmann’s</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>15 (78.9%)</td>
<td>40 (62.5%)</td>
<td>55 (66.3%)</td>
</tr>
<tr>
<td>Persistent Vegetative State</td>
<td>0 (0%)</td>
<td>2 (3.1%)</td>
<td>2 (2.4%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>2 (10.5%)</td>
<td>7 (10.9%)</td>
<td>9 (10.8%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>1 (5.3%)</td>
<td>5 (7.8%)</td>
<td>6 (7.2%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>1 (5.3%)</td>
<td>10 (15.6%)</td>
<td>11 (13.3%)</td>
</tr>
<tr>
<td>Total</td>
<td>19 (100.0%)</td>
<td>64 (100.0%)</td>
<td>83 (100%)</td>
</tr>
</tbody>
</table>

Cross-tabulation

Similarly, patients who did not receive crystalloids in the pre-hospital setting demonstrated improved outcomes (GOS 4 or 5) compared to those who did receive crystalloids. Poorer outcomes (GOS 1, 2 or 3) were demonstrated in 89.4% of patients receiving crystalloids versus 76.5% in those not receiving crystalloids. Improved outcomes were confirmed in 10.6% in those receiving crystalloids as opposed to 23.4% in those not receiving crystalloids (refer to Table 53). Analysis found no significant
correlation between Injury Severity Score and crystalloid administration (Spearman’s rho = −0.037, p = 0.520, 2-tailed).

Table 54  Fluid Administration in the Pre-Hospital Setting and GOS

<table>
<thead>
<tr>
<th>Glasgow Outcome Scale</th>
<th>Fluids</th>
<th>Nil Fluids</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>65 (45.1%)</td>
<td>71 (40.1%)</td>
<td>136 (42.4%)</td>
</tr>
<tr>
<td>Persistent Vegetative State</td>
<td>9 (6.3%)</td>
<td>6 (3.4%)</td>
<td>15 (4.7%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>25 (17.4%)</td>
<td>36 (20.3%)</td>
<td>61 (19.0%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>12 (8.3%)</td>
<td>17 (9.6%)</td>
<td>29 (9.0%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>33 (22.9%)</td>
<td>47 (26.6%)</td>
<td>80 (24.9%)</td>
</tr>
<tr>
<td>Total</td>
<td>144 (100.0%)</td>
<td>177 (100.0%)</td>
<td>321 (100.0%)</td>
</tr>
</tbody>
</table>

Cross-tabulation

Patients who did not receive fluid resuscitation in the pre-hospital setting (crystalloid and/or colloid) demonstrated improved outcomes (GOS 4 or 5) compared to those who did receive fluid resuscitation. Poorer outcomes (GOS 1, 2 or 3) were demonstrated in 68.8% of patients receiving fluid resuscitation versus 63.8% in those not receiving fluid resuscitation. Improved outcomes were confirmed in 31.2% in those receiving fluid resuscitation as opposed to 36.2% in those not receiving fluid resuscitation (refer to Table 54).
Overall, the benefit of fluid administration in patients with severe brain injuries in the pre-hospital setting remains uncertain ($p = 0.570$). Correlation analysis failed to demonstrate improved outcomes in hypotensive patients who received pre-hospital fluid volume replacement (Spearman’s rho = 0.201; $p = 0.068$).

7.2.2 Results - Pre-Hospital Fluid Resuscitation of Hypotensive and Normotensive Patients

A complete data set containing statistics on GOS, first emergency department systolic blood pressure, pre-hospital systolic blood pressure and fluid administration is known for 318 patients. Episodes of pre-hospital hypotension occurred in 86 patients (26.4%) and fluid resuscitation was not undertaken in 33 of those 86 patients with pre-hospital hypotension (10.1% of the total cohort and 38.4% of hypotensive patients). Fluid resuscitation was administered in 52 cases of pre-hospital hypotension (16.4%). Of those who received pre-hospital fluid resuscitation, 18 patients (5.7%) remained hypotensive on arrival at the emergency department, while 11 patients (3.5%) who did not receive pre-hospital fluid resuscitation remained hypotensive on arrival at the emergency department.

Fluid administration appeared inconsistent with fluids being administered to a number of both hypotensive and normotensive patients. Conversely despite the treatment protocol indicating fluids should be administered, they were withheld or not administered in some hypotensive patients. Amongst this cohort, 26.1% ($n = 83$) of patients were identified as experiencing an episode of pre-hospital hypotension. Fluids were administered to 16.4% ($n = 52$) and fluid was not administered to 9.7% of patients ($n = 31$). Of those who
received pre-hospital fluid resuscitation; 5.7% \((n = 18)\) patients remained hypotensive on arrival at the emergency department, while 3.5% \((n = 11)\) did not receive pre-hospital fluid resuscitation remained hypotensive on arrival at the emergency department. The outcomes for hypotensive and normotensive patients receiving and not receiving pre-hospital fluid resuscitation are presented in Table 55.

Table 55  
Fluid Administration in the Pre-Hospital Setting to Hypotensive and Normotensive Patients

<table>
<thead>
<tr>
<th>Glasgow Outcome Scale</th>
<th>Hypotensive With Fluid Admin</th>
<th>Hypotensive Without Fluid Admin</th>
<th>T-Test p value</th>
<th>Normotensive With Fluid Admin</th>
<th>Normotensive Without Fluid Admin</th>
<th>T-Test p Value</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>38 (73.1%)</td>
<td>17 (54.8%)</td>
<td>0.015*</td>
<td>27 (29.4%)</td>
<td>53 (37.1%)</td>
<td>0.030*</td>
<td>135 (42.5%)</td>
</tr>
<tr>
<td>Persistent Vegetative State</td>
<td>1 (1.9%)</td>
<td>1 (3.2%)</td>
<td>0.421</td>
<td>8 (8.7%)</td>
<td>5 (3.5%)</td>
<td>0.010*</td>
<td>15 (4.7%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>6 (11.5%)</td>
<td>3 (9.7%)</td>
<td>0.702</td>
<td>19 (20.7%)</td>
<td>33 (23.1%)</td>
<td>0.839</td>
<td>61 (19.2%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>2 (3.8%)</td>
<td>4 (12.9%)</td>
<td>0.024*</td>
<td>10 (10.9%)</td>
<td>12 (8.4%)</td>
<td>0.425</td>
<td>28 (8.8%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>5 (9.7%)</td>
<td>6 (19.4%)</td>
<td>0.009*</td>
<td>28 (30.3%)</td>
<td>40 (27.9%)</td>
<td>0.493</td>
<td>79 (24.8%)</td>
</tr>
<tr>
<td>Total</td>
<td>52 (100.0%)</td>
<td>31 (100.0%)</td>
<td></td>
<td>92 (100.0%)</td>
<td>143 (100%)</td>
<td></td>
<td>318 (100.0%)</td>
</tr>
</tbody>
</table>

Cross-tabulation: frequency, per cent and t-test p values for hypotensive and normotensive patients.
*p-value significant at the 0.05 level
Stepwise linear regression revealed the withholding or non-administration of fluid volume replacement in the pre-hospital setting in hypotensive patients to be a predictor of improved outcome (GOS 4 or 5) ($r = 0.219$, $p = 0.049$, 95% CI = 0.004 to 1.362). Those who were hypotensive but did not receive pre-hospital fluid resuscitation had markedly higher rates of improved outcome 32.3% versus 13.5% respectively (t-test, $p = 0.001$). Hypotensive patients who received pre-hospital fluid resuscitation demonstrated higher mortality (73.1% vs 54.8%) (t-test, $p = 0.015$).

Pre-hospital fluid administration was carried out in 129 cases (39.6% of the total cohort) where the systolic blood pressure was greater than 90 mmHg. The administration of fluid resuscitation in the pre-hospital setting was not consistent with the Ambulance Service of NSW treatment protocol for hypovolaemia with key signs of severe shock (Ambulance Service of New South Wales, 1997). A significant correlation was not demonstrated between pre-hospital the systolic blood pressure and the administration of fluid volume replacement in the pre-hospital setting (Spearman’s rho = $-0.085$, $p = 0.443$, 2-tailed). Colloid solution (Haemaccel) was administered in 15.7% of cases where systolic blood pressure was greater than 90 mmHg ($n = 37$ cases) and crystalloid solution administered in 24.7% of cases where systolic blood pressure was greater than 90 mmHg ($n = 58$ cases). The mean volume of colloid infused was 401 mL (Std Deviation ± 274 ml) and the mean volume of crystalloid fluid infused was 620 mL (Std Deviation ± 437 ml).
7.2.3 Discussion – Pre-Hospital Fluid Administration and Patient Outcome

Analysis failed to demonstrate improved outcomes in persons with severe traumatic head injury who received pre-hospital fluid resuscitation. Outcomes for those who received pre-hospital fluid resuscitation were poorer than those who did not receive fluid resuscitation, regardless of the presence or absence of hypotension (refer to Table 76). While a significant correlation was found between Injury Severity Score and the administration of colloids (p < 0.001), no significant correlation was found between Injury Severity Score and the administration of crystalloids (p = 0.520). Overall, those who did not receive pre-hospital fluid resuscitation demonstrated improved outcomes when compared with those who did receive fluids.

A systolic blood pressure of 90 mmHg has been assigned as a resuscitation end-point in adults. It is unknown if systolic pressures of greater than 90 mmHg would be desirable during both the pre-hospital and in-hospital resuscitation phase (Brain Trauma Foundation, 2000a). To date, no studies have been undertaken to investigate the optimal upper limit of systolic blood pressure during resuscitation of patients with brain injuries. However, Greaves et al. (2002) recommend that over-infusion be avoided in order to forestall exacerbation of cerebral oedema and deleterious alterations in cerebral blood flow. Greaves et al. (2002) recommend fluids be titrated in small boluses of 250 mL until a radial pulse is restored. Palpation of a radial pulse may indicate an adequate mean arterial pressure has been achieved. The Brain Trauma Foundation (2000b, 2007) suggests that it is important that paramedics aim to prevent systolic blood pressure from dropping below 90 mmHg in the pre-hospital setting. The optimal blood pressure in cases
of severe traumatic brain injury has not yet been established (Brain Trauma Foundation, 2007).

Studies by Chi et al. (2005), Stocchetti et al. (1996), Manley et al. (2001) and Bourma et al. (1991) all determined episodes of hypotension in the pre-hospital setting to be deleterious to outcome in persons with traumatic injuries. Maintenance of cerebral blood flow and mean arterial pressure is required to avoid the damaging effects of cerebral ischaemia. Bickell et al. (1994) in a prospective study of patients with penetrating torso injuries and low (< 90 mmHg) systolic blood pressure (n = 598) found delayed fluid resuscitation to be associated with improved outcome. The group receiving delayed fluid resuscitation had a lower mortality rate with 70% surviving to discharge as compared with the immediate resuscitation group with only 62% surviving to discharge (x², p = 0.04). Also within the delayed fluid resuscitation group there was a lower incidence of adult respiratory distress syndrome, sepsis syndrome, acute renal failure, coagulopathy, wound infection and pneumonia (23% vs 30% for the immediate fluid resuscitation group) (x², p = 0.08). The benefit of pre-hospital fluid resuscitation was not demonstrated in individuals with penetrating torso injuries (Bickell et al., 1994).

In the current study, episodes of hypotension (systolic blood pressure less than 90 mmHg) in the pre-hospital setting and hypotension on arrival at the emergency department were significant predictors of poorer outcome (r = 0.250, p < 0.001, 95% CI = 0.538 to 1.342 and r = 0.241, p < 0.001, 95% CI = 0.759 to 1.993, respectively). Hypotensive patients who received pre-hospital fluid resuscitation demonstrated
significantly higher mortality than those hypotensive patients who did not receive fluid resuscitation (73.1% vs 54.8%) (p = 0.015). Further, improved outcomes (moderate disability and good recovery) were noted in 13.5% of patients (n = 7) with pre-hospital hypotension and who received fluid resuscitation as opposed to 32.3% of patients (n = 10) with pre-hospital hypotension and who did not received fluid resuscitation (p = 0.001).

7.2.4 Summary – Pre-Hospital Fluid Administration and Patient Outcome

Analysis failed to support the hypothesis that patients with severe head injuries and hypotension will benefit from early intravenous fluid volume replacement to maintain a systolic blood pressure of greater than 90 mmHg. However, no significant relationship was found between pre-hospital systolic blood pressure and the administration of fluid volume replacement. A total of 92 patients received fluids but were not hypotensive. Further research is required to investigate the efficacy of fluid volume resuscitation in hypotensive patients with severe traumatic head injury.
CHAPTER EIGHT

TIME IN THE PRE-HOSPITAL SETTING AND TIME TO CRANIOTOMY

8.1 Introduction to the Chapter

Chapter Eight will present results and discussion on the data analysis for research question number 4: functional outcome of patients with head injuries is inversely related to pre-hospital times, time at scene, the total time in the pre-hospital setting and time to neurosurgical intervention (craniotomy): as pre-hospital time and time to craniotomy increase, functional outcome decreases. This chapter will present results and discussion relating to response time, time at scene, treatment time, transport time, total time in the pre-hospital setting and time to craniotomy. The above listed variables and their relationship to the depended variable (GOS) will be discussed (refer to Figure 1).

8.2 The Relationship between Pre-Hospital Time and Patient Outcome (GOS)

8.2.1 Results - Pre-Hospital Response Time, Scene Time, Treatment Time, Transport Time and Patient Outcome

The Glasgow Outcome Scale (GOS) was used as the dependent variable to investigate the effect of pre-hospital times on functional outcomes of persons with severe traumatic brain injury. Stepwise linear regression found that a scene time less than or equal to 10 minutes was a weak but significant predictor of outcome ($r = -0.122$, $p = 0.036$, 95% CI = $-1.024$ to $-0.023$). Two-tailed correlation analysis also demonstrated the significance of scene times less than or equal to 10 minutes to outcome determined by the Glasgow Outcome
Scale (Spearman’s rho = −0.126, p = 0.031, 2-tailed). Response time, scene time, treatment time, transport time, total time in the pre-hospital setting and time to neurosurgical intervention results are presented in Table 56.

Table 56  **Response Time, Scene Time, Treatment Time, Transport Time, Total Pre-Hospital Time and Time to Neurosurgical Intervention**

<table>
<thead>
<tr>
<th>Times</th>
<th>Number</th>
<th>Mean Time (Minutes)</th>
<th>Std Deviation (Minutes)</th>
<th>Minimum (Minutes)</th>
<th>Maximum (Minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Response</td>
<td>296</td>
<td>8.8</td>
<td>4.8</td>
<td>1</td>
<td>35</td>
</tr>
<tr>
<td>Scene</td>
<td>296</td>
<td>21.3</td>
<td>12.0</td>
<td>3</td>
<td>77</td>
</tr>
<tr>
<td>Treatment</td>
<td>296</td>
<td>30.9</td>
<td>13.2</td>
<td>6</td>
<td>84</td>
</tr>
<tr>
<td>Transport</td>
<td>298</td>
<td>10.0</td>
<td>5.8</td>
<td>1</td>
<td>37</td>
</tr>
<tr>
<td>Total Pre-Hospital</td>
<td>295</td>
<td>40.0</td>
<td>15.3</td>
<td>12</td>
<td>100</td>
</tr>
<tr>
<td>Neurosurgical</td>
<td>70</td>
<td>805.9</td>
<td>3352.2</td>
<td>53</td>
<td>27465</td>
</tr>
</tbody>
</table>

Missing = 30

Response time (r = 0.004, p = 0.997), treatment time (r = −0.077, p = 0.537), transport time (r = 0.111, p = 0.372), total time in the pre-hospital setting (r = −0.017, p = 0.892) and time to neurosurgical intervention (r = −0.021, p = 0.866) failed to demonstrate significant results in predicting outcome. Response time was further subdivided into response time less than or equal to 5 minutes (r = 0.088, p = 0.479) and response time less than or equal than 10 minutes (r = 0.079, p = 0.523), which also failed to demonstrate
significant results in a stepwise linear regression analysis. A response time of less than or equal to 5 minutes was achieved in only 18.4% of cases ($n = 60$).

Significant results were identified when scene time was further categorised in groupings of scene time less than or equal to 10 minutes versus scene time greater than 10 minutes. A scene time of less than or equal to 10 minutes was a weak but significant predictor of improved outcome ($r = -0.122$, $p = 0.036$, 95% CI = $-1.024$ to $-0.023$). A summary of scene time less than or equal to 10 minutes and greater than 10 minutes is outlined in Table 57.

### Table 57  
Scene Time Less Than or Equal to 10 Minutes, Scene Time Greater Than 10 Minutes and GOS

<table>
<thead>
<tr>
<th>GOS</th>
<th>Scene Time Less Than or Equal to 10 minutes</th>
<th>Scene Time Greater Than 10 minutes</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>18 (34.6%)</td>
<td>111 (46.1%)</td>
<td>129 (44.0%)</td>
</tr>
<tr>
<td>Vegetative State</td>
<td>3 (5.8%)</td>
<td>10 (4.1%)</td>
<td>13 (4.4%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>6 (11.5%)</td>
<td>46 (19.1%)</td>
<td>52 (17.7%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>6 (11.5%)</td>
<td>19 (7.9%)</td>
<td>25 (8.5%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>19 (36.6%)</td>
<td>55 (22.8%)</td>
<td>74 (25.3%)</td>
</tr>
<tr>
<td>Total</td>
<td>52 (100.0%)</td>
<td>241 (100.0%)</td>
<td>293 (100%)</td>
</tr>
</tbody>
</table>

Missing = 33
When the percentage of patients with a scene time of less than or equal to 10 minutes is compared with a scene time of greater than 10 minutes, those cases with a scene time of less than or equal to 10 minutes attained a more favourable outcome in all GOS categories with the exception of persistent vegetative state (refer to Table 57) (Spearman’s rho = 0.140, p = 0.016, 2-tailed).

A poor outcome (death, vegetative state or severe disability) was observed in 51.9% of patients \((n = 27)\) when scene time was less than or equal to 10 minutes Whereas when scene time was greater than 10 minutes, poor outcomes were observed in 69.4% of patients \((n = 167)\) (Spearman’s rho = -0.140, p = 0.016, 2-tailed). Patients demonstrated improved outcomes (moderate disability or good recovery) when scene time was less than or equal to 10 minutes when compared to those where scene time was greater than 10 minutes \((48.1\%, n = 25 \text{ versus } 30.7\%, n = 74 \text{ respectively})\) (Spearman’s rho = 0.138, p = 0.018, 2-tailed).

Regression analysis failed to find a significant relationship between the first pre-hospital GCS and systolic blood pressure less than or equal to 90 mmHg for both scene time of less than or equal to 10 minutes and transport time to hospital as presented in Table 58.
Scene time was less than or equal to 10 minutes in only 17.7% \((n = 52)\) of cases whereas scene time exceeded 10 minutes in 82.3% of cases \((n = 241)\). A low GCS and/or pre-hospital hypotension are considered to be indicators of significant injury (Ambulance Service of New South Wales, 1997, 2007) and patients presenting with such parameters require urgent transport to an area trauma facility. However, a trend of dual response was noted in the results which may be contributing to increased scene time. In 53.4% of cases \((n = 174)\) an ambulance crew of two paramedics was responded to the scene and waited at the scene for the arrival of a second crew. In 50.3% of cases \((n = 164)\) the first crew on scene was non-intensive care. In 70.1% of cases dual response, the second crew was an intensive care crew \((n = 122)\). Spearman correlation identified a significant relationship between the level of clinical response of the first crew to arrive at the scene and the subsequent clinical level of the second crew to arrive (Spearman’s rho = 0.297, \(p = 0.006\)). Results suggest that non-intensive care paramedics are waiting at the scene for the arrival of intensive care paramedics. Moreover, the presence of intensive care paramedics was not associated with improved outcomes.

\[\begin{array}{|c|c|c|}
\hline
\text{Independent Variable} & \text{Scene Time} \leq 10 \text{ mins} & \text{Transport Time to Hospital} \\
\hline
\text{First Pre-Hospital GCS} & r = 0.111, \ p = 0.088 & r = 0.101, \ p = 0.111 \\
\hline
\text{Systolic Blood Pressure} < 90 \text{ mmHg} & r = 0.078, \ p = 0.382 & r = -0.059, \ p = 0.350 \\
\hline
\end{array}\]
Stepwise linear regression revealed the presence of intensive care paramedics at the scene to be a significant predictor of scene time greater than or equal to 10 minutes \( (r = 0.198, p = 0.001, 95\% \text{ CI} = 0.087 \text{ to } 0.319) \). While analysis indicated that correlation coefficient was statistically significant, the correlation was relatively weak. The result may have been influenced by the disproportionate response by intensive care paramedics and non-intensive care paramedics. Intensive care paramedics were present at the scene and intervened in patient treatment in 82.8\% of cases \((n = 245)\) as opposed to 17.2\% of non-intensive care attendance only \((n = 53)\) for cases where the level of clinical response was known \((n = 298)\).

In 62\% of cases \((n = 203)\), transport time to hospital was less than or equal to 10 minutes. The mean transport time to hospital was 10.0 minutes; however the mean scene time was 21.3 minutes. Total mean time in the pre-hospital setting was 40.0 minutes which included response time, scene time and transport time (refer to Table 54). Time in the pre-hospital setting could be reduced if, where possible, the first crew to arrive at the scene were to immediately transport the patient and not wait for the arrival of an intensive care crew. The benefit of interventions provided by intensive care paramedics was not demonstrated in the current study. Reducing time to definitive treatment at hospital may potentially improve outcome following severe traumatic head injury, particularly as transport time was less than or equal to 10 minutes in the majority of cases.
8.2.2 Discussion – Pre-Hospital Response Time, Scene Time, Treatment Time, Transport Time and Patient Outcome

The current study investigated the issues of time in the pre-hospital setting and advanced life support interventions on the functional outcome of persons with severe traumatic injury and found time at the scene to be significant in influencing the outcome of persons with severe traumatic head injury. A scene time of less than or equal to 10 minutes was associated with not only reduced mortality and morbidity but also with improved outcomes. Total mean time in the pre-hospital setting was found to be 40.0 minutes and this may be influenced by dual response with non-intensive care paramedics waiting at the scene for the arrival of intensive care paramedics and therefore increasing time to definitive treatment within the hospital setting. Time at the scene averaged 21.3 minutes. While response times and transport times may be difficult to decrease due to a variety of variables such as the location of available paramedic crews, traffic conditions and the distance of appropriate hospitals from the scene, time at scene could be decreased by immediate transportation of the patient. The current study demonstrated no benefit from interventions provided by intensive care paramedics and therefore non-intensive care crews should not wait at the scene for intensive care paramedics. Persons with severe traumatic head injury should be transported as soon as possible to an area trauma hospital and advanced life support interventions, if required, should be undertaken en-route. Scene time should not be extended for the undertaking of advanced life support interventions.

Similarly, other studies have found time in the pre-hospital setting to be an important variable in the outcome of persons with severe traumatic head injury. Rainer et al. (1997)
in their prospective study of trauma patients admitted to an accident and emergency department in an Edinburgh hospital found that on-scene times were significantly longer when ALS skills were undertaken, including intravenous cannulation, fluid administration and/or tracheal intubation \( (x^2, p < 0.001) \). For patients who received no ALS interventions, scene time was significantly shorter (18 minutes for paramedics vs 15 minutes for technicians; \( x^2, p < 0.01 \)). Moreover, improved outcome following ALS interventions was not established. There was no significant reduction in mortality or length of stay in the intensive care unit for those patients who received ALS interventions in the pre-hospital setting (Rainer et al., 1997).

In their studies of pre-hospital times, Harvey et al. (1998) and Demetriades et al. (1996) found mean time to definitive treatment to be 34.9 and 37.0 minutes respectively. Demetriades et al. (1996) compared paramedic versus private transportation of persons with severe traumatic injuries and found mean scene time for the paramedic group to be 22.5 minutes. In the absence of extended scene time and advanced life support interventions, the non-paramedic group reached definitive treatment 30 minutes prior to the paramedic group. Mortality was found to be significantly higher in the paramedic group with an adjusted mortality rate of 28.2% in the paramedic group versus 17.9% in the private group \( (p < 0.001) \). It was estimated that for every 10 minutes delay in receiving definitive treatment, survival drops by 10% (Demetriades et al., 1996). Morbidity rates were also found to increase when pre-hospital time was extended. Regal et al. (1997) retrospectively studied 1,223 multi-trauma patients and found that there was
a significantly higher rate of delayed complications including multiple organ failure in patients with a rescue time in excess of 30 minutes.

8.2.3 Summary—Pre-Hospital Response Time, Scene Time, Treatment Time, Transport Time and Patient Outcome

The current study demonstrated on-scene time of less than or equal to 10 minutes to be associated with improved neurological outcomes following severe traumatic head injury. This finding is supported in the studies of Harvey et al. (1998) and Demetriades et al. (1996) and Regal et al. (1997). It is recommended that, where possible, paramedics limit scene time to 10 minutes or less and expedite transportation of persons with severe traumatic head injury to an area trauma facility.

8.3 Time to Neurosurgical Intervention, Intracranial Pressure, Direct Trauma Centre Admission, Type of Intracranial Injury and Patient Outcome

8.3.1 Results - Time to Neurosurgical Intervention and Patient Outcome

Analysis failed to establish time to neurosurgical intervention as a significant predictor of outcome; however, intracranial pressure greater than 40 mmHg was found to be a strong predictor of outcome (refer to Table 59). Two-tailed correlation found ICP greater than 40 mmHg to be significant at the 0.01 level (Spearman’s rho = 0.474; p < 0.001).
Table 59  
**Stepwise Linear Regression - Intracranial Pressure, Minutes to Craniotomy and GOS**

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Correlation Coefficient (r)</th>
<th>p Value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intracranial Pressure &gt; 40 mmHg</td>
<td>0.640</td>
<td>p &lt; 0.001</td>
<td>1.322 – 2.545</td>
</tr>
<tr>
<td>Minutes to Craniotomy</td>
<td>-0.044</td>
<td>p = 0.661</td>
<td>n/a</td>
</tr>
</tbody>
</table>

Dependent variable – GOS

Results for intracranial pressure less than and greater than 40 mmHg and GOS are presented in Table 60.

Table 60  
**Intracranial Pressure and GOS**

<table>
<thead>
<tr>
<th>GOS</th>
<th>ICP Less than 40 mmHg</th>
<th>ICP Greater than 40 mmHg</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>10 (13.3%)</td>
<td>46 (52.3%)</td>
<td>56 (34.3%)</td>
</tr>
<tr>
<td>Vegetative State</td>
<td>4 (5.4%)</td>
<td>10 (11.4%)</td>
<td>14 (8.6%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>25 (33.3%)</td>
<td>20 (22.7%)</td>
<td>45 (27.6%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>13 (17.3%)</td>
<td>5 (5.7%)</td>
<td>18 (11.0%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>23 (30.7%)</td>
<td>7 (7.9%)</td>
<td>30 (18.4%)</td>
</tr>
<tr>
<td>Total</td>
<td>75 (100.0%)</td>
<td>88 (100.0%)</td>
<td>163 (100.0%)</td>
</tr>
</tbody>
</table>
A total of 79 patients underwent surgical craniotomy for the evacuation of a mass intra-cranial lesion (24.2% of the total cohort and 48.5% of those with intra-cranial pressure monitoring). Time to craniotomy was known for 70 of the 79 patients. Mean time to neurosurgical intervention was 805.9 minutes or 13.4 hours (Std Deviation = 3,352.2 minutes). Minimum time to craniotomy was 53 minutes and maximum time was 27,465 minutes, however this surgery was to evacuate a chronic subdural haemorrhage which had collected 7.6 days post-admission.

For patients requiring surgical removal of a mass intra-cranial lesion, craniotomy within one hour from time of injury was achieved in only 2.9% cases (n = 2). Craniotomy within two hours from injury was achieved in 11.4% of cases (n = 8) and craniotomy within three hours from injury was achieved in 44.3% of cases (n = 31). Outcome for those patients who received craniotomy with 3 hours from injury are presented in Table 61.
Table 61  Neurosurgical Intervention (Craniotomy) and GOS

<table>
<thead>
<tr>
<th>GOS</th>
<th>Craniotomy Less Than 180 mins</th>
<th>Craniotomy Greater than 180 mins</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>16 (50.0%)</td>
<td>12 (31.6%)</td>
<td>28 (40.0%)</td>
</tr>
<tr>
<td>Vegetative State</td>
<td>2 (6.3%)</td>
<td>6 (15.8%)</td>
<td>8 (11.4%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>5 (15.6%)</td>
<td>9 (23.7%)</td>
<td>14 (20.0%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>3 (9.4%)</td>
<td>5 (13.2%)</td>
<td>8 (11.4%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>6 (18.8%)</td>
<td>6 (15.8%)</td>
<td>12 (17.1%)</td>
</tr>
<tr>
<td>Total</td>
<td>32 (100.0%)</td>
<td>38 (100.0%)</td>
<td>70 (100%)</td>
</tr>
</tbody>
</table>

No significant correlation was found between craniotomy within three hours from injury and GOS (Spearman’s rho = 0.105, p = 0.387, 2-tailed). A significant correlation was found between ISS and ICP (Pearson correlation = 0.203, p = 0.01), indicating that high injury severity is associated with higher ICP. Significant correlations were not demonstrated for improved outcomes where craniotomy was performed within 3 hours from injury (GOS 4 or 5) (Spearman’s rho = −0.113, p = 0.406, 2-tailed).
8.3.2 Discussion – Time to Neurosurgical Intervention and Patient Outcome

The studies of Rose, Valtonen and Jennet (1977) and Lind, Heppner, Robins, and Mee (2005) established a relationship between time to definitive neurosurgical intervention and outcome in persons with severe traumatic head injury. Results of both studies suggested that a delay to definitive neurosurgical intervention was associated with adverse outcome. Wright et al. (1996) advocated a reduction in time to craniotomy has been shown to result in improved neurological outcome.

The current study failed to support the hypothesis that that functional outcome of patients with head injuries is inversely related to time in the pre-hospital setting and time to neurosurgical intervention (craniotomy); as pre-hospital time and time to craniotomy increases, functional outcome decreases. The current study found no significant relationship between the onset of severe traumatic head injury and time to definitive neurosurgical intervention. However, the benefit of early evacuation of a mass intracranial lesion cannot be discounted even though significant results were not demonstrated. The majority of patients requiring neurosurgical intervention where time to craniotomy was recorded \( n = 70 \) experienced delay to craniotomy. A total of 55.7\% \( n = 39 \) patients did not receive neurosurgical intervention within three hours from injury. Similarly, Lind et al. (2005) in their study found the majority of patients did not receive neurosurgical intervention within the recommended four hours from the time of injury. Time to craniotomy may indeed be an important variable in outcome following severe traumatic head injury, however any benefit associated with early neurosurgical
intervention may not become apparent until larger cohorts of severely head injured patients receive craniotomy within one to two hours from the time of injury.

The extremely small number of cases in the current study (2.9%, n = 2) where time to craniotomy was less than 60 minutes may have resulted in type II error. In the two cases that did receive craniotomy within one hour from injury, one patient had a good recovery while the other patient died. A larger cohort of patients undergoing craniotomy within one hour from injury would be required to undertake further meaningful analysis in the area of time to neurosurgical intervention.

8.3.3 Summary – Time to Neurosurgical Intervention and Patient Outcome

The current study has found that time to definitive neurosurgical intervention remains long. Further study is required to identify factors that may contribute to delays. Time to computed tomography, other imaging and time within the emergency department was not considered in this study. Further analysis of these factors may provide additional data which may assist in reducing time to neurosurgical intervention. It is recommended that paramedics limit scene time to less than 10 minutes when possible and transport persons with severe traumatic head injury directly to a neurosurgical facility in order to reduce delays to definitive neurosurgical intervention.
8.3.4 Results - Level of Receiving Hospital and Patient Outcome

The level of receiving hospital was not found to be a significant predictor of outcome. Possible transport decisions for paramedics explored in this study included: a) nearest hospital which is an area trauma hospital, b) nearest hospital which is not an area trauma hospital or, c) bypass the local hospital for an area trauma hospital. The summary of transport decisions made in the pre-hospital setting is outlined in Table 62.

Table 62  Transport Decisions and Glasgow Outcome Scale

<table>
<thead>
<tr>
<th>Transport Decision</th>
<th>Death</th>
<th>Vegetative State</th>
<th>Severe Disability</th>
<th>Moderate Disability</th>
<th>Good Recovery</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
</tr>
<tr>
<td>Trauma Centre</td>
<td>88 (65.7%)</td>
<td>9 (60.0%)</td>
<td>35 (58.3%)</td>
<td>12 (46.2%)</td>
<td>52 (68.4%)</td>
<td>196 (63.0%)</td>
</tr>
<tr>
<td>District Hospital</td>
<td>11 (8.2%)</td>
<td>3 (20.0%)</td>
<td>8 (13.3%)</td>
<td>5 (19.2%)</td>
<td>3 (3.9%)</td>
<td>30 (9.6%)</td>
</tr>
<tr>
<td>Bypass to Trauma Centre</td>
<td>31 (23.1%)</td>
<td>3 (20.0%)</td>
<td>14 (23.3%)</td>
<td>8 (30.8%)</td>
<td>21 (27.7%)</td>
<td>77 (24.8%)</td>
</tr>
<tr>
<td>Medical Ambulance to Trauma Centre</td>
<td>4 (3.0%)</td>
<td>0 (0%)</td>
<td>3 (5.0%)</td>
<td>1 (3.8%)</td>
<td>0 (0%)</td>
<td>8 (2.6%)</td>
</tr>
<tr>
<td>Total</td>
<td>134 (100.0%)</td>
<td>15 (100.0%)</td>
<td>60 (100.0%)</td>
<td>26 (100.0%)</td>
<td>76 (100.0%)</td>
<td>311 (100.0%)</td>
</tr>
</tbody>
</table>

No correlation between the level of the initial receiving hospital and Glasgow Outcome score was evident (Spearman’s rho = −0.050, p = 0.369, 2-tailed). Nor was a significant correlation found for direct admission to a trauma centre and Glasgow Outcome Scale (Spearman’s rho = −0.032, p = 0.583, 2-tailed). The transport decision made by
ambulance paramedics was not a significant predictor of outcome (Spearman’s rho = 0.014, p = 0.803, 2-tailed).

Direct transportation from the scene to a district hospital occurred in 9.6% of cases (n = 30). All 30 patients were transferred to a trauma centre following initial treatment in the emergency department of the district hospital. A total of 12 of those 30 patients (40.0%) underwent neurosurgical intervention (craniotomy). Of those 12 patients only one received a craniotomy within three hours from the time of injury (8.3%) versus 30 of the 58 patients who were transported to an area trauma hospital (51.7%). Spearman correlation revealed a significant relationship between level of receiving hospital and time to craniotomy (Spearman’s rho = 0.258, p = 0.031). A pre-hospital GCS of 8 or less was documented in 29 of the 30 patients; with 17 of those having a GCS of 3. Pre-hospital guidelines indicate that all of the 30 patients should have been transported directly to an area trauma facility. Outcome of those patients transferred from district hospital to trauma centre are outlined in Table 63.
Table 63  Patients Transported from the Scene to a District Hospital – Neurosurgical Intervention Status and Outcome (GOS)

<table>
<thead>
<tr>
<th>GOS</th>
<th>Craniotomy</th>
<th>Nil Craniotomy</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>4 (33.3%)</td>
<td>7 (38.9%)</td>
<td>11 (36.7%)</td>
</tr>
<tr>
<td>Vegetative State</td>
<td>3 (25.0%)</td>
<td>0 (0%)</td>
<td>3 (10.0%)</td>
</tr>
<tr>
<td>Severe Disability</td>
<td>3 (25.0%)</td>
<td>5 (27.8%)</td>
<td>8 (26.7%)</td>
</tr>
<tr>
<td>Moderate Disability</td>
<td>2 (16.7%)</td>
<td>3 (16.7%)</td>
<td>5 (16.7%)</td>
</tr>
<tr>
<td>Good Recovery</td>
<td>0 (0%)</td>
<td>3 (16.7%)</td>
<td>3 (10.0%)</td>
</tr>
<tr>
<td>Total</td>
<td>12 (100.0%)</td>
<td>18 (100.0%)</td>
<td>30 (100%)</td>
</tr>
</tbody>
</table>

8.3.5 Discussion – Level of Receiving Hospital and Patient Outcome

A significant correlation was not found for direct admission to a trauma centre and Glasgow Outcome Scale (Spearman’s rho = −0.032, p = 0.583, 2-tailed). The transport decision made by ambulance paramedics was not a significant predictor of outcome (Spearman’s rho = 0.014, p = 0.803, 2-tailed). The result may be subject to type II error as 90.4% of patients were transported directly to a trauma centre as opposed to 9.6% being transported to a district hospital. The disproportionate number of patients in each category, trauma centre versus district hospital may have confounded results. Wright et al. (1997) suggest that outcome following severe traumatic brain injury is improved when
the patient is transported directly to a trauma centre as time to definitive treatment and neurosurgical intervention is reduced. Any benefit of direct transport to a trauma centre may not have been evident as a result of unequal group size.

The Ambulance Service of NSW (1997) Protocols, Pharmacology and Procedures state that any patient with severe single system injury, multiple system injury, central nervous system injury, vehicular crash greater than 60 Km/hour, major deformation of vehicle, fatal injury in the vehicle, person ejected from within or on the vehicle, fall from height greater than 5 metres or cyclist or pedestrian hit by vehicle travelling greater than 30 Km/hour should be transported directly to a trauma centre. Results suggest that ambulance paramedics were reasonably compliant with the trauma triage criteria as specified in the protocols. Criteria for direct transportation to a trauma centre were applicable to all patients entered into the study. Transportation directly to a trauma centre occurred in 90.4% of cases. Of the 30 patients (9.6%) who were transported to a district hospital, 17 had a GCS of 3. Further, of the 30 patients, 12 required neurosurgical intervention for the removal of a mass intracranial lesion. Poor outcomes (GOS 1, 2 or 3) were noted in 83.3% \( (n = 10) \) of those patients requiring craniotomy but transported directly to a district hospital. Moderate disability was observed in 16.7% of patients \( (n = 2) \). Moreover, no patient was found to have a good recovery.
8.3.6 Summary – Level of Receiving Hospital and Patient Outcome

Delay preceding evacuation of an intracranial haematoma has been found by Rose et al. (1977) to significantly increase the rate of mortality in patients with head injuries. Therefore it could be postulated that direct transportation to a neurosurgical facility may have benefited those 12 patients requiring craniotomy by decreasing time to definitive treatment. It is recommended that paramedics transport persons with severe traumatic head injury directly to a trauma facility.

8.3.7 Results - Type of Intracranial Injury and Patient Outcome

Subarachnoid haemorrhage, subdural haemorrhage and epidural haemorrhage were all significant predictors of outcome. Excluded variables were cerebral contusion, intracerebral haemorrhage, diffuse axonal injury and penetrating cranial wound. Results of type of injury and Glasgow Outcome Scale are presented in Table 64. A significant correlation was found between intracranial pressure greater than 40 mmHg and the presence of subdural and subarachnoid haemorrhage (Spearman correlation, 2-tailed; subdural haemorrhage \( r = 0.291, p < 0.001 \); subarachnoid haemorrhage \( r = 0.293, p < 0.001 \)).
Table 64  Stepwise Linear Regression – Type of Intracranial Injury and GOS

<table>
<thead>
<tr>
<th>Intracranial Injury Type</th>
<th>Correlation Coefficient (r)</th>
<th>p Value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subarachnoid</td>
<td>0.288</td>
<td>p &lt; 0.001</td>
<td>0.592 – 1.272</td>
</tr>
<tr>
<td>Subdural</td>
<td>0.267</td>
<td>p &lt; 0.001</td>
<td>0.262 – 0.953</td>
</tr>
<tr>
<td>Epidural</td>
<td>-0.142</td>
<td>p = 0.008</td>
<td>-1.117 to -0.177</td>
</tr>
<tr>
<td>Cerebral Contusion</td>
<td>-0.038</td>
<td>p = 0.535</td>
<td>n/a</td>
</tr>
<tr>
<td>Diffuse Axonal Injury</td>
<td>0.047</td>
<td>p = 0.859</td>
<td>n/a</td>
</tr>
<tr>
<td>Intra-cerebral Haemorrhage</td>
<td>0.032</td>
<td>p = 0.541</td>
<td>n/a</td>
</tr>
<tr>
<td>Penetrating Cranial Wound</td>
<td>0.047</td>
<td>p = 0.507</td>
<td>n/a</td>
</tr>
</tbody>
</table>

Stepwise Linear Regression – Dependent variable – GOS

8.3.8 Discussion – Type of Intracranial Injury and Patient Outcome

The implication of this finding for pre-hospital practice is uncertain. In the absence of computed tomography paramedics cannot be certain of the type of intracranial lesion. However, paramedics should be cognisant that certain lesions are associated with poorer outcomes and expedite persons with severe traumatic head injury to a neurosurgical facility where possible. An intracranial pressure of greater than 40 mmHg was found to be a significant predictor of outcome \( r = 0.640, p = 0.001, 95\% \text{ CI} = 1.322 \text{ to } 2.545 \) and
both subdural and subarachnoid haemorrhage was associated with ICP greater than 40 mmHg. Increased mortality and morbidity was observed in patients with ICP greater than 40 mmHg. A total of 28.2% of patients with an ICP greater than 40 mmHg died as opposed to only 6.1% of those with an ICP less than 40 mmHg. Similar rates of morbidity were demonstrated with 18.4% of those with an ICP greater than 40 mmHg experiencing poor outcome (vegetative state or severe disability) versus 17.8% with an ICP less than 40 mmHg.

8.3.9 Summary – Intracranial Injury and Patient Outcome

A relationship may exist between time in the pre-hospital setting and time to craniotomy for those patients whose ICP was greater than 40 mmHg at time of craniotomy. It is reasonable that those patients whose ICP is markedly raised on arrival at hospital would benefit from early decompressive craniotomy. However, as only 2.9% of craniotomies were performed within one hour from injury, the significance of this assumption is unproven and further research is required. A conceivable conjecture would be to limit time in the pre-hospital setting and expedite neurosurgical intervention post admission to hospital.
CHAPTER NINE

OTHER FINDINGS

9.1 Introduction to the Chapter

This chapter will briefly present results and discussion of age and gender variables and their relationship to the depended variable (GOS) will be discussed (refer to Figure 1).

9.2 Age and Gender

9.2.1 Results - Age and Functional Outcome

Age was found to be a significant predictor of outcome. Stepwise linear regression revealed age as a significant predictor of outcome when GOS was used as the dependent variable ($r = -0.298$, $p < 0.001$; 95% CI $-0.029$ to $-0.014$). A significant correlation was also found to exist between age and GOS (Pearson correlation $= -0.298$, $p < 0.001$, 2-tailed). Generally, as age increases, neurological outcome decreases. A marked increase in poorer outcome was noted in the over-55 age group. A total of 20.7% of patients ($n = 67$) fell into this category with 18.0% of the sample and 86.6% of the age group ($n = 58$) having a poor outcome (death, persistent vegetative state or severe disability). Only 2.3% of the sample and 13.4% of the age group had a good outcome (moderate disability or good recovery) ($n = 9$). Conversely, while the 16–35 year age group accounted for the majority of cases (46.1%, $n = 149$) and the greatest number of poorer outcomes (28.8% of the sample, 62.4% of the age group, $n = 93$), there were a higher percentage with good outcomes (17.3% of the sample, 37.6% of the age group, $n = 56$).
9.2.2 Discussion - Age and Functional Outcome

Similar results were found in studies by Vollmer, Torner, Jane et al. (1991), Ritchie, Cameron, Ugoni et al. (2000) and Masson, Thicoipe, Monke et al. (2003). All of the studies reviewed found advancing age to be a predictor of poorer outcome following severe traumatic head injury.

9.2.3 Summary - Age and Functional Outcome

Paramedics should be mindful that advancing age is associated with poorer outcome and as such, limit scene time and expedite those over 55 years of age and with severe traumatic head injury to definitive management at a trauma facility.

9.2.4 Results - Gender and Functional Outcome

Gender was not found to be a significant predictor of outcome in this study (Stepwise linear regression; $r = -0.049, p = 0.357$). Males were over represented in the cohort of the current study by a ratio of 2:1 (male $n = 222$ vs female $n = 101$). Gender and final Glasgow Coma score revealed a higher percentage of males in all GCS and GOS ranges, as outlined in Tables 65 and 66.
Table 65  Gender and Final Glasgow Coma Scale Range

<table>
<thead>
<tr>
<th>GCS</th>
<th>3-4</th>
<th>5-7</th>
<th>8-10</th>
<th>11-13</th>
<th>14-15</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>87(26.9%)</td>
<td>5 (1.5%)</td>
<td>12 (3.7%)</td>
<td>21 (6.5%)</td>
<td>97 (30.0%)</td>
<td>222 (68.7%)</td>
</tr>
<tr>
<td>Female</td>
<td>49 (15.2%)</td>
<td>2 (0.6%)</td>
<td>8 (2.5%)</td>
<td>3 (0.9%)</td>
<td>39 (12.1%)</td>
<td>101 (31.3%)</td>
</tr>
<tr>
<td>Total</td>
<td>136 (42.1%)</td>
<td>7 (2.2%)</td>
<td>20 (6.2%)</td>
<td>24 (7.4%)</td>
<td>136 (42.1%)</td>
<td>323 (100.0%)</td>
</tr>
</tbody>
</table>

Final outcome is unknown for three patients due to incomplete medical records due to inter-hospital transfer. One of these patients was female and two were male.

Table 66  Gender and Glasgow Outcome Scale

<table>
<thead>
<tr>
<th>GOS</th>
<th>Death</th>
<th>Vegetative State</th>
<th>Severe Disability</th>
<th>Moderate Disability</th>
<th>Good Recovery</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>87 (26.9%)</td>
<td>10 (3.1%)</td>
<td>49 (15.2%)</td>
<td>18 (5.6%)</td>
<td>58 (18.0%)</td>
<td>222 (68.7%)</td>
</tr>
<tr>
<td>Female</td>
<td>50 (15.5%)</td>
<td>5 (1.5%)</td>
<td>13 (4.0%)</td>
<td>11 (3.4%)</td>
<td>22 (6.8%)</td>
<td>101 (31.3%)</td>
</tr>
<tr>
<td>Total</td>
<td>137 (42.4%)</td>
<td>15 (4.6%)</td>
<td>62 (19.2%)</td>
<td>29 (9.0%)</td>
<td>80 (24.8%)</td>
<td>323 (100.0%)</td>
</tr>
</tbody>
</table>
9.2.5 Discussion – Gender and Functional Outcome

While the studies by Farace and Alves (2000), Kraus, Peek-Asa, McArthur et al. (2000) and Slewa-Younan, Green, Baguley et al. (2004) demonstrated that women display a poorer outcome than males, males were overly represented by a ratio of 2:1 or more. Slewa-Younan, Green, Baguley et al. (2004) suggest that for those studies that revealed significance between gender and outcome may be affected by type I error, due to uneven sample size and males being overly represented within the cohorts.

9.2.6 Summary – Gender and Functional Outcome

The results of this study suggest gender is not a predictor of functional outcome and no additional or specific pre-hospital interventions are required for females with severe traumatic head injury.
CHAPTER TEN

LIMITATIONS OF THE STUDY AND IMPLICATIONS FOR PARAMEDIC PRACTICE AND FUTURE RESEARCH

10.1 Limitations of the Study

The study design used was a retrospective cohort study. According to the Brain Trauma Foundation (2000a, 2007) retrospective cohort studies are considered to yield class III evidence and the degree of clinical certainty is not established. However, a preponderance of class III evidence can contribute to the development of a guideline and has been found to be useful for educational purposes and guiding future studies. Therefore a class III study is of most value when considered in conjunction with other class II and class III evidence.

The data collected in this study dates from 1999 to 2003, however, little has changed in the management and interventions provided by paramedics within the pre-hospital setting. The protocol for the management of head injury and the urgent transport protocol remain the same in the 1997 and the more recent 2007 edition of the Ambulance Service of NSW Protocols (Ambulance Service of New South Wales, 1997, 2007). No new pharmacological agents or interventions have been introduced since the data was collected. The data and results can be considered in relation to current practice. However, the findings of this study should not be considered a definitive composition but should be considered along with other studies and evidence to establish an immensity of data and
information to contribute to existing knowledge in the pre-hospital management of severe traumatic head injury.

Data was gathered from Ambulance Service of NSW and hospital documentation. As such the accuracy and completeness of the each record cannot be confirmed. Incomplete or inaccurate documentation may potentially affect findings. The study assumed the presence of a rigorous level of accuracy and completeness of documentation.

Large samples are usually less likely to incur error than smaller samples (Minichiello, Sullivan, Greenwood et al., 1999). While a larger sample size would have been preferable, the current sample of 326 patients was considered appropriate to yield a good statistical power and be representative of the population of persons with severe traumatic head injury in the Sydney and metropolitan area.

Injury severity score was collected and entered into the data base in this study. Stepwise linear regression analysis was used to evaluate the efficacy of advanced pre-hospital interventions on patient outcome (GOS) and ISS was entered into the analysis. However, analysis of subsets of patients with higher or lower injury severity was not undertaken in the study analysis. Therefore the full relationship between ISS and the efficacy of advanced pre-hospital interventions such as endotracheal intubation may not be fully understood which may be a limitation in this study. Further research is required in this area. A larger cohort size and comparison of patients with isolated brain injury versus
multi-system trauma may potentially provide further understanding of the impact of ISS on the benefit derived from advanced pre-hospital interventions.

10.2 Key Findings and Implications for Paramedic Practice

10.2.1 Review of Research Questions

Key findings and implications for paramedic practice have been extrapolated from the results of the current study. Implications are presented in sections 10.2.2 to 10.2.6. Key findings and implications relate to the research questions of this study which are as follows:

1. Persons with severe traumatic head injury will benefit from the attendance of intensive care paramedics and pre-hospital endotracheal intubation provided by intensive care paramedics.

2. Maintenance of a clear and patent airway and maintenance of oxygen saturation of greater than or equal to 95% will benefit patients with severe traumatic head injury.

3. Patients with severe head injuries and hypotension will benefit from early intravenous fluid volume replacement to maintain a systolic blood pressure of greater than 90 mmHg.

4. Functional outcome of patients with head injuries is inversely related to pre-hospital response time, time at scene, the total time in the pre-hospital setting and time to neurosurgical intervention (craniotomy): as pre-hospital time and time to craniotomy increases, functional outcome decreases.
10.2.2 Key Findings and Implications: Attendance of Intensive Care Paramedics and Endotracheal Intubation Provided by Intensive Care Paramedics

1. Stepwise linear regression failed to demonstrated improved outcomes for the level of clinical response \( (r = 0.003, p = 0.952) \) or the attendance of intensive care paramedics \( (r = -0.005, p = 0.931) \) at the scene and the Glasgow Outcome score of the patients. Time at scene was significantly increased with the attendance of intensive care paramedics \( (\text{Spearman’s rho} = -0.228, p < 0.001, \text{2-tailed}) \). Intensive care interventions and dual response to the scene was found to increase time at scene.

Pre-hospital Implications:

*In order to limit time at scene to less than 10 minutes, the first crew to arrive at the scene should, where possible, expedite the transportation of the patient to a trauma facility. Time at scene should not, if possible, be delayed for the arrival of intensive care paramedics. Intensive care interventions should be undertaken judiciously in the Sydney and metropolitan areas. Consideration should be given to withholding intensive care interventions if such interventions will result in scene time exceeding 10 minutes or where transport time to hospital is less than 10 minutes and there is no immediate life threat to the patient. Expedited transport to a trauma facility may be more beneficial for persons with severe traumatic head injury in the Sydney and metropolitan areas.*
2. Improved outcomes were not demonstrated in patients who received pre-hospital endotracheal intubation. Pre-hospital endotracheal intubation was significantly correlated to increased scene time (Spearman’s rho = −0.186, p = 0.001, 2-tailed). Scene time greater than 10 minutes was associated with poorer outcomes.

**Pre-hospital Implications:**

**Consideration should be given to withholding pre-hospital endotracheal intubation in persons with severe traumatic head injury in the Sydney and metropolitan areas if the airway, ventilation and oxygenation can be maintained adequately in the absence of endotracheal intubation. Patients should be expedited to a trauma facility for definitive management and scene time minimised to less than 10 minutes where possible.**

10.2.3 Key Findings and Implications: Maintenance of a Clear and Patent Airway and Oxygen Saturations Greater than 95%

1. Airway obstruction on arrival at the emergency department was observed in 25.2% of patients (n = 82). A weak correlation was demonstrated between a clear and patent airway on arrival at the emergency department and improved outcome (Spearman’s rho = −0.114, p = 0.042, 2-tailed). Stepwise linear regression revealed that airway obstruction was a significant predictor of SpO2 range (< 85%; 85-89%; 90-94%; 95-100%) on arrival at the emergency department (r = −0.247, p < 0.001, 95% CI = −0.728 to −0.266). Regression analysis also revealed SpO2 range on arrival at the emergency department to be a significant predictor
of outcome \( r = 0.202, p = 0.001, 95\% \text{ CI} = 0.159 \text{ to } 0.591 \). Poor outcome (GOS 1, 2 or 3) was demonstrated in 78.1\% of patients with an airway obstruction on arrival at the emergency department \( n = 64 \) versus 62.4\% of patients with a clear airway \( n = 148 \). Improved outcomes (GOS 4 or 5) were observed in 21.9\% of patients with an airway obstruction \( n = 18 \) versus 37.6\% of patients with a clear airway \( n = 89 \). An unrelieved airway obstruction in the pre-hospital setting was associated with higher morbidity (33.0\% vs 20.7\%) and higher mortality (45.1\% vs 41.8\%).

**Pre-hospital Implications:**

*Maintaining a patent airway is important in the management of persons with severe traumatic head injury. Consideration should be given for more frequent inspect and/or clearing of the airway in persons with severe traumatic head injury in order to minimise the incidence of airway obstruction.*

2. The current study found that 20.8\% of patients \( n = 58 \) were documented as having a SpO2 of less than 95\% on arrival at the emergency department. Stepwise linear regression revealed that initial emergency department SpO2 was a significant predictor of GOS \( r = 0.208, p = 0.001, 95\% \text{ CI} = 0.169 \text{ to } 0.599 \). Higher oxygen saturation range was associated with improved outcome with 39.0\% of patients with a SpO2 between 95-100\% \( n = 85 \text{ of } 218 \text{ patients} \) showing improved outcome (GOS 4 or 5). Whereas for those patients with a SpO2 < 95\%, only 24.1\% of patients demonstrated an improved outcome \( n = 14 \).
of 58 patients). It should be noted that pre-hospital pulse oximetry readings were documented in only 13.5% of the total cases ($n = 44$).

*Pre-hospital Implications:*

*Further research to investigate airway maintenance and manual ventilation by paramedics on persons with severe traumatic head injury to identify opportunities for improvement in airway maintenance, manual ventilation and oxygenation is required. Persons with severe traumatic head injury should have oxygen saturations maintained at 95% or greater during pre-hospital management and all ambulance vehicles should be equipped with pulse oximetry monitoring devices.*

3. Emergency department end-tidal carbon dioxide levels were found to be a weak but significant predictor of outcome ($r = 0.192$, $p = 0.013$, 95% CI = 0.008 to 0.066). Forty-five percent (45%) of normocapnoeic patients (PaCO$_2 = 30$ to 45 mmHg) were observed to have improved outcomes ($n = 45$) (GOS 4 or 5) versus 27.8% ($n = 17$) of hypocapnoeic patients (PaCO$_2 < 30$ mmHg). End-tidal carbon dioxide readings of less than 30 mmHg were associated with poor outcome. Of those patients experiencing hypocapnoeic episodes in the emergency department 72.1% ($n = 44$) had a poor outcome (GOS 1, 2 or 3) as compared with the normocapnoeic group (55%, $n = 55$). Emergency department hypocapnoea was associated with high mortality (37.7%, $n = 23$).
Pre-hospital Implications:

*Due to the deleterious effects of hypocapnoea, the introduction of end-tidal carbon dioxide measurement into the pre-hospital setting may be desirable.*

4. Airway adjuncts and suction equipment are routinely removed from sterile packaging and stored in a non-sterile environment, such as within an oxygen carry case or bag. Airway adjuncts were used in 59% of the 326 patients \( (n = 193) \). Of those 193 who received an airway adjunct 41.7% \( (n = 136) \) developed a respiratory infection. Therefore a total of 70.5% of patients who received an airway adjunct in the pre-hospital setting developed a respiratory tract infection. Stepwise linear regression showed the onset of respiratory infection to be a weak but significant predictor of outcome using the GOS as the dependent variable \( (r = -0.140, p = 0.012, 95\% \text{ CI} = -0.816 \text{ to } -0.103) \); and the insertion of an airway adjunct in the pre-hospital setting to be a weak but significant predictor of the onset of respiratory infection \( (r = 0.147, p = 0.01, 95\% = 0.017 \text{ to } 0.123) \).

Respiratory infection was associated with increased morbidity with 45.9% \( (n = 62) \) of patients with respiratory infection experiencing a poor outcome (vegetative state or severe disability) versus 7.6% of those who did not experience the onset of respiratory infection \( (n = 14) \). The mortality rate associated with respiratory infection was 21.5%. Stepwise linear regression demonstrated significant results between the onset of respiratory infection and increased total ICU stay \( (r = \)
- 0.444, p < 0.001, 95% CI = −0.027 to −0.017), total in-patient days (r = −0.396, p < 0.001, 95% CI = −0.011 to −0.006) and total days intubated (r = −0.528, p < 0.001, 95% CI = −0.070 to −0.048).

**Pre-hospital Implications:**

The removal of airway adjuncts from the sterile packaging and storage in paramedic practice and the effect on patient outcome following severe traumatic head injury remains unclear. Further research into this practice is required. Consideration should be given to removing airway adjuncts from their sterile packaging only immediately prior to insertion. Removal of airway adjuncts from sterile packaging and storage in carry bags or kits should be avoided where possible.

10.2.4 Key Findings and Implications: Intravenous Fluid Volume Replacement in Hypotensive Patients with Severe Traumatic Head Injury

1. A statistically significant correlation for the administration of pre-hospital fluid volume replacement in hypotensive patients (systolic blood pressure < 90 mmHg) and improved outcomes for persons with severe traumatic head injury was not demonstrated in this study. While pre-hospital hypotension was found to be a significant predictor of poorer outcome (r = 0.250, p < 0.001, 95% CI = 0.538 to 1.342), pre-hospital fluid volume replacement did not demonstrate improved outcomes for hypotensive patients with severe traumatic head injury (r = 0.019, p = 0.742). Hypotensive patients who did not receive fluid resuscitation in the pre-
hospital setting demonstrated improved outcomes (GOS 4 or 5) when compared to those who did receive fluid resuscitation. Improved outcomes were confirmed in 13.5% of hypotensive who received fluid resuscitation as opposed to 32.3% in those not receiving fluid resuscitation (p = 0.001). However, no significant correlation was found between pre-hospital systolic blood pressure and the administration of fluid volume replacement (p = 0.443)

**Pre-hospital Implications:**

*Consideration should be given to administering pre-hospital fluid volume replacement in persons with severe traumatic head injury only to those patients presenting with hypotension. Consideration should be given to withholding fluid volume replacement in non-hypotensive persons if undertaking this intervention will result in time at scene exceeding 10 minutes or if transport time to hospital is less than 10 minutes.*

### 10.2.5 Key Findings and Implications: Time in the Pre-Hospital Setting

1. Stepwise linear regression found that a scene time less than or equal to 10 minutes was a weak but significant predictor of outcome (r = −0.122, p = 0.036, 95% CI = −1.024 to −0.023). A poor outcome (death, vegetative state or severe disability) was observed in 51.9% of patients (n = 27) when scene time was less than or equal to 10 minutes. Whereas when scene time was greater than 10 minutes, poor outcomes were observed in 69.4% of patients (n = 167). Patients demonstrated improved outcomes (moderate disability or good recovery) when scene time was
less than or equal to 10 minutes when compared to those where scene time was
greater than 10 minutes (48.1%, n = 25 versus 30.7%, n = 74 respectively). Scene
time was less than or equal to 10 minutes in only 17.7% (n = 52) of cases whereas
scene time exceeded 10 minutes in 82.3% of cases (n = 241).

Pre-hospital Implications:

Where possible, paramedic should consider expedited transport of patients with severe traumatic head injury and limit time at scene to less than 10 minutes. Consideration should be given to withholding intensive care interventions and pre-hospital fluid if no immediate threat to life exists and if such procedures will increase time to definitive treatment and result in time at scene exceeding 10 minutes.

10.3 Future Research

10.3.1 Pre-Hospital Ventilation of Patients with Brain Injuries

The frequency of inadvertent hyperventilation in the pre-hospital setting could not be
determined in this study. The relationship between the knowledge and experience of the paramedic in airway management and ventilation and the outcome of patients with brain injuries is for the most part, unknown. There is a dearth of literature recounting this pre-hospital intervention. Ventilation of the patient with severe brain injuries in the pre-hospital phase of management may be a crucial intervention in determining neurological outcome following severe traumatic head injury. Further investigation in this area under discussion is required to provide meaningful and consequential data.
10.3.2 The Efficacy of the Use of Rigid Cervical Collars in Patients with Severe Traumatic Head Injuries

The application of a rigid cervical collar may be deleterious to patient outcome by way of associated airway obstruction, reduced ventilation and increased intracranial pressure. No significant benefit in the application of a cervical collar was demonstrated for patients with a cervical spine injury. The application of a rigid cervical collar may adversely affect those patients without cervical spine injury. Further research is required to investigate the effect of rigid cervical collar application on airway management and ventilation and neurological outcome in patients with severe head injuries.

10.3.3 The Efficacy of Pre-Hospital Advanced Life Support in Severe Traumatic Head Injury in Rural and Remote Areas of Australia

The cohort of patients with brain injuries in the current study was selected from the Sydney and metropolitan regions. Similarly, other major studies have predominantly considered samples of patients with brain injuries from major metropolitan or large regional areas that are serviced by a trauma facility. There is a paucity of evidence relating to the efficacy of pre-hospital advanced life support in persons with traumatic injuries in rural and remote areas. As such, further research is required in this area.

10.3.4 The Pre-Hospital Management of Severe Head Trauma and its Relationship to In-Hospital Management and Patient Outcome

Little is known of the relationship between the pre-hospital management of persons with severe head injury and the subsequent in-hospital management. The decisions made in the pre-hospital setting may potentially impact on the ongoing management of the patient
within the in-hospital environment. Investigation and analysis of the impact of pre-
hospital management on in-hospital management of persons with severe head injury was
beyond the scope of this study and as such, further investigation is required.

10.3.5 Internal Organisational Training versus University Based
Education in Paramedic Practice

The past decade has seen a sharp increase in the number of paramedic programs within
the higher education sector. While a number of these programs have been producing
graduates for over ten years, little is known of the efficacy of the university programs
versus the internal training of paramedics which is still the main source of employment
for some Ambulance Services. Therefore research investigating the efficacy of university
based education programs on paramedic cognitive, psychomotor and/or affective
attributes would increase the body of knowledge within the profession and potentially
contribute to the understanding and development of paramedic practice.

10.3.6 Pre-Hospital Management and Patient Outcome in Severe
Traumatic Head Injury

Retrospective studies yield class III data. Prospective studies are considered to be higher
levels of evidence. A revised data collection form based on the findings of this study and
its integration into a prospective multi-centre study would be a recommendation from this
study. Inclusion of all area trauma hospitals in NSW into a prospective study would
potentially provide a significant body of knowledge within paramedicine to develop
revise treatment algorithms for the pre-hospital management of severe traumatic head injury.
CHAPTER ELEVEN

CONCLUSIONS

Analysis failed to support the hypothesis that persons with severe traumatic head injury will benefit from the attendance of intensive care paramedics at the scene or from endotracheal intubation provided by intensive care paramedics. Stepwise linear regression analysis found pre-hospital endotracheal intubation to be a significant predictor of poorer outcome. Pre-hospital endotracheal intubation was also found to correlate with increased scene time and increased scene time was a significant predictor of poorer outcome.

A scene time of less than or equal to 10 minutes was associated with improved neurological outcome and reduced rates of mortality and morbidity. Total time at the scene averaged 21.3 minutes. Scene time of less than or equal to 10 minutes were noted in 17.7% of cases. In 82.3% of cases scene time exceeded 10 minutes.

Regression analysis identified emergency department oxygen saturation as a significant predictor of outcome. A SpO2 greater than 95% on arrival at the emergency department was associated with improved functional outcome. Patients who experienced pre-hospital hypoxaemia demonstrated poorer outcomes than those who did not experience pre-hospital hypoxaemia.

A significant correlation was found between airway obstruction on arrival at the emergency department and a SpO2 less than 95% on arrival at the emergency department.
department. A weak but significant relationship was found between airway obstruction on arrival at the emergency department and poorer neurological outcome (GOS 1, 2 or 3). Higher percentages of morbidity were noted in patients with an obstructed airway compared to those with a clear airway. Patients with a clear airway on arrival at the emergency department demonstrated improved outcomes in comparison to those with an obstructed airway.

Episodes of hypotension in the pre-hospital setting and hypotension on arrival at the emergency department were significant predictors of poorer outcome. However, fluid administration in the pre-hospital setting was not found to be a significant predictor of improved outcome in hypotensive patients with severe traumatic head injury. Outcomes for those who received pre-hospital fluid resuscitation were poorer than those who did not receive fluid resuscitation, regardless of the presence or absence of hypotension. Analysis failed to support the hypothesis that patients with severe head injuries and hypotension will benefit from early intravenous fluid volume replacement to maintain a systolic blood pressure of greater than 90 mmHg.

Ambulance Service of NSW protocols guiding the management of pre-hospital management of severe traumatic head injury has changed little over the past decade. The efficacy of advanced pre-hospital interventions remains unclear, however the preponderance of evidence suggest that advanced life support interventions in metropolitan areas is may not result in improved neurological outcomes. Further research
is required to determine the efficacy of pre-hospital interventions and amend the management protocols as guided by research findings.


DATA COLLECTION FORM

Pre-Hospital / Pt Information

Identifier Code: ________________________ Injury Severity Score (ISS) __________
Hospital Code: ________________________
Date Of Incident: _______________________
D.O.B. ________________________
Age: ________________________

1. Birth – 1 [ ]  2. 1-5 [ ]  3. 6-11 [ ]  4. 12-15 [ ]
5. 16-29 [ ]  6. 30-39 [ ]  7. 40-49 [ ]  8. 50-59 [ ]
9. 60-69 [ ]  10. 70-79 [ ]  11. 80-89 [ ]  12. 90 + [ ]

Sex:  1. Male [ ]  2. Female [ ]

Suburb and postcode of Incident: ________________________________

Times

Times Booked (Time Zero): __________
Out: _________________ Location: _________________

Depart: _________________ Destination: _________________

Response Time: __________

1. <5 mins [ ]  2. 6-10 mins [ ]  3. 11–15 mins [ ]  4. 16-20 mins [ ]
5. >20 mins [ ]

Response Time </ = 5 mins:  1. Yes [ ]  2. No [ ]

Response Time </ = 10 mins:  1. Yes [ ]  2. No [ ]

Scene Time: __________

Scene Time </ = 10 mins:  1. Yes [ ]  2. No [ ]
Total Pre-Hosp Rx Time: ____________  Total Pre-hospital Time: ____________

Total Pre-hospital Rx time:
1. <10 mins [ ]  2. 10 – 20 mins [ ]
3. 21 -30 mins [ ]  4. 31 -40 mins [ ]  5. > 40 mins [ ]

Transport Time: ____________
Factors Relating To the Circumstances of the Injury

**Type of Injury**

1. Blunt  [ ]  2. Burn  [ ]  3. Cold  [ ]  
4. Penetrating  [ ]  5. Asphyxia  [ ]  6. Fracture  [ ]  
10. Multiple  [ ]  11. Other  [ ] (Specify):  
12. Intentional Injury  [ ]  

DOCS notification  1. Yes [ ]  2. No [ ]  3. N/A [ ]

**Mechanism Of Injury**

**Transport**

Type:

1. Motor Vehicle (car or truck)  [ ]  2. Cycle  [ ]  
3. Motor Cycle  [ ]  4. Plane  [ ]  
5. Train  [ ]  6. Boat  [ ]  
7. Other (specify)  [ ]  8. N/A  [ ]  
9. ATV  [ ]  10. Horse/Animal  [ ]

Role in Vehicle:

1. Driver  [ ]  2. Occupant  [ ]  3. Pedestrian  [ ]  
4. Rider  [ ]  5. Pillion  [ ]  6. Pilot  [ ]  
7. Other  [ ]  8. N/A  [ ]

Position in Vehicle:

1. Front Driver  [ ]  2. Front Passenger  [ ]  
3. Rear Driver  [ ]  4. Rear Passenger  [ ]  
5. Other  [ ]  6. N/A  [ ]
Type of Impact:
1. Head on [ ]
2. Rear end [ ]
3. Slide [ ]
4. Roll-over [ ]
5. Ejection [ ]
6. Other [ ]
7. N/A [ ]

Time Extricated: ________________

Entrapment:
1. Nil [ ]
2. Confinement [ ]
3. Compression [ ]
4. N/A [ ]

Protection/Restraining Devices:
1. Seat belt [ ]
2. Air bags [ ]
3. Helmet [ ]
4. Other [ ]
5. N/A [ ]
6. Not worn [ ]
7. Unknown [ ]
8. Body Armour [ ]

Impact Speed:
1. Low (<60kmh) [ ]
2. Medium (60-80kmh) [ ]
3. High (>80kmh) [ ]
4. N/A [ ]

Fall
Height ………………. Landing Surface ………………………………………………….
1. < 3m [ ]
2. 3-10m [ ]
3. > 10m [ ]
4. N/A [ ]

Location/Place of Injury
1. Home [ ]
2. Work [ ]
3. Public Area [ ]
4. Street (road) [ ]
5. School [ ]
6. Public Build. [ ]
7. Industrial [ ]
8. Farming [ ]
9. Sports [ ]
10. Other [ ] (Specify)
11. N/A [ ]
12. Playground [ ]
Pre-Hospital Assessment

Chief Complaint ………………………………………………………………………………………………………

Airway: 1. Clear [ ] 2. Obstructed [ ]

Breathing 1. Present [ ] 2. Absent [ ]

Resp Effort 1. Normal [ ] 2. Shallow [ ]
   3. Deep [ ] 4. Not Docu [ ]

Circulation: 1. Present [ ] 2. Absent [ ]

Skin Temp: 1. Normal [ ] 2. Hot [ ] 3. Cold [ ]

Blood Loss: 1. Nil [ ] 2. <500ml [ ] 3. >500ml [ ]

Burns: 1. Nil [ ] 2. Superficial [ ] 3. Partial [ ]
   4. Full [ ] % & location…………………………

Vomiting: 1. Nil [ ] 2. Yes [ ]

Fitting: 1. Nil [ ] 2. Yes [ ]

No. & duration (Seconds) ______________________________


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Pre-Hospital Observations

First Observations

Time: _______________  From Time Zero: _______________

Pulse: ______

1. 0 [ ]  2. < 60 [ ]  3. 60-100 [ ]  4. > 100 [ ]
5. Not Documented [ ]

BP: _______________

1. < 50 [ ]  2. 50-69 [ ]  3. 70-90 [ ]  4. > 90 [ ]
5. Not Documented [ ]  6. Unpalpable [ ]

Resp Rate: __________

1. 0 [ ]  2. < 10 [ ]  3. > 35 [ ]  4. 25-35 [ ]
5. 10-24 [ ]  6. Not Documented [ ]

GCS:
Eye opening ..........  Verbal Response ............... Motor ............... Total: ...............  

1. 3-4 [ ]  2. 5-7 [ ]  3. 8-10 [ ]  4. 11-13 [ ]  5. 14-15 [ ]

Temp: ____________

1. < 35 [ ]  2. 35-37.5 [ ]  3. > 37.5 [ ]  4. Not Taken [ ]
Pupil Size:
Right
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]
Left
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Pupil Reaction:
Right
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]
Left
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

Breath Sounds: (Specify R & L if unequal/asymmetrical)
1. Normal [ ] 2. Decreased [ ] 3. Wheezes [ ]

BSL: mmol/l …………………
1. < 4 [ ] 2. 4-6 [ ] 3. 6-10 [ ] 4. > 10 [ ] 5. Not Taken [ ]

SpO2: % ______________
1. < 85 [ ] 2. 85-89 [ ] 3. 90-94 [ ] 4. 95-100 [ ]
5. Not Documented [ ]

ECG:
1. Sinus [ ] 2. Brady [ ] 3. Tachy [ ]
4. VT [ ] 5. VF [ ] 6. Asystole [ ]
7. Multi [ ] 8. Not Documented [ ]
9. Other [ ] (Specify)
Last Observations

Time: ___________ From Time Zero: ___________

Pulse: _________
1. 0 [ ] 2. < 60 [ ] 3. 60-100 [ ] 4. >100 [ ]
5. Not Documented [ ]

BP: _____________
1. < 50 [ ] 2. 50-69 [ ] 3. 70-90 [ ] 4. >90 [ ]
5. Not Documented [ ] 6. Unpalpable [ ]

Resp Rate: ____________
1. 0 [ ] 2. < 10 [ ] 3. > 35 [ ] 4. 25-35 [ ]
5. 10-24 [ ] 6. Not Documented [ ]

GCS:
Eye opening …………. Verbal Response ………………..Motor ………….. Total: …………………
1. 3-4 [ ] 2. 5-7 [ ] 3. 8-10 [ ] 4. 11-13 [ ] 5. 14-15 [ ]

Temp: _____________
1. < 35 [ ] 2. 35-37.5 [ ] 3. > 37.5 [ ] 4. Not Taken [ ]
Pupil Size:

Right
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Left
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Pupil Reaction:

Right 1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

Left 1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

Breath Sounds: (Specify R & L if unequal/asymmetrical)

1. Normal [ ] 2. Decreased [ ] 3. Wheezes [ ]


BSL: mmol/l ..................

1. < 4 [ ] 2. 4-6 [ ] 3. 6-10 [ ] 4. > 10 [ ] 5. Not Taken [ ]

SpO2: % .................

1. < 85 [ ] 2. 85-89 [ ] 3. 90-94 [ ] 4. 95-100 [ ]

5. Not Documented [ ]

ECG:

1. Sinus [ ] 2. Brady [ ] 3. Tachy [ ]

4. VT [ ] 5. VF [ ] 6. Asystole [ ]

7. Multi [ ] 8. Not Documented [ ]

9. Other [ ] (Specify)
Pre-hospital Pt Management

Suction: 1. Yes [ ] 2. No [ ]

Airway Adjunct:
1. Nil [ ] 2. Oral [ ] 3. Nasal [ ] 4. ETT [ ]

Trismus: 1. Yes [ ] 2. No [ ]

ETI:
1. Not Attempted [ ] 2. Successful [ ] 3. Unsuccessful [ ]

Time of Advanced Airway Management: ………………………
1. < 15 mins from incident [ ] 2. 15-30 mins from incident [ ]
3. 31-45 mins from incident [ ] 4. 46-60 mins from incident [ ]
5. > 60 mins from incident [ ] 6. N/A [ ]

Breathing:
1. Spontaneous [ ] 2. Shallow/Reactive [ ] 3. Hyperventilating [ ]

Resuscitation / IPPV: 1. Yes [ ] 2. No [ ]

Pressure Head 1. Yes [ ] 2. No [ ]

Bag/mask 1. Yes [ ] 2. No [ ]

Other (Specify)

SpO2 <= 95%: 1. Yes [ ] 2. No [ ]

BP <= 90systolic: 1. Yes [ ] 2. No [ ]
Posture:
1. Sitting [ ] 2. Lateral [ ] 3. Supine [ ]
4. Other [ ] (Specify)

Supine: 1. Yes [ ] 2. No [ ]

O2 Therapy:
1. 28% [ ] 2. High Conc. [ ] 3. Low flow [ ] 4. High flow [ ]
5. Other [ ] 6. Not Docu [ ]

IV Access: 1. Yes [ ] 2. No [ ] Gauge ............ Time .................

Splints:
1. Traction [ ] 2. Cervical [ ] 3. KED/RED [ ]
4. Other [ ] (Specify) 5. Not Documented [ ]

Cervical Collar: 1. Yes [ ] 2. No [ ]

Stretcher:
4. Scoop [ ] 5. Stokes [ ] 6. Other (Specify) [ ]
7. Not Docu [ ]

MAST: 1. No [ ] 2. In-situ [ ] 3. Inflated [ ]

Cardiac Arrest: 1. Yes [ ] 2. No [ ]

CPR: 1. Yes [ ] 2. No [ ]
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<td>2. Yes</td>
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### Pre-hospital Pharmacology:

1. Nil [ ]  
2. Adrenaline [ ]  
3. Atropine [ ]  
4. Calc Chloride [ ]  
5. Dextrose 50% [ ]  
6. Diazepam [ ]  
7. Entonox [ ]  
8. Frusemide [ ]  
9. Glucagon [ ]  
10. GTN [ ]  
11. Haemaccel [ ]  
12. Hartmanns [ ]  
13. Lignocaine [ ]  
14. Maxalon [ ]  
15. Morphine [ ]  
16. Naloxone [ ]  
17. Salbutamol [ ]  
18. Sodium Bic [ ]  
19. Aspirin [ ]  
20. Midazolam [ ]  
21. NaCl 0.9% [ ]

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**Prehospital Care Factors**

A/O Level: First Response (enter highest level)

1 [ ]  2 [ ]  3 [ ]  4 [ ]  5 [ ]
6. Other (Specify) [ ]

A/O Level: Second Response (enter highest level)

1 [ ]  2 [ ]  3 [ ]  4 [ ]  5 [ ]
6. Other (Specify) [ ]  7 Nil [ ]

Medical Officer on Scene:  
1. Yes [ ]  2. No [ ]

Rx Given by Medical Officer:

1. ETI [ ]  2. IV Access [ ]  3. Induction [ ]
4. Mannitol [ ]  5. Fluids [ ]  6. Nil [ ]
7. N/A [ ]  8. Other [ ] (Specify)

Delay Access / Extrication  
1. Yes [ ]  2. No [ ]  
Prehospital delay time: …………

Transport Decision:

1. Nearest Hospital which is an Area Trauma Hosp [ ]
2. Nearest Hospital which is not an Area Trauma Hosp [ ]
3. Bypass Local hops for an Area Trauma Hosp [ ]

Medical/ambulance Hospital:  
1. Yes [ ]  2. No [ ]
Emergency Department Assessment and Interventions

Level of Hospital: 1. Area Trauma [ ] 2. District [ ] 3. Other [ ]

Triage Date: …………………………………

Triage Time: …………………….. From Time Zero: ………………………

Triage Category: 1 [ ] 2 [ ] 3 [ ] 4 [ ] 5 [ ]

Patient Assessment

Regions Injured:
4. Abdo / Pelvis [ ] 5. Chest [ ] 6. GIT / Haematuria [ ]
7. Upper Limb [ ] 8. Lower Limb [ ] 9. Other (Specify) [ ]

Airway on Arrival at ED:
1. Clear [ ] 2. Obstructed [ ] Cause: _________________________________

Airway Obstruction:
5. Vomitus [ ] 6 Other [ ]

Weight in Kg: …………….. 2. Not Doc [ ] 3. N/A [ ]

Time Weighed: …………….. Estimated Weight [ ] Pt Weighted [ ]

Height: ………………………
**ED Patient Observations**

1st Obs [ ]  
2nd Obs [ ]  
3rd Obs [ ]  
Last Obs [ ]

Other (Specify).

Date: ______________

Time: ______________  From Time Zero: ______________

Pulse: ____________

1. 0 [ ]  
2. < 60 [ ]  
3. 60-100 [ ]  
4. > 100 [ ]  
5. Not Documented [ ]

BP: ____________

1. < 50 [ ]  
2. 50-90 [ ]  
3. 70-90 [ ]  
4. > 90 [ ]  
5. Not Documented [ ]

Resp Rate: ____________

1. 0 [ ]  
2. < 10 [ ]  
3. > 35 [ ]  
4. 25-35 [ ]  
5. 10-24 [ ]  
6. Not Documented [ ]

Ventilated: (Mechanical or Manual)

1. Yes [ ]  
2. No [ ]  
Rate: .......................  

GCS:

Eye Opening .............. Verbal Response .............. Motor .............. Total ..............

1. 3-4 [ ] 2. 5-7 [ ] 3. 8-10 [ ] 4. 11-13 [ ] 5. 14-15 [ ]

Sedated:

1. Yes [ ]  
2. No [ ]
Pupil Size:
Right
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]
Left
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Pupil Reaction:
Right 1. Present [ ] 2. Absent [ ] 3. Unknown [ ]
Left 1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

SpO2: % __________
1. < 85 [ ] 2. 85-89 [ ] 3. 90-94 [ ] 4. 95-100 [ ]
5. Not Documented [ ]

SpO2 ≤ 95%: 1. Yes [ ] 2. No [ ]

ETCO2: PaCO2 ................. 1st Reading.................. Time............
1. Not Intubated [ ] 2. < 25 mmHg [ ] 3. 25-35 mmHg [ ]
4. > 35mmHg [ ] 5. Other [ ] (Specify) 6. Not Documented [ ]

ECG:
1. Sinus [ ] 2. Brady [ ] 3. Tachy [ ]
4. VT [ ] 5. VF [ ] 6. Asystole [ ]
7. Multi [ ] 8. Not Documented [ ]
9. Other [ ] (Specify)

Temp: __________
1. < 35 [ ] 2. 35-37.5 [ ] 3. > 37.5 [ ] 4. Not Doc [ ]
ED Patient Observations

1st Obs [ ]  2nd Obs [ ]  3rd Obs [ ]  Last Obs [ ]

Other (Specify).

Date: ______________

Time: ______________  From Time Zero: ______________

Pulse: ______________

1. 0 [ ]  2. < 60 [ ]  3. 60-100 [ ]
4. > 100 [ ]  5. Not Documented [ ]

BP: ______________

1. < 50 [ ]  2. 50-90 [ ]  3. 70-90 [ ]
4. > 90 [ ]  5. Not Documented [ ]

BP < = 90systolic:  1. Yes [ ]  2. No [ ]

Resp Rate: ______________

1. 0 [ ]  2. < 10 [ ]  3. > 35 [ ]  4. 25-35 [ ]
5. 10-24 [ ]  6. Not Documented [ ]

Ventilated: (Mechanical or Manual)

1. Yes [ ]  2. No [ ]  Rate: .........................

GCS:

Eye Opening .............. Verbal Response .................Motor .................Total .................

1. 3-4 [ ]  2. 5-7 [ ]  3. 8-10 [ ]  4. 11-13 [ ]  5. 14-15 [ ]

Sedated:

1. Yes [ ]  2. No [ ]
Pupil Size:

Right
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Left
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Pupil Reaction:

Right
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

Left
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

SaO2: %

1. < 85 [ ] 2. 85-89 [ ] 3. 90-94 [ ] 4. 95-100 [ ]
5. Not Documented [ ]

ETCO2: PaCO2

1. Not Intubated [ ] 2. < 25 mmHg [ ] 3. 25-35 mmHg [ ]
4. > 35 mmHg [ ] 5. Other [ ] (Specify) 6. Not Documented [ ]

ECG:

1. Sinus [ ] 2. Brady [ ] 3. Tachy [ ]
4. VT [ ] 5. VF [ ] 6. Asystole [ ]
7. Multi [ ] 8. Other [ ] (Specify)…………………
9. Not Documented [ ]

Temp: ____________

1. < 35 [ ] 2. 35-37.5 [ ] 3. > 37.5 [ ] 4. Not Doc [ ]
Discharged ED: Date: ……………………… Time: ……………………..

Discharged To: ………………………

1. Theatres [ ] 2. ICU [ ] 3. Wards [ ] 4. Other [ ]

ED Time Minutes: ……………………..

ED Time <= 60 mins 1. Yes [ ] 2. No [ ]

ED time Hours: ………………………

Inter-hospital Retrieval: 1. Yes [ ] 2. No [ ]
**Diagnostic Tests**

Date: _____________

Type of Lesion: ____________________________________________________________

1. Nil [ ]
2. Subdural [ ]
3. Epidural [ ]
4. Subarachnoid [ ]
5. Cerebral Contusion [ ]
6. Diffuse Axonal Injury [ ]
7. Intracerebral Haematoma [ ]
8. Penetrating Cranial Wound [ ]
9. Other [ ] (Specify)

Diffuse Injury Grade:

1. Normal CT [ ]
2. Cisterns Present [ ]
3. Cisterns Present Shift < 5mm [ ]
4. Cisterns Compressed/Absent Shift < 5mm [ ]
5. Shift > 5mm [ ]
6. Other [ ]

Type of Fracture: ____________________________________________________________

1. Nil [ ]
2. Depressed Skill # [ ]
3. Basal Skull # [ ]
4. Other [ ]

MRI: 1. Yes [ ]
      2. No [ ]

CT: 1. Yes [ ]
     2. No [ ]
Diagnostic Tests / Biochemistry

Test Result Range = Nil Test / Entry = 0  Normal = 1  High = 2  Low = 3

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<td>K+ (3.5-4.8 mmol/L)</td>
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<td>CO2 (24-30 mmol/L)</td>
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<td>Urea (2.8-7.8 mmol/L)</td>
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<td>Creatinine (0.06-0.11 mmol/L)</td>
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<td>Glucose (3.0-5.8 mmol/L)</td>
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<td>Albumin (35-55g/L)</td>
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<tr>
<td>AST (SGOT) (5-35 U/L)</td>
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<td>Lactate (0.4-1.7 mmol/L)</td>
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<td>pH (7.38-7.44)</td>
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</table>
Chemical + Normal Range

PCO2: (32-46 mmHg)

PO2: (74-108 mmHg)

Base Excess: (-2+2 mmol/L)

Calc. Bicarb: (21-29 mmol/L)

Total Hb:

Ethanol: (Legal Limit 0.5 = 11 mmol/L)

**Diagnostic Tests / Haematology**

WBC: (3.0-100 x 10^9/L)

Hb: (12.0-18.0 g/dL)

PLT: (150-400 x 10^9 /L)

RBC: (4.5-6.5 million/mm3)

INR: (0.9-1.1)
Surgery / Operation Report:

Date: _____________________________

Operating Theatre From:
1. ED [ ]  2. ICU [ ]  3. Wards [ ]  4. Other [ ]

Operating Theatre To:
1. ED [ ]  2. ICU [ ]  3. Wards [ ]  4. Other [ ]

Operation Type: _________________________________________________________________

Operation Diagnosis:
________________________________________________________________________
________________________________________________________________________

Specify- Procedure & Time:
**OR Patient Observations**

1st Obs [ ] 2nd Obs [ ] 3rd Obs [ ] Last Obs [ ]

Other (Specify).

Date: ________________

Time: ________________  From Time Zero: ________________

Pulse: ____________
1. 0 [ ] 2. < 60 [ ] 3. 60-100 [ ]
4. > 100 [ ] 5. Not Documented [ ]

BP: ____________
1. < 50 [ ] 2. 50-90 [ ] 3. 70-90 [ ]
4. > 90 [ ] 5. Not Documented [ ]

Resp Rate: ____________
1. 0 [ ] 2. < 10 [ ] 3. > 35 [ ] 4. 25-35 [ ]
5. 10-24 [ ] 6. Not Documented [ ]

Ventilated: (Mechanical or Manual)
1. Yes [ ] 2. No [ ] Rate: ......................

GCS:
Eye Opening ………… Verbal Response ……………………Motor ………………… Total …………………
1. 3-4 [ ] 2. 5-7 [ ] 3. 8-10 [ ] 4. 11-13 [ ] 5. 14-15 [ ]

Sedated:
1. Yes [ ] 2. No [ ]
Pupil Size:
Right
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]
Left
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Pupil Reaction:
Right
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]
Left
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

SpO2: % __________
1. < 85 [ ] 2. 85-89 [ ] 3. 90-94 [ ] 4. 95-100 [ ]
5. Not Documented [ ]

ETCO2: PaCO2 ............... 
1. Not Intubated [ ] 2. < 25 mmHg [ ] 3. 25-35 mmHg [ ]
4. > 35mmHg [ ] 5. Other [ ] (Specify) 6. Not Documented [ ]

ECG:
1. Sinus [ ] 2. Brady [ ] 3. Tachy [ ]
4. VT [ ] 5. VF [ ] 6. Asystole [ ]
7. Multi [ ] 8. Other [ ] (Specify) .................
9. Not Documented [ ]

Temp: __________
1. < 35 [ ] 2. 35-37.5 [ ] 3. > 37.5 [ ] 4. Not Doc [ ]
Intracranial Pressure Monitoring:

1. Nil [ ]
2. 0-10 mmHg [ ]
3. 11-20 mmHg [ ]
4. 20-40 mmHg [ ]
5. > 40 mmHg [ ]

ICP = _________________________

Ventricular Drain:

1. Yes [ ]
2. No [ ]
OR Patient Observations

1st Obs [ ] 2nd Obs [ ] 3rd Obs [ ] Last Obs [ ]

Other (Specify).

Date: ________________
Time: ________________ From Time Zero: ________________

Pulse: ________________
1. 0 [ ] 2. < 60 [ ] 3. 60-100 [ ]
4. > 100 [ ] 5. Not Documented [ ]

BP: ________________
1. < 50 [ ] 2. 50-90 [ ] 3. 70-90 [ ]
4. > 90 [ ] 5. Not Documented [ ]

Resp Rate: ________________
1. 0 [ ] 2. < 10 [ ] 3. > 35 [ ] 4. 25-35 [ ]
5. 10-24 [ ] 6. Not Documented [ ]

Ventilated: (Mechanical or Manual)
1. Yes [ ] 2. No [ ] Rate: ......................

GCS:
Eye Opening ................. Verbal Response ................. Motor ................. Total .................
1. 3-4 [ ] 2. 5-7 [ ] 3. 8-10 [ ] 4. 11-13 [ ] 5. 14-15 [ ]

Sedated:
1. Yes [ ] 2. No [ ]
Pupil Size:

Right
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Left
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Pupil Reaction:

Right
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

Left
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

SpO2: %

1. < 85 [ ] 2. 85-89 [ ] 3. 90-94 [ ] 4. 95-100 [ ]
5. Not Documented [ ]

ETCO2: PaCO2

1. Not Intubated [ ] 2. < 25 mmHg [ ] 3. 25-35 mmHg [ ]
4. > 35mmHg [ ] 5. Other [ ] (Specify) 6. Not Documented [ ]

ECG:

1. Sinus [ ] 2. Brady [ ] 3. Tachy [ ]
4. VT [ ] 5. VF [ ] 6. Asystole [ ]
7. Multi [ ] 8. Other [ ] (Specify) …………..
9. Not Documented [ ]

Temp: ____________

1. < 35 [ ] 2. 35-37.5 [ ] 3. > 37.5 [ ] 4. Not Doc [ ]
Intracranial Pressure Monitoring:

1. Nil [ ] 2. 0-10 mmHg [ ] 3. 11-20 mmHg [ ]
4. 20-40 mmHg [ ] 5. > 40 mmHg [ ]

ICP = _______________________

Ventricular Drain:

1. Yes [ ] 2. No [ ]
Intensive Care Unit

Date Admitted: __________________________

ICU From
1. ED [ ] 2. Theatre [ ] 3. Ward [ ] 4. Other [ ]

ICU To:
1. Theatres [ ] 2. ICU [ ] 3. Ward [ ] 4. Other [ ]

Date Discharged: ________________________

Time In ICU: (days) ______________________

Time in ICU: (mins) ______________________
ICU Patient Observations

1st Obs [ ]  2nd Obs [ ]  3rd Obs [ ]  Last Obs [ ]

Other (Specify).

Date: _______________

Time: _______________  From Time Zero: _______________

Pulse: _____________

1. 0 [ ]  2. < 60 [ ]  3. 60-100 [ ]

4. > 100 [ ]  5. Not Documented [ ]

BP: ______________

1. < 50 [ ]  2. 50-90 [ ]  3. 70-90 [ ]

4. > 90 [ ]  5. Not Documented [ ]

Resp Rate: ____________

1. 0 [ ]  2. < 10 [ ]  3. > 35 [ ]  4. 25-35 [ ]

5. 10-24 [ ]  6. Not Documented [ ]

Ventilated: (Mechanical or Manual)

1. Yes [ ]  2. No [ ]  Rate: ......................

GCS:

Eye Opening ............... Verbal Response .................... Motor ................. Total .................

1. 3-4 [ ]  2. 5-7 [ ]  3. 8-10 [ ]  4. 11-13 [ ]  5. 14-15 [ ]

Sedated:

1. Yes [ ]  2. No [ ]
Pupil Size:

Right
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Left
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Pupil Reaction:

Right
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

Left
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

SpO2:  % __________

1. < 85 [ ] 2. 85-89 [ ] 3. 90-94 [ ] 4. 95-100 [ ]

5. Not Documented [ ]

ETCO2:  PaCO2 .................

1. Not Intubated [ ] 2. < 25 mmHg [ ] 3. 25-35 mmHg [ ]

4. > 35mmHg [ ] 5. Other [ ] (Specify) 6. Not Documented [ ]

ECG:

1. Sinus [ ] 2. Brady [ ] 3. Tachy [ ]

4. VT [ ] 5. VF [ ] 6. Asystole [ ]

7. Multi [ ] 8. Other [ ] (Specify) .................

9. Not Documented [ ]

Temp: __________

1. < 35 [ ] 2. 35-37.5 [ ] 3. > 37.5 [ ] 4. Not Doc [ ]
**Intracranial Pressure Monitoring:**

1. Nil [ ]
2. 0-10 mmHg [ ]
3. 11-20 mmHg [ ]
4. 20-40 mmHg [ ]
5. > 40 mmHg [ ]

ICP = _______________________

**Ventricular Drain:**

1. Yes [ ]
2. No [ ]
# ICU Patient Observations

<table>
<thead>
<tr>
<th>1&lt;sup&gt;st&lt;/sup&gt; Obs [ ]</th>
<th>2&lt;sup&gt;nd&lt;/sup&gt; Obs [ ]</th>
<th>3&lt;sup&gt;rd&lt;/sup&gt; Obs [ ]</th>
<th>Last Obs [ ]</th>
</tr>
</thead>
</table>

Other (Specify).

Date: ______________

Time: ______________ From Time Zero: ______________

**Pulse:** ________

1. 0 [ ] 2. < 60 [ ] 3. 60-100 [ ]

4. > 100 [ ] 5. Not Documented [ ]

**BP:** ________

1. < 50 [ ] 2. 50-90 [ ] 3. 70-90 [ ]

4. > 90 [ ] 5. Not Documented [ ]

**Resp Rate:** ________

1. 0 [ ] 2. < 10 [ ] 3. > 35 [ ] 4. 25-35 [ ]

5. 10-24 [ ] 6. Not Documented [ ]

**Ventilated:** (Mechanical or Manual)

1. Yes [ ] 2. No [ ] Rate: .....................

**GCS:**

Eye Opening .............. Verbal Response .................Motor .................Total .................

1. 3-4 [ ] 2. 5-7 [ ] 3. 8-10 [ ] 4. 11-13 [ ] 5. 14-15 [ ]

**Sedated:**

1. Yes [ ] 2. No [ ]
Pupil Size:

Right
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Left
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Pupil Reaction:

Right
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

Left
1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

SpO2: % _________

1. < 85 [ ] 2. 85-89 [ ] 3. 90-94 [ ] 4. 95-100 [ ]

5. Not Documented [ ]

ETCO2: PaCO2 .................

1. Not Intubated [ ] 2. < 25 mmHg [ ] 3. 25-35 mmHg [ ]

4. > 35mmHg [ ] 5. Other [ ] (Specify) 6. Not Documented [ ]

ECG:

1. Sinus [ ] 2. Brady [ ] 3. Tachy [ ]

4. VT [ ] 5. VF [ ] 6. Asystole [ ]

7. Multi [ ] 8. Other [ ] (Specify) .................

9. Not Documented [ ]

Temp: __________

1. < 35 [ ] 2. 35-37.5 [ ] 3. > 37.5 [ ] 4. Not Doc [ ]

315
Intracranial Pressure Monitoring:
1. Nil [ ] 2. 0-10 mmHg [ ] 3. 11-20mmHg [ ]
4. 20-40 mmHg [ ] 5. > 40mmHg [ ]

Ventricular Drain:
1. Yes [ ] 2. No [ ]
**Ward Assessments**

Date Admitted: __________________________

Ward From:
1. ED [ ]  2. Theatre [ ]  3. Ward [ ]  4. Other [ ]

Ward To / Discharged To:
1. Theatres [ ]  2. ICU [ ]  3. Other Ward [ ]  4. Home [ ]
5. Brain Injury Unit [ ]  6. Private Hospital [ ]  7. Rehab Unit [ ]
8. Mortuary [ ]  9. Other [ ]

Date Discharged: __________________________

Time In Ward: (days) __________________________

Time in Ward: (mins) __________________________
Ward Patient Observations

1st Obs [ ] 2nd Obs [ ] 3rd Obs [ ] Last Obs [ ]

Other (Specify).

Date: ________________

Time: ________________  From Time Zero: ________________

Pulse: ________________

1. 0 [ ] 2. < 60 [ ] 3. 60-100 [ ]
4. > 100 [ ] 5. Not Documented [ ]

BP: ________________

1. < 50 [ ] 2. 50-90 [ ] 3. 70-90 [ ]
4. > 90 [ ] 5. Not Documented [ ]

Resp Rate: ________________

1. 0 [ ] 2. < 10 [ ] 3. > 35 [ ] 4. 25-35 [ ]
5. 10-24 [ ] 6. Not Documented [ ]

Ventilated: (Mechanical or Manual)

1. Yes [ ] 2. No [ ] Rate: ……………………

GCS:

Eye Opening …………… Verbal Response ……………… Motor ……………… Total ………………

1. 3-4 [ ] 2. 5-7 [ ] 3. 8-10 [ ] 4. 11-13 [ ] 5. 14-15 [ ]

Sedated:

1. Yes [ ] 2. No [ ]
Pupil Size:

Right
1. Normal [ ]  2. Dilated [ ]  3. Constricted [ ]  4. Unknown [ ]

Left
1. Normal [ ]  2. Dilated [ ]  3. Constricted [ ]  4. Unknown [ ]

Pupil Reaction:

Right 1. Present [ ]  2. Absent [ ]  3. Unknown [ ]

Left 1. Present [ ]  2. Absent [ ]  3. Unknown [ ]

SpO₂: %

1. < 85 [ ]  2. 85-89 [ ]  3. 90-94 [ ]  4. 95-100 [ ]

5. Not Documented [ ]

ETCO₂: PaCO₂

1. Not Intubated [ ]  2. < 25 mmHg [ ]  3. 25-35 mmHg [ ]

4. > 35 mmHg [ ]  5. Other [ ] (Specify)  6. Not Documented [ ]

ECG:

1. Sinus [ ]  2. Brady [ ]  3. Tachy [ ]

4. VT [ ]  5. VF [ ]  6. Asystole [ ]

7. Multi [ ]  8. Other [ ] (Specify) …………..

9. Not Documented [ ]

Temp: __________

1. < 35 [ ]  2. 35-37.5 [ ]  3. > 37.5 [ ]  4. Not Doc [ ]
### Ward Patient Observations

1<sup>st</sup> Obs [ ] 2<sup>nd</sup> Obs [ ] 3<sup>rd</sup> Obs [ ] Last Obs [ ]

Other (Specify).

Date: ________________

Time: ________________ From Time Zero: ________________

Pulse: ________________

1. 0 [ ] 2. < 60 [ ] 3. 60-100 [ ]
4. > 100 [ ] 5. Not Documented [ ]

BP: ________________

1. < 50 [ ] 2. 50-90 [ ] 3. 70-90 [ ]
4. > 90 [ ] 5. Not Documented [ ]

Resp Rate: ________________

1. 0 [ ] 2. < 10 [ ] 3. > 35 [ ] 4. 25-35 [ ]
5. 10-24 [ ] 6. Not Documented [ ]

Ventilated: (Mechanical or Manual)

1. Yes [ ] 2. No [ ] Rate: ......................

GCS:

Eye Opening ............... Verbal Response .................. Motor ............... Total ..................

1. 3-4 [ ] 2. 5-7 [ ] 3. 8-10 [ ] 4. 11-13 [ ] 5. 14-15 [ ]

Sedated:

1. Yes [ ] 2. No [ ]
Pupil Size:
Right
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]
Left
1. Normal [ ] 2. Dilated [ ] 3. Constricted [ ] 4. Unknown [ ]

Pupil Reaction:
Right 1. Present [ ] 2. Absent [ ] 3. Unknown [ ]
Left 1. Present [ ] 2. Absent [ ] 3. Unknown [ ]

SpO2: %
1. < 85 [ ] 2. 85-89 [ ] 3. 90-94 [ ] 4. 95-100 [ ]
5. Not Documented [ ]

ETCO2: PaCO2 .................
1. Not Intubated [ ] 2. < 25 mmHg [ ] 3. 25-35 mmHg [ ]
4. > 35 mmHg [ ] 5. Other [ ] (Specify) 6. Not Documented [ ]

ECG:
1. Sinus [ ] 2. Brady [ ] 3. Tachy [ ]
4. VT [ ] 5. VF [ ] 6. Asystole [ ]
7. Multi [ ] 8. Other [ ] (Specify) .................
9. Not Documented [ ]

Temp: 
1. < 35 [ ] 2. 35-37.5 [ ] 3. > 37.5 [ ] 4. Not Doc [ ]
**Interventions**

Resuscitation of BP:  
Date: .....................  Day: ............

1. No [ ]  
2. Pre-hospital [ ]  
3. ED [ ]  
4. Theatres [ ]  
5. ICU [ ]  
6. Ward [ ]

Endotracheal Intubation:  
Date: .....................  Day: ............

1. No [ ]  
2. Pre-hospital [ ]  
3. ED [ ]  
4. Theatres [ ]  
5. ICU [ ]  
6. Ward [ ]

Pre-hospital ETI:  
1. Yes [ ]  
2. No [ ]

Rapid Sequence Induction/Sedation & ETI:  
Date: .....................  Day: ............

1. No [ ]  
2. Pre-hospital [ ]  
3. ED [ ]  
4. Theatres [ ]  
5. ICU [ ]  
6. Ward [ ]

Hyperventilation:  
Date: .....................  Day: ............

1. No [ ]  
2. Pre-hospital [ ]  
3. ED [ ]  
4. Theatres [ ]  
5. ICU [ ]  
6. Ward [ ]

Mannitol:  
Date: .....................  Day: ............

1. No [ ]  
2. Pre-hospital [ ]  
3. ED [ ]  
4. Theatres [ ]  
5. ICU [ ]  
6. Ward [ ]

Intracranial Pressure Monitoring: (highest reading):  
Date: .....................  Day: ............

1. No [ ]  
2. 0-10 mmHg [ ]  
3. 11-20 mmHg [ ]  
4. 20-40 mmHg [ ]  
5. > 40mmHg [ ]

Highest Reading: _____________mmHg  
Date: .....................  Day: ............
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<th>Section</th>
<th>Date:</th>
<th>Day:</th>
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<tbody>
<tr>
<td>Craniotomy:</td>
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<tr>
<td>1. Yes [ ]</td>
<td>2. No [ ]</td>
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<td>Ventricular Drain:</td>
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<td></td>
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<tr>
<td>1. Yes [ ]</td>
<td>2. No [ ]</td>
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<td>Management of Hyperthermia:</td>
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<td>1. No [ ]</td>
<td>2. Pre-hospital [ ]</td>
<td>3. ED [ ]</td>
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<td>5. ICU [ ]</td>
<td>6. Ward [ ]</td>
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<td>Management of Hypertension:</td>
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<td>1. No [ ]</td>
<td>2. Pre-hospital [ ]</td>
<td>3. ED [ ]</td>
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<td>4. Theatres [ ]</td>
<td>5. ICU [ ]</td>
<td>6. Ward [ ]</td>
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<td>Management of Seizures:</td>
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<tr>
<td>1. No [ ]</td>
<td>2. 0-10 mmHg [ ]</td>
<td>3. 11-20 mmHg [ ]</td>
</tr>
<tr>
<td>4. 20-40 mmHg [ ]</td>
<td>5. &gt;40mmHg [ ]</td>
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<td>Management of Other Injuries:</td>
<td></td>
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</tr>
<tr>
<td>1. No [ ]</td>
<td>2. Crush Injury of Legs or trunk [ ]</td>
<td></td>
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<tr>
<td>3. Amputation or ischemia of a limb [ ]</td>
<td>4. Severe faciomaxillary injury [ ]</td>
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<tr>
<td>5. Moderately severe Injury to 2 or more areas of: head, neck, chest, abdomen, pelvis, back, femur [ ]</td>
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<td>6. Multiple fractures of long bones or pelvis [ ]</td>
<td>7. Spinal [ ]</td>
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<td>8. Other [ ]</td>
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Specify: ____________

Stable: [ ] Unstable: [ ]
Surgery for Other Injuries:  
Date: ..................  Day: ............

1. No [ ]  
2. Faciomaxillary [ ]  
3. Chest [ ]  
4. Abdomen [ ]  
5. Upper limb [ ]  
6. Lower limb [ ]  
7. Pelvis [ ]  
8. Back [ ]  
9. Other [ ]  

Specify: .............................................................................................................
OTHER INTERVENTIONS: Specify

Intervention: ___________________________________________ Date: ________  Day: _______

Intervention: ___________________________________________ Date: ________  Day: _______

Intervention: ___________________________________________ Date: ________  Day: _______

Intervention: ___________________________________________ Date: ________  Day: _______

Intervention: ___________________________________________ Date: ________  Day: _______

Intervention: ___________________________________________ Date: ________  Day: _______
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<th>Day</th>
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<th>Drug Code</th>
<th>Dose</th>
<th>Dose Range</th>
<th>Time</th>
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<th>Route &amp; Code</th>
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</table>
Outcome Details

Date Admitted Hospital: __________________________

Date Discharged Hospital: ________________________

Total Days in Hospital: __________________________

Discharged to:
1. Brain Injury Unit [ ]
2. Local Hospitals [ ]
3. District Hospital [ ]
4. Private Hospital [ ]
5. Home [ ]
6. Mortuary [ ]
7. Other [ ]

Outcome Score

Initial GCS:
Eye Opening ............. Verbal Response .................Motor ...............Total ...............  
1. 3-4 [ ] 2. 5-7 [ ] 3. 8-10 [ ] 4. 11-13 [ ] 5. 14-15 [ ]

Final GCS:
Eye Opening ............. Verbal Response .................Motor ...............Total ...............  
1. 3-4 [ ] 2. 5-7 [ ] 3. 8-10 [ ] 4. 11-13 [ ] 5. 14-15 [ ]
Glasgow Outcome Score:

1. Death [ ]
2. Persistent Vegetative State [ ]
3. Severe Disability [ ]
4. Moderate Disability [ ]
5. Good Recovery [ ]

Dead: 1. Yes [ ] 2. No [ ]

Disability Rating Score:

1. Death N/A [ ]
2. Extreme Vegetative State [ ]
3. Vegetative State [ ]
4. Extremely Severe [ ]
5. Severe [ ]
6. Moderately Severe [ ]
7. Moderate [ ]
8. Partial [ ]
9. Mild [ ]
10. None [ ]

**Airway**

Time to advanced airway management (ETI) (minute): .........................

1. < 15 mins from incident [ ]
2. 15-30 mins from incident [ ]
3. 31-45 mins from incident [ ]
4. 46-60 mins from incident [ ]
5. > 60 mins from incident [ ]
6. N/A [ ]

Date and Time Extubated: .................................................................

Total time intubated (hours): ..............

1. Nil [ ]
2. Less than 24 hrs [ ]
3. 1-7 days [ ]
4. 1-2 weeks [ ]
5. 3-5 weeks [ ]
6. Greater than 5 weeks [ ]
Coma

Date and Time of onset coma/sedation: _________________________

Date and Time of return of consciousness: _______________________

Duration of unconsciousness/coma (hours): _______________________  

1. Nil [ ]  2. Less than 24 hrs [ ]  3. 1-7 days [ ]
4. 1-2 weeks [ ]  5. 3-5 weeks [ ]  6. Greater than 5 weeks [ ]
7. Death [ ]

Cardiac Arrest

Cardiac Arrest:  1. Yes [ ]  2. No [ ]  Time ___________________

Place:
1. N/A [ ]  2. Pre-hospital [ ]  3. ED [ ]
4. OR [ ]  5. ICU [ ]  6. Ward [ ]


Shocks:
1. Nil [ ]  2. 200J [ ]  3. 360J [ ]  4.>3 [ ]

ROSC:  1. N/A [ ]  2. Yes [ ]  3. No [ ]  Time ______________
Mortality

Date of Death: _________________________

Dead on Arrival at Hospital: 1. Yes [ ] 2. No [ ]

Died in Hospital: 1. Yes [ ] 2. No [ ] Specify

Cause of Death: (specify)

1. Head Injury Direct cause [ ] 2. Complications of Head Injury [ ] 3. Other [ ]

Adverse Factors

Airway problems:

1. No [ ] 2. Pre-hospital [ ] 3. Emergency Dept [ ]

4. Theatres [ ] 5. ICU [ ] 6. Ward [ ]

7. Other [ ] (Specify)

Episode/s of Hypotension (BP <90 sys)

1. No [ ] 2. Pre-hospital [ ] 3. Emergency Dept [ ]

4. Theatres [ ] 5. ICU [ ] 6. Ward [ ]

7. Other [ ] (Specify)

Episode/s of De-saturation (PaO2 <60mmHg / SaO2 <85mmHg):

1. No [ ] 2. Pre-hospital [ ] 3. Emergency Dept [ ]

4. Theatres [ ] 5. ICU [ ] 6. Ward [ ]

7. Other [ ] (Specify)
Episode/s of Hypocarbia (PaCO2 <28mmHg):
1. No [ ] 2. Pre-hospital [ ] 3. Emergency Dept [ ]
4. Theatres [ ] 5. ICU [ ] 6. Ward [ ]
7. Other [ ] (Specify)

Episode/s of Seizure Activity:
1. No [ ] 2. Pre-hospital [ ] 3. Emergency Dept [ ]
4. Theatres [ ] 5. ICU [ ] 6. Ward [ ]
7. Other [ ] (Specify)

Time in ICU: ____________________
1. < 7 days [ ] 2. 7-14 days [ ] 3. > 14 days [ ] 4. Nil [ ]

Infection/Sepsis:
1. Yes [ ] 2. No [ ] Specify

Respiratory Sepsis
1. Yes [ ] 2. No [ ] Specify

Age (>40 yrs)
1. Yes [ ] 2. No [ ] Specify

Co-morbid conditions
1. Nil [ ] 2. Cardiovascular [ ] 3. Hypertension [ ]
4. CNS [ ] 5. Respiratory [ ] 6. Per Vascular [ ]
7. Renal [ ] 8. Endocrine [ ] 9. Epilepsy [ ]
10. GIT [ ] 11. Obesity [ ] 12. Immunological [ ]
13. Haematological [ ] 14. Pregnancy [ ]
15. Other [ ] (Specify)

Comorbidity: 1. Yes [ ] 2. No [ ]
Alcohol and Drugs:
9. Opiates [ ]

Blood Alcohol Level:  Reading: _________________________________
1. Not Documented [ ]  2. Nil [ ]  3. <11 mmol/L
4. 11-22 mmol/L [ ]  5. >22 mmol/L [ ]

Inter-hospital Transfer (for treatment/assessment – not for re-hab)
1. Not Transferred from Area trauma hospital [ ]
2. Transferred from Local or District Hosp to area Trauma [ ]
3. Not Transferred from Local or district Hospital [ ]
4. Transferred from Area Trauma to Area Trauma [ ]

Other Adverse factor: ........................................................................................................

Other Adverse factor: ........................................................................................................

Other Adverse factor: ........................................................................................................

Other Adverse factor: ........................................................................................................
GLASGOW OUTCOME SCORE

1. Death (prior to regaining consciousness) [ ]
2. Persistent Vegetative State [ ]
3. Severe Disability (conscious but disabled) [ ]
4. Moderate Disability (disabled but independent) [ ]
5. Good Recovery (resume normal life – may have minor neurological or psychological deficits) [ ]

DISABILITY RATING SCALE

<table>
<thead>
<tr>
<th>Total DR Score</th>
<th>Level of Disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. 25 – 29</td>
<td>Extreme Vegetative State</td>
</tr>
<tr>
<td>2. 22 – 24</td>
<td>Vegetative State</td>
</tr>
<tr>
<td>3. 17 – 21</td>
<td>Extremely Severe</td>
</tr>
<tr>
<td>4. 12 – 16</td>
<td>Severe</td>
</tr>
<tr>
<td>5. 7 – 11</td>
<td>Moderately Severe</td>
</tr>
<tr>
<td>6. 4 – 6</td>
<td>Moderate</td>
</tr>
<tr>
<td>7. 2 – 3</td>
<td>Partial</td>
</tr>
<tr>
<td>8. 1</td>
<td>Mild</td>
</tr>
<tr>
<td>9. 0</td>
<td>None</td>
</tr>
<tr>
<td>10. N/A</td>
<td>Death</td>
</tr>
</tbody>
</table>
# DISABILITY RATING SCALE

<table>
<thead>
<tr>
<th>Arousability, Awareness &amp; Responsivity</th>
<th>Eye Opening</th>
<th>0 Spontaneous</th>
<th>1 To Speech</th>
<th>2 To Pain</th>
<th>3 None</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Verbalisation</td>
<td>0 Orientated</td>
<td>1 Confused</td>
<td>2 Inappropriate</td>
<td>3 Incomprehensible</td>
</tr>
<tr>
<td></td>
<td>Communication Ability</td>
<td>0 Obeying</td>
<td>1 Localising</td>
<td>2 Withdrawing</td>
<td>3 Flexing</td>
</tr>
<tr>
<td></td>
<td>Motor Response</td>
<td>0 Complete independent</td>
<td>1 Independent (special environment)</td>
<td>2 Mildly dependent (non-resident helper)</td>
<td>3 Moderately dependent (person in home)</td>
</tr>
<tr>
<td>Cognitive ability for self care activities (dose Pt known how and when)</td>
<td>Feeding</td>
<td>0 Complete</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 Partial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 Minimal</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>3 None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Toileting</td>
<td>0 Complete</td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>1 Partial</td>
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<td>2 Minimal</td>
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</tr>
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<td></td>
<td>3 None</td>
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<td></td>
<td>Grooming</td>
<td>0 Complete</td>
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<tr>
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<td></td>
<td>1 Partial</td>
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<td></td>
<td></td>
<td>2 Minimal</td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>3 None</td>
<td></td>
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<tr>
<td>Psychosocial adaptability</td>
<td>Employability</td>
<td>0 Not restricted</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>1 Selected jobs - competitive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 Sheltered workshop – non-competitive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 Not employable</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Total DR Score</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
OTHER OUTCOME MEASURES – Westmead PTA

1. Westmead PTA (yes) [ ]  2. Westmead PTA (no) [ ]

<table>
<thead>
<tr>
<th>Duration of PTA</th>
<th>Severity of Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>(calculated from time of injury &amp; includes period of coma)</td>
<td></td>
</tr>
<tr>
<td>1. Less than 5 minutes</td>
<td>Very Mild</td>
</tr>
<tr>
<td>2. 5 – 60 minutes</td>
<td>Mild</td>
</tr>
<tr>
<td>3. 1 – 24 hours</td>
<td>Moderate</td>
</tr>
<tr>
<td>4. 1 – 7 days</td>
<td>Severe</td>
</tr>
<tr>
<td>5. 1-4 weeks</td>
<td>Very Severe</td>
</tr>
<tr>
<td>6. Greater than 4 weeks</td>
<td>Extremely Severe</td>
</tr>
<tr>
<td>7. N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>ID CODE</td>
<td>DEPARTMENT</td>
</tr>
<tr>
<td>---------</td>
<td>------------</td>
</tr>
<tr>
<td>DRUG NAMES</td>
<td>DOSE</td>
</tr>
</tbody>
</table>