QUALITY ASSESSMENT FOR PATIENTS WITH TRAUMATIC BRAIN INJURY: IMPLEMENTATION OF TECHNOLOGY, DETERMINATION OF PHYSIOLOGIC CORRELATES OF BRAIN TISSUE OXYGENATION, AND DEVELOPMENT OF A TREATMENT ALGORITHM

by

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A Thesis Submitted to the Faculty of the

COLLEGE OF NURSING

In Partial Fulfillment of the Requirement for the Degree of

MASTER OF SCIENCE

in the Graduate College

THE UNIVERSITY OF ARIZONA

2008
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ACKNOWLEDGMENTS

I would like to acknowledge the nurses on the Surgical/Trauma ICU at University Medical Center who cared for the patients in this study and diligently documented all the excessive notations required when caring for these patients. Without their intricate and detailed charting, interpretation of the results would have been inconceivable. I would also like to acknowledge the time, direction, consideration, and guidance of the Committee members: Leslie Ritter, Carrie Merkle, and Kara Snyder. Without their expert knowledge and encouragement throughout this process, this study would never have been completed.
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Abstract

Traumatic brain injury (TBI) is a costly disease that impacts millions of lives. Standardized treatment options are essential in this population. A monitoring device which measures the partial pressure of brain tissue oxygenation (PbtO₂) has recently been recommended as an adjunct in tailoring therapy. Using a descriptive design, the three aims of this study were to 1) implement new technology; 2a) perform a thorough literature review of PbtO₂ to determine physiologic correlates; 2b) compare these correlates in a patient data assessment; and 3) develop a treatment algorithm for patients with TBI that included PbtO₂ as a variable. Assessment of current protocols at the institution revealed an opportunity to update standards and technology. Findings from the literature review were also relevant when compared with patient data. A treatment algorithm was developed based on the literature and data review for patients with TBI, providing a tool for clinicians treating these patients in the future.
CHAPTER I

Introduction

Traumatic brain injury (TBI) is caused by a blow, jolt, or penetrating injury to the head which disrupts normal tissue and function of the brain. Approximately 1.4 million people sustain TBI every year in the United States; 50,000 people with these injuries die, while 235,000 are hospitalized. Both direct medical and indirect rehabilitation and long term care costs are estimated to be around $60 billion a year (Centers for Disease Control and Prevention, 2006). Of patients who sustain severe TBI, approximately 30-36% die, 5% remain in a persistent vegetative state, 15% live with severe disability, 15-20% have moderate disability, and 25% have a good outcome based on the Glasgow Outcome Scale (Valadka & Robertson, 2007).

Given the possibility for good outcome in this patient population, early assessment, invasive monitoring, and aggressive treatment are essential. Traditional measures for assessment and optimizing treatment are important; however, updating technology can aid in improving the structure of care delivery and patient outcomes. Understanding how to use a new technology and implementing a technology that may improve outcomes are important aspects of improving the structure of quality care delivery (Donabedian, 1966).

One parameter that has been introduced recently to further individualize treatment pathways with the aim to improve outcomes and quality of care provided is brain tissue
oxygenation monitoring. Assessment of the partial pressure of brain tissue oxygenation (PbtO$_2$) is significant when determining treatment options, assessing effectiveness of interventions and therapeutic goals, prognostication of outcomes, and evaluating endpoints of resuscitation.

Purpose and specific aims

This study was the first step towards reaching the ultimate goal of the trauma/surgical intensive care unit at a level I trauma center to implement practice change in the treatment for patients with TBI using new technology and standards of care. A quality assessment framework which encompasses structure, process, and outcomes is ideal for guiding the aims of this study. The overall purpose of this study is to evaluate the need for possible changes in structure and process at this institution as they relate to improved patient outcomes. There are three specific aims of this study.

Aim 1

The first aim of this study was to assess organizational structure as a part of quality of care by reviewing current use of the technology and implementing the PbtO$_2$ monitor, which included education related to its use and technical aspects of introducing the technology.

Aim 2

There are two parts to the second aim of this study; part a) consisted of an in-depth literature review to identify the physiologic correlates of PbtO$_2$; part b) consisted of
comparing variables identified from the literature to patient data of those patients who received the monitor at this institution.

Aim 3

The third aim of the study is to develop an algorithm for treatment of patients with TBI, with the goal to improve patient outcomes. The results of this study could be used in guiding an evaluation of patient outcomes after introducing the treatment algorithm which includes the use of current technology in patients with TBI.

Significance to advanced practice nursing

Andrews (2001) describes five roles of the advanced practice nurse (APN). These include collaborator, leader, educator, researcher, and clinical expert. Several of these roles are demonstrated in the development, execution, and completion of this study. As a collaborator, an APN must work with other healthcare professionals to improve patient outcomes. Several different providers needed to collaborate to make this study a success, including the APN, physicians of different specialties, residents, nurses, respiratory therapists, pharmacists, and product sales representatives. APNs were the primary initiators of bringing in this new technology, and played a leadership role for data collection and literature review. Education of nurses and physicians was also one of the roles done by the APN in this study. APNs were also the primary researchers in this study, thus fulfilling the important role of carrying out research. APNs are involved in development and revision of current protocols and standards of care, and do so autonomously (Andrews, 2001). This is an important part of quality assessment that was
done by an APN in this study. To ensure optimal care provision, an APN identifies problems, visualizes and formulates answers, and identifies areas for improvement as a part of being a clinical expert. The clinical expertise required for this study involved knowledge in acute care; specifically, critically injured patients with TBI. As the role of the APN continues to expand and be redefined, it is important to contribute to what encompasses this role through defining it, organizing projects which develop these roles, and demonstrating the significance of the APN as a healthcare provider in diverse settings, such as acute care.
CHAPTER II

Background

*Physiology of traumatic brain injury*

Traumatic brain injury occurs in two processes: primary and secondary injury. Primary injury occurs upon impact and causes initial tissue damage. Examples of this primary process are fractures, diffuse axonal injury, hematoma, or hemorrhage. One of the paramount ways in which health care professionals can intervene in this process is through prevention activities. Secondary injury refers to those processes occurring after the initial injury; minimizing damage from those injuries is the aim of medical intervention and treatment. Secondary injury is caused by either intracranial or extracranial factors, and both lead to further brain damage and ischemia (Bader, 2006; Chestnut, Marshall, Klauber, Blunt, Baldwin, Eisenberg et al, 1993; Dutton & McCunn, 2003).

Intracranial factors of secondary injury include cerebral edema, alterations in cerebral blood flow (CBF), disruptions of autoregulation, changes in cerebral metabolism, and chemical derangements. Cerebral edema is caused by a disruption in the blood brain barrier and a net plasma efflux into the brain tissue. According to the Monro-Kellie doctrine, the cranial vault is a closed system, which holds brain tissue, cerebral spinal fluid (CSF), and blood. Increases in volume of any of these substances causes a decrease in the other two, until the brain can no longer regulate changes and intracranial hypertension which may lead to herniation of the brain tissue occurs. The significant
edema leading to herniation is a result of shifting tissue, which is evident on radiographic and clinical exam, and is known as mass effect (Bader, 2006).

Alteration in cerebral blood flow is a factor which occurs as a result of changes in the cerebral vasculature and impaired cellular integrity (Bader, 2006; Barton, Hemphill, Morabito, & Manley, 2005; Chestnut et al, 1993; Dutton & McCunn, 2003). Studies have evaluated how changes in cerebral blood flow occur following TBI, and they found that there are three phases or patterns of flow after the initial injury (FIGURE 1). The first 24 hours after injury, hypoperfusion occurs due to increases in microcirculation resistance. The next 24 to 96 hours are characterized by hyperemia, hyperperfusion, edema, and some degree of vasospasm. The final phase occurs between days 5 through 14, which is marked by vasospasm and reduced perfusion. It is important to consider these phases when optimizing treatment for secondary injury (Martin, Patwardhan, Alexander, Africk, Lee, Shalmon et al, 1997; Marion, Darby, & Yonas, 1991).

CHANGES IN CBF:

<table>
<thead>
<tr>
<th>Phases of injury</th>
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<tbody>
<tr>
<td><strong>Phase I:</strong></td>
</tr>
<tr>
<td>1st 24 hours</td>
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<tr>
<td>HYPOPERFUSION</td>
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<tr>
<td>Ensure adequate B/P</td>
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<td>Fluid resuscitation</td>
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<td>ICP/ PbO₂ monitor</td>
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<td>Surgical intervention</td>
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<td><strong>Phase II:</strong></td>
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<tr>
<td>24-96 hours</td>
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<tr>
<td>HYPEREMIA</td>
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<tr>
<td>HYPERPERFUSION</td>
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<tr>
<td>EDEMA</td>
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<tr>
<td>Optimize ICP/CPP</td>
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<tr>
<td>Drain CSF</td>
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<tr>
<td>Monitor PbO₂</td>
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<tr>
<td><strong>Phase III:</strong></td>
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<tr>
<td>5th-14th days</td>
</tr>
<tr>
<td>VASOSPASM</td>
</tr>
<tr>
<td>REDUCED PERFUSION</td>
</tr>
<tr>
<td>Continue monitoring</td>
</tr>
<tr>
<td>Euvolemia</td>
</tr>
<tr>
<td>Monitor PbO₂</td>
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*CBF*-cerebral blood flow  
*B/P*-blood pressure  
*ICP*-intracranial pressure  
*CPP*-cerebral perfusion pressure  
*CSF*-cerebrospinal fluid
Another important intracranial process which is impaired by TBI is autoregulation. This is the process by which the brain normally maintains perfusion by releasing chemical transmitters to impact peripheral blood pressure, or by shunting or decreasing production of CSF. In severe TBI, the brain is no longer able to ensure adequate perfusion as secondary injury leads to edema, which also leads to intracranial hypertension and ischemia. As autoregulation is impaired, adequate CBF is no longer ensured by normal cerebral mechanisms, leading to a reduction in oxygen delivery.

Brain metabolism is also impacted when the cerebral metabolic rate increases as the brain changes from aerobic to anaerobic metabolism following cellular injury and disruptions in homeostasis. Normally, the brain tissue optimizes use of energy through intact cellular processes. In TBI, mitochondrial damage and the inflammatory process can impact metabolism by increasing the demand for energy to support these processes, leading to inefficient metabolic function in the damaged cells. Secondary injury also occurs due to chemical derangements such as the formation of free radicals, glutamate excitotoxicity, and calcium-induced damage to the cellular structure (Bader, 2006; Dutton & McCunn, 2003; Jeremitsky, Omert, Dunham, Protetch, & Rodriguez, 2003; Lang, Czosnyka, & Mehdorn, 2003).

Extracranial factors resulting in secondary injury occur from either a reduction in oxygen delivery or cerebral blood flow, or from alterations in neurochemical processes.
These include hypoxia, hypotension, hypocapnia or hypercapnia, anemia, acidosis, hyperglycemia, and hyperthermia (Bader, 2006; Barton et al, 2005; Dutton & McCunn, 2003; Jeremitsky et al, 2003; Lang et al, 2003; Stocchetti, Furlan, Adriano, & Volta, 1996; Valadka & Robertson, 2007). These intracranial and extracranial causes of secondary injury are the primary targets for medical intervention and prevention of brain damage. The technology which monitors these causes of secondary injury is an important aspect of guiding treatment for improved outcomes in patients who sustain TBI.

**History of treatment for traumatic brain injury**

Historically, monitoring of intracranial pressure (ICP) and mean arterial blood pressure (MAP) has provided an equation for the hydraulic estimation of cerebral perfusion pressure (CPP). The MAP less the ICP estimates CPP, and treatment is aimed at maintaining the CPP above a standard threshold thought to ensure adequate perfusion of brain tissue. CPP below this threshold was considered to be suboptimal, leading to further declines in oxygen delivery. Treatment typically revolves around maintaining an ICP of less than 20mmHg and a CPP of greater than 60mmHg. Osmotic therapy, drainage of cerebral spinal fluid (CSF), surgical removal of lesions, high dose barbituates, and decompressive craniectomy are all interventions aimed at decreasing intracranial hypertension (Brain Trauma Foundation, 2007). For maintenance of CPP, achieving euvolemia and the use of drugs to supplement MAP and cardiac output is the standard treatment. As technology has advanced, another variable has been added which further
directs and fine tunes therapy: the measurement of PbtO$_2$. As an adjunct to traditional monitoring parameters, this variable permits the provider to have a direct measurement of oxygenation, rather than estimation using CPP (Valadka & Robertson, 2007). Overall, this measurement broadens the perspective of how effective a treatment is, or allows for recognition of the need for further intervention using information not provided by monitoring only ICP and CPP (Bader, 2006).

**Brain tissue oxygen monitoring**

Measurement of PbtO$_2$ has been recently recognized as an important variable when determining if brain perfusion and oxygenation are adequate. The PbtO$_2$ monitor was developed by Wolfgang Fleckenstein in Kiel, Germany and has been used since the 1980s, though in the United States it has only been in use for about 6 years. Based on a technology discovered by Dr. Clark in 1956, the partial pressure of oxygen is measured using a Clark cell probe-type electrode, which is a polarized amperometric cell with an externally provided voltage of about 800 mV. When oxygen reduction occurs at the probe tip, usually between 400 to 1200mV, four electrons are released and generate a current which is directly proportionate to the oxygen consumption at the cathode (Eutech Instruments, 1997). The PbtO$_2$ monitor is a Clark-cell probe with two covered electrodes, one gold and one silver. It is inserted into the brain parenchyma to a depth of 25 to 35 mm; as dissolved oxygen in the brain tissue crosses the membrane, an electrical current is generated and transmitted to a transducer, which is then digitally converted to measure PbtO$_2$ (mmHg). The probe also measures brain temperature.
Though the area of optimal measurement for oxygenation is still controversial, in patients with TBI the probe is often placed in the penumbra region surrounding the area of greatest injury, as this region is considered to be the area which will be at the highest risk for ischemic damage. Although the probe is only sensitive for regional oxygenation values, changes at the penumbra are important indicators when predicting minute fluctuations which may reflect whole brain oxygenation. (Valadka, Gopinath, Masahiko, & Robertson, 1998; van den Brink, van Santbrink, Steyerberg, Avezaat, Suazo, Hogesteege et al, 2000). Although other types of PbtO$_2$ monitors exist, the LICOX® (Integra Neurosciences, Plainsboro, NJ) probe used in this study is considered more reliable than other brands due to overestimation of oxygenation by other probes, and is the only probe currently available to practitioners in the United States (Valadka et al, 1998). The Brain Trauma Foundation (BTF) Guidelines for Treatment of Severe Traumatic Brain Injury (2007) now recognize monitoring brain tissue oxygenation as an adjunct variable in determining secondary injury and tailoring treatment options to the patient, though it is only a level 3 recommendation due to lack of evidence from a large randomized, controlled trial. As an independent indicator of mortality, it is recommended that PbtO$_2$ is maintained above 15 mmHg for adequate oxygenation.

Given the importance of monitoring brain oxygenation along with ICP/CPP, therapy aimed at optimizing all three variables may help minimize or prevent secondary injury in patients with TBI. Equally important is determining which interventions and treatments can help balance these three variables. Treatment for ICP/CPP is mentioned
above, and a review of the literature revealed several variables that can optimize PbtO$_2$. Decreased perfusion, intracranial hypertension, hypoxia and hypoxemia, hypocapnia, hyperthermia, and anemia are all factors which contribute to decreased PbtO$_2$ levels (Artru, Jourdan, Perret-Liaudet, Charlot, & Mottolese, 1998; Bader, 2006; Coles, Minhas, Fryer, Smielewski, Aigbirihio, Donovan et al, 2002; Gracias, Guillamondegui, Stiefel, Wilensky, Bloom, Gupta et al, 2004; Jeremitsky et al, 2003; Lang et al, 2003; Meixensberger, Vath, Dings, Kunze, & Roosen, 2003; Palmer & Bader, 2005; Smith, Stiefel, Magge, Frangos, Bloom, Gracias et al, 2005; Soehle, Jaeger, & Meixensberger, 2003; Stieffel, Udoetuk, Spiotta, Gracias, Goldberg, Malone-wilensky et al, 2006; Tokutomia, Morimoto, Miyagi, Yamaguchi, Ishikawa, & Shigemori, 2003; Valadka et al, 1998; van den Brink et al, 2000; van Santbrink, Maas, & Avezaat, 1996). The following studies were designed to determine treatment and prognostic thresholds of PbtO$_2$ in order to help guide clinicians in using this variable in treatment protocols and in assessment of injury severity.

One study by van den Brink et al (2000) found that a significant and direct correlation between depth and duration of cerebral hypoxia and mortality. The researchers found that a PbtO$_2$ <5mmHg for only 30 minutes was associated with a 50% risk of death. Another study by van Santbrink et al (1996) showed that when comparing patient groups, those with a PbtO$_2$ less than 5mmHg had high mortality compared to those who never had a PbtO$_2$ less than 5mmHg. In their study, low PbtO$_2$ (<15mmHg) correlated with a poorer outcome at 6 months and increased risk of death.
Valadka et al (1998) found an increased risk of death in patients with PbtO₂ less than 6mmHg, and though adequate PbtO₂ (>20mmHg) did not necessarily indicate good outcomes, it was a helpful variable when guiding therapy for prevention of secondary injury. Stiefel et al (2006) found that in guiding therapy for prevention of cerebral hypoxia and ischemia, direct monitoring of PbtO₂ was an important adjunct variable along with ICP and CPP. They found that 33% of patients with optimal ICP/CPP still had episodes of hypoxia, with mortality at 66% if PbtO₂ was not greater than 20mmHg post-resuscitation. In this study, mortality was 30% in patients with a PbtO₂ greater than 25mmHg, 43% in patients with a PbtO₂ less than 20mmHg, and 50% in patients with PbtO₂ less than 15mmHg.

Another study by Meixensberger et al (2003) compared the use of therapy directed toward optimizing only ICP/CPP goals (group 1) versus ICP/CPP/PbtO₂ (group 2). The latter group had less hypoxic events, though both groups showed ischemic events occurring most frequently in the first day after injury and during days five and six. By assessing outcome using the Glasgow Outcome Scale, the patients in group 2 had a higher proportion of good outcomes than group 1 at 6 months (65% vs. 54%).

These studies support the use of PbtO₂ as another variable in treatment of TBI; however, it is important to continue research which can further validate the use of this technology. Prior to implementation of this monitoring device, familiarity with the measurement, treatment goals, and correlates of PbtO₂ needed to be determined and outlined for providers. Additionally, implementing the technology requires in depth
understanding of the tool and technical aspects surrounding its use. Designing protocols or treatment algorithms which incorporate \( \text{PbtO}_2 \) as another variable to direct therapy is one way to establish evidence-based practice and reach outcome goals. This is significant as an assessment of quality care delivery at centers which frequently treat patients who sustain TBI.

**Conceptual Framework**

*Principles of Donabedian’s framework for quality assessment*

Donabedian (1966) is considered the “grandfather” of quality assurance, and his framework is the theoretical basis behind this study. He determined that there are certain aspects of medical care that must be considered when approaching assessment of the quality of that care. These ideas became the three parts of the framework for assessing quality care, which are structure, process, and outcome.

Donabedian (1966) defines structure as the relative stable characteristics of the organizational setting. The term embraces the number, distribution, and qualification of professional personnel, number and size of equipment, and ways in which delivery of health services is organized. Structure characteristics are the measurable and objective factors (Donabedian, 1966). Structure is relevant to quality of care as aspects of the organizational structure may increase or decrease good performance.

Process factors relate to how things work within an organization and the framework that guides the design of the organization, which can influence outcome. They
are on-going and continuously redefined, and are influenced by the structure or setting in which care takes place. They define the mechanisms responsible for producing intended outcomes and include accessibility, continuity of care, professional protocols or standards for the delivery of care, interpersonal and interdisciplinary management of patient care, and the actual care provided compared to current protocols in place. Process assessment is concerned with the application of what is now known to be the best care; which requires that specific relevant dimensions, values, and standards of care are developed and instituted. As such, these factors outline the processes responsible for producing desired outcomes and, subsequently, the effectiveness of the organization. The assessment of process is complex, and often structure factors, process factors, and outcome factors overlap (Donabedian, 1966).

The aspect of quality assessment that is most commonly used and often thought to be the most valid indicator of quality is outcome. This looks at the outcome of medical care delivered, in terms of recovery, restoration of function, survival, or patient satisfaction. These factors are fairly concrete, they can be easy to measure based on several outcome scales which relate to specific disease states. However, since outcome is not always the relevant measure, and process or structure factors impact outcome, these are equally valid measures of quality assessment. Donabedian (1966) states that good structure increases the likelihood for good process, which in turn increases the likelihood for good outcomes (FIGURE 2). Based on this framework, it may be inferred that improvement in structure will help build better processes; and ultimately lead to better
outcomes and optimal quality of care. In this study, aspects of structure and process were evaluated with the goal of eventually optimizing quality of care and improving outcomes for patients with TBI.

**FIGURE 2: Donabedian’s framework for quality assurance (modified from Donabedian, 1966)**

*Quality assessment and brain tissue oxygen monitoring*

The first aim of the study was to evaluate structure factors which are relevant to assessment of quality care specific to treatment of TBI patients. These include presence and participation of trauma and neurosurgical teams, availability of services and space, availability of experienced staff to care for the patients, availability of equipment necessary for monitoring of intracranial pressure and brain tissue oxygenation, technical aspects related to introducing a new technology, and appropriate education for use of this technology. Application of new technology to a setting can be a catalyst for change in
structure, and may improve quality of care if that technology is well validated. However, care must be taken in the institution where this new technology is implemented, as it can be overwhelming if education about the meaning of measurements or use of technology is not fastidiously introduced, and positive outcomes as a result of the technology are not reinforced. As stated previously, often factors in each area of quality of assessment can overlap; in this study, education is a part of the assessment of structure as it relates to the proficiency of professional personnel. It is important that while introducing the PbtO$_2$ monitor, staff become familiarized with the proper use and interpretation of the values.

Additionally, product introduction mandates education on technical aspects such as storage, placement, and availability of and familiarity with the monitor. Implementation of new technology may improve overall assessment of the patient condition, allow treatment to be optimized, and lead to improved outcomes for these patients. Assessment of structure and planning changes to structure factors should be slowly initiated as practitioners and nurses become acquainted with and educated about the technology. Changes in structure which update the organization to current recommendations and guidelines requires adjustment at both individual and organizational levels.

The second aim of this study is to assess process through an in-depth literature review of correlates of PbtO$_2$ and compare those variables to data sets from patients who had the monitor. It is important to learn what factors impact PbtO$_2$, how each factor influences this measurement, and how it compared to a patient population at this institution as a part of guiding the design of the organization. Assessment of current
literature regarding the new technology aids in the determining if standards of practice are being met at the institution, by specifying criteria which should be a part of protocols and indicating deficits of care delivery that may justify introduction of new technology (Duff, 1992).

Another assessment of process, which comprised the third aim of this study, was the development of an algorithm for treatment of patients with TBI. After determining which interventions may impact values, it is important to apply that information in an organized and realistic fashion. As such, the literature review and patient data sets can help guide development of an algorithm for care of patients with TBI at this institution. When considering process factors in this study, such as accessibility and continuity of care, it is important to use standardized protocols as guidelines to practice. Reviewing current standard protocols in the literature and applying them when developing a treatment algorithm at the institution is one aspect of process that is a valuable product of this study. Donabedian (1966) states that “conformity of practice to accepted standards has a kind of interim validity which may be more relevant to the purposes of assessment in specific instances (p.186).” As such, it is important to maintain current standards at what experts in the field may recommend. For TBI patients, the BTF (2007) document that outlines current guidelines of treatment for patients with severe traumatic brain injury is the most up-dated and comprehensive source of treatment recommendations. Using these guidelines as the normative standard, assessment of this institutions standards may be improved upon and updated, leading to improved patient outcomes. This has
been demonstrated in other organizations, as evidenced in the study by Palmer et al (2001) where outcomes before and after implementation of standardized guidelines as recommended by BTF guidelines were compared. The group of patients treated post-implementation of the guidelines showed outcomes greatly improved, with a 9.13 times higher odds ratio of a good outcome when compared to the group analyzed before treatment was standardized. Therefore, process is impacted as implementation of the most current recommendations from the BTF guidelines are translated to an algorithm for treatment that includes PbtO$_2$ as a parameter for optimizing quality of care.

According to Donabedian’s framework, it can be expected that by impacting structure and process as indicated above, outcomes for patients with TBI will be improved, along with overall quality of care (FIGURE 3). Outcomes assessment is beyond the scope of this study, yet assessment of structure and process as factors which influence good outcome provide the background for a future study that can assess outcome using the new technology and treatment algorithm. The ultimate goal is that through assessment of factors impacting outcome, improvements can be made and higher quality of care delivered.

AIM 1: Structure assessment
Assess presence of neurosurgical/truma teams and current protocols, availability of space/equipment/personnel, education about and implementation of new technology

AIM 2: Process assessment
- a) literature review of correlates of PbtO₂,
- b) compare with patient data set

AIM 3: Process assessment
Development of an algorithm for treatment

Not assessed, goal is that current study will provide background for assessment of outcome after changes in structure and process following this study
CHAPTER III

Materials and Methods

This study was a quality assessment of aspects of structure and process, which are the initial steps toward a goal of eventually assessing outcome in patients who sustain TBI. A descriptive design was used to assess these aspects. The first aim of the study was to assess structure, especially in implementation of a new technology. The two parts of the second aim were to a) assess process through a literature review which would help identify physiologic correlates of PbtO$_2$, and b) compare these correlates to data sets from a small group of patients who had received the monitor. The third aim was to assess process by forming a standardized treatment algorithm which incorporates the information gathered during assessment of structure, the literature review, and patient data which would include current recommendations on treatment for patients with TBI.

The study was conducted at a 350 bed Level I trauma center. This hospital is the only trauma center in the area, and is the only hospital equipped to admit the patients with severe trauma in the region. Also, the institution is an academic center and as such, there is 24 hour availability of several specialties, including trauma and neurosurgery. The patients which sustain severe TBI are usually admitted to the surgical/trauma intensive care unit, a 15 bed unit which provides care for patients with surgical interventions or traumatic injuries.
Aim 1: Assessing structure

To assess structure, the first step was ensuring the presence and cooperation of the providers of patients with TBI. These providers included a trauma team and a critical care medicine team made up of interns, residents, and attendings. Also, a neurosurgical team was involved, with primarily neurosurgical residents and attendings caring for patients in the ICU. The nursing staff on the surgical/trauma unit and the critical care advanced practice nurse who was responsible for introducing the PbtO$_2$ monitor to the institution were also involved in care. Although the trauma and critical care providers did provide care for these patients, especially those with multiple injuries, the neurosurgical team was the only team inserviced on placing the monitor. This team was involved in discussions about implementation of the monitor to ensure cooperation. This was done by the sales rep for LICOX® in a mandatory meeting for both residents and attendings. Also, for implementation of the monitor, it was necessary to educate the nursing staff about the product, and familiarize them with technical aspects of caring for patients with the monitor, including storage, set-up, and additional documentation that would be used during the patient data review. This education involved having inservices for the nurses on several different days, and only those nurses which attended these meetings were allowed to care for the patients with the monitor. Additionally, availability of or need for any additional equipment that would be required when using the monitor was determined after speaking with the sales representative. A review of current standards of practice,
including order sets currently in place at the institution, was conducted to determine if there were standards of practice which needed up-dated.

**Aim 2: Literature review**

To determine the physiologic correlates of PbtO₂ in the literature, a search was done using the MEDLINE® database, with keywords “brain tissue oxygenation,” “brain tissue oxygen monitoring,” and “traumatic brain injury” used in the initial search to identify relevant articles. Several articles were retrieved from the references of relevant articles. Additionally, review of the BTF (2007) guidelines revealed several relevant studies which had been systematically reviewed and included in the document based on level of recommendation that could be made from evidence provided from the studies. The studies found in the literature search and from within the BTF (2007) guidelines provided the information needed to determine the physiologic correlates of PbtO₂ that could be examined and compared with the patient data information reviewed from this institution.

**Aim 2: Patient data assessment**

A retrospective chart review was completed on all patients from January 2007 through June 2007 who were admitted following a severe head injury and received the LICOX® brain tissue oxygen monitor. Data was evaluated on each patient to determine the physiologic correlates of PbtO₂ and compare that with those variables indicated in the literature as correlates.
The decision of which patients would receive the monitor and subsequent placement was made by the neurosurgical team. There was only one monitor on the unit, and this one monitor was utilized for each patient. All patients were mechanically ventilated, and standard critical care monitoring of heart rate via telemetry, arterial blood pressure, temperature, pulse oximetry, and respiratory rate were recorded. Intracranial pressure, cerebral perfusion pressure, and partial pressure of brain tissue oxygen were also continuously monitored and documented. For patients with a pulmonary artery catheter, cardiac index, output, and pulmonary artery wedge pressure was also recorded. These variables are monitored per pre-established protocols, the only new variable monitored was the PbtO$_2$. The choice to record and evaluate these specific variables for the retrospective chart review was made after in depth literature review indicated which variables tended to correlate with changes in PbtO$_2$. Since no data were gathered nor treatments initiated based on changes in PbtO$_2$, some variables indicated in the literature to impact PbtO$_2$ were not recorded continuously, as it was not standard protocol at this institution. These variables included more frequent monitoring of carbon dioxide via either blood gas assessment of the partial pressure of carbon dioxide (PaCO$_2$) or end-tidal carbon dioxide monitoring (EtCO$_2$), and hemoglobin.

Each patient with the PbtO$_2$ monitor had these monitored physiologic variables recorded every fifteen minutes, which were printed at the end of each twelve hour shift by the patient’s nurse. Monitoring during patient transport for computed tomography scans was not recorded, and patients who did not have a PAC did not have cardiac index,
output, or pulmonary artery wedge pressure recorded. The nurse caring for the patient also documented each intervention during his or her shift, including assessments and any subsequent treatment. These included administration or titration of medications or fluids, ventilator changes, and standard nursing care (i.e. suctioning, turning, or bathing). All interventions for the patients with the PbtO₂ monitor are considered standard of practice from the cerebral perfusion pressure (CPP) protocol used at the institution; no interventions were made based on PbtO₂ monitor measurements.

During the retrospective chart review, printed data from each patient were retrieved and the pre-determined variables were chronologically recorded in a computerized spreadsheet. This included blood pressure, mean arterial pressure, heart rate, oxygen saturation, temperature, respiratory rate, intracranial pressure, cerebral perfusion pressure, PbtO₂, and, if available, cardiac output and pulmonary artery wedge pressure. Basic run charts of the data were created and data were analyzed to determine which of the monitored and recorded physiologic variables correlated with changes in PbtO₂. Marked changes of PbtO₂ initiated a review of the documentation by the patient’s nurse to determine if the source of the fluctuation may be explained, or if other co-founders existed to explain the change. Statistical analysis of the data was not done in this descriptive study; any relationship observed between PbtO₂ and the correlates monitored was simply described, using examples from the data sets in run charts to illustrate possible correlations.
Aim 3: Development of an algorithm

The final aim of the study was to develop an algorithm for treatment of patients with TBI based on information gathered from the literature review and patient data comparison. Using recommendations for the BTF (2007) guidelines for treatment of severe TBI, an algorithm was created that instituted treatment for parameters which fell outside of thresholds which have been shown to effect patient outcome, including ICP, CPP, and PbtO$_2$. 
CHAPTER IV

Results

Aim 1: Structure assessment and monitor introduction

The current standard of practice and protocols for patients with head injury did follow BTF (2007) guideline recommendations. The current order set for treating patients with TBI included a CPP protocol which initiated therapy based on elevation in ICP or decreases in CPP below a certain threshold. This set did not include standards and treatment modalities aimed at treating the measurements provided by the PbtO₂ monitor, a variable that was recently recommended in the 2007 guidelines. It was determined by the critical care advanced practice nurse that initiation of an updated treatment plan and new technology, the PbtO₂ monitor, may lead to improvements in patient outcome for this population.

Prior to purchasing the monitor, meetings to include neurosurgical staff in implementation of this technology were held, with discussion focusing on introducing this monitor. A request was made by the Chairman of Neurosurgery to gather more information about the monitor and PbtO₂, including how it is used, what is the significance of the variable, and whether the literature matched what we would find in our patients. This request, along with the hope to gain familiarity with the product, initiated the current study of structure and process in treatment of patients with TBI at this institution. This was done in cooperation with the advanced practice nurse on the unit, a critical care clinical nurse specialist. One monitor was obtained as a “loan” from
the company, and was used to familiarize both the neurosurgery team, consisting of residents and attending physicians, as well as the nursing staff on the trauma surgical unit, which was the only admitting unit for any patient determined to be a candidate for placement of the monitor. The PbtO\textsubscript{2} monitor was introduced to the neurosurgery team by a sales representative from LICOX\textregistered during educational meetings which focused on placement of the monitor and indications for use. The staff nurses on the unit which would be caring for the patients with the monitor were also educated on assisting the physicians with placement and general information about the monitor during formal inservices also done by the sales representative. Support from the product manufacturer was available at all times for any issues related to placement, use, technical support, or trouble-shooting of the monitor. Other aspects associated with introducing the monitor were addressed, such as purchase of a refrigerator as the probe needed to be kept cold to avoid drying out.

**Aim 2: Process assessment through literature review**

A review of the current literature on which physiologic variables tended to correlate with changes in brain tissue oxygen revealed that increases or decreases in PbtO\textsubscript{2} may be due to changes in oxygenation (PaO\textsubscript{2}), intracranial pressure (ICP), temperature, carbon dioxide (PaCO\textsubscript{2}), blood pressure, cerebral perfusion pressure, and blood count (Artru, Jourdan, Perret-Liaudet, Charlot, & Mottolese, 1998; Bader, 2006; Coles, Minhas, Fryer, Smielewski, Aigbirihio, Donovan et al, 2002; Gracias, Guillamondegui, Stiefel, Wilensky, Bloom, Gupta et al, 2004; Jeremitsky et al, 2003;
Lang et al, 2003; Meixensberger, Vath, Dings, Kunze, & Roosen, 2003; Smith, Stiefel, Magge, Frangos, Bloom, Gracias et al, 2005; Soehle, Jaeger, & Meixensberger, 2003; Stieffel, Udoetuk, Spiotta, Gracias, Goldberg, Malone-wilensky et al, 2006; Tokutomia, Morimoto, Miyagi, Yamaguchi, Ishikawa, & Shigemori, 2003; Valadka et al, 1998; van den Brink et al, 2000; van Santbrink, Maas, & Avezaat, 1996). Additionally, literature showed another important aspect which correlated with a measurement by the probe; the determination of brain death. Also important when considering treatment for patients is acquiring evidence which confirms that any further treatment is futile. Research showed that upon brain death in patients with the monitor, the value dropped to zero (van Santbrink et al, 1996, Palmer & Bader, 2005).

**Aim 2: Process and patient data assessment**

When a patient was determined a candidate for the probe by the neurosurgeon, he or she then placed the probe per product instructions into the penumbra region. The probe is placed along with the ICP monitor, and measured ICP, brain temperature, and PbtO$_2$. All patients underwent a CT scan to confirm proper probe placement. None of the patients experienced any adverse effects following placement of the probe. A period of stabilization is required before readings are accurate, which accounts for micro-trauma at the site; this time is approximately one hour after placement.

During the study period, a total of seven patients were admitted to the institution following a severe head injury and had the monitor placed. One patient who received the monitor had a subarachnoid hemorrhage from aneurysmal rupture, and was excluded
from the study; therefore only six patient data sets were included in the analysis, all with a diagnosis of TBI. All six patients were adults, with age range of 18-67 years. All the patients studied were also male, which is representative of the greater male to female ratio for TBI (BTF, 2007). Of the six patients, four of the patients died either directly from secondary brain injury or due to family decision to withdraw life support. One patient had a subdural hematoma (SDH) and underwent craniotomy for evacuation of the hematoma. The other five patients all had closed head injuries (CHI) which required no surgical interventions, though two were put into a pharmacologically induced coma with a medication called pentobarbital, which was assessed on an electroencephalogram by burst suppression. One patient only had the monitor in for a few hours before it was removed, as the patient already had signs of brain death and was declared brain dead later that day. Two patients had the monitor in between 24 to 96 hours, and the monitor was removed due to patient expiring. Of the three patients that had the monitor for greater than 96 hours, two survived and the monitor was removed due to patient improvement; the other patient died and had the monitor removed (TABLE 1).
### DEMOGRAPHICS

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
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</tr>
<tr>
<td>Age range (mean)</td>
<td>18-67 years (42y)</td>
</tr>
<tr>
<td>Gender</td>
<td>6 Male 0 Female</td>
</tr>
<tr>
<td>Overall mortality</td>
<td>66% (4 pts died)</td>
</tr>
<tr>
<td>Mechanism of injury</td>
<td>SDH 1 CHI 5</td>
</tr>
<tr>
<td>Time monitor in place:</td>
<td></td>
</tr>
<tr>
<td>&lt;24 hours</td>
<td>1 patient</td>
</tr>
<tr>
<td>24-96 hours</td>
<td>2 patients</td>
</tr>
<tr>
<td>&gt;96 hours</td>
<td>3 patients</td>
</tr>
</tbody>
</table>

**TABLE 1.** Study patient demographics.

*Oxygenation*

All patients were mechanically ventilated and monitored via pulse oximetry, which measures the oxygen saturation (SpO₂). Of the six patients, three experienced a drop in oxygenation measured by SpO₂ which significantly reduced the PbtO₂. Treatment for the decreased SpO₂ was instituted in one patient, who underwent manual ventilation with an ambu-bag and sterile suctioning through the endotracheal tube. In each patient, the recovery of SpO₂ was more rapid than the PbtO₂. Figure 4 is an example of the decrease in PbtO₂ with a drop in SpO₂ from one patient in the study.
FIGURE 4. Example of SpO₂ desaturation and drop in PbtO₂.

**Perfusion**

All patients had the CPP recorded as the MAP minus the ICP. Five of the six patients required institution of pharmacological support of the MAP to maintain the CPP above the 60mmHg as per the protocol parameters. The patient who did not require pressor support was declared brain dead soon after placement of the ICP/PbtO₂ monitor. Of the six patients, four patients experienced a significant drop in PbtO₂ related to decreased CPP from a drop in the MAP. Decreases in MAP were usually related to interventions such as medication titration or turning or suctioning the patient; however,
some drops did not coincide with any interventions documented by the nursing staff.

Figure 5 is an example of the decrease in PbtO$_2$ when the CPP drops due to a low MAP from one patient in the study.

![PATIENT 1 MAP/CPP](image)

**FIGURE 5.** Example of decreased PbtO$_2$ and drop in CPP due to low MAP.

*Intracranial pressure*

All patients underwent ICP monitoring along with the PbtO$_2$ monitor. All six patients had either sustained elevations of ICP or sudden spikes of elevated ICP which correlated significantly with a drop in PbtO$_2$. The drops in CPP which were unrelated to MAP were due to elevations of ICP. Patients were treated for elevations in ICP with a
the osmotic diuretic mannitol. Sustained elevations in two patients resulted in induction of a pentobarbital coma. Figure 6 is an example of the decrease in PbtO$_2$ when the ICP has a sudden increase from one patient in the study.

FIGURE 6. Example of sudden spike in ICP and immediate drop in PbtO$_2$.

Temperature

Temperature was monitored in all six patients, and although brain temperature was available, it was not recorded. Recorded temperatures used for the study were either core temperatures measured from the PAC, or bladder or esophageal temperatures. Although spikes in temperature were not shown to parallel with sudden drops in PbtO$_2$, ...
trending the temperature over time showed correlation between elevated temperatures and decreased in PbtO$_2$ in three of the patients, even during normal circadian rhythm fluctuations of temperature. Some patients received medication for elevated temperature above 38.5°C. None were treated with a standard cooling protocol using evaporative cooling. Figure 7 is an example of the fluctuations in PbtO$_2$ with body temperature changes from one patient in the study.

![PATIENT 1 TEMP](image)

**FIGURE 7.** Example of temperature changes and fluctuations in PbtO$_2$.

*Carbon dioxide and anemia*

Although carbon dioxide (PaCO$_2$) and hemoglobin were measured in these patients as a part of routine lab analysis for critically ill patients, neither measure was
monitored at a frequency that would allow determination of correlation in this patient population. Fluctuations in either value that occurred in 6 to 24 hour intervals did not provide sufficient data points to correlate the PbtO₂.

*Brain tissue oxygenation and brain death*

Overall, four of the six study patients died. Of the four patients who died, three were declared brain dead following protocols at the institution, either via clinical absence of brain function and an apnea test, or via a nuclear scan showing no perfusion. All three patients declared brain dead had a PbtO₂ of zero. Figure 8 is an example of one patient who was declared brain dead and had a PbtO₂ of zero.

![PATIENT 5 Brain Dead](image)

**FIGURE 8.** Example of PbtO₂ to zero in brain dead patient
Aim 3: Development of a treatment algorithm

After reviewing structure and process, the third aim was to develop an algorithm for treatment using recommendations in the literature and findings from the study of patient data correlates of PbtO₂. The algorithm begins with primary survey and initial assessment and management for any patient with suspected TBI. An important part of the algorithm is a standard method of determining which patients are appropriate as candidates to begin ICP/PbtO₂ monitoring as recommended by the BTF guidelines (2007). Once a patient is a candidate for treatment of severe TBI, initial therapy is listed, along with standard interventions and TBI goals for all patients. Next, the algorithm focuses on specific treatments and therapeutic goals aimed at optimizing the physiologic parameters, such as ICP, CPP, and PbtO₂, which should be monitored according to the BTF (2007) recommendations. Starting with the option of elevations in ICP, the algorithm directs treatments based on the threshold of 20mmHg and includes therapeutic interventions for values above this number. Additionally, sustained elevations in ICP are addressed, with treatment options as recommended by the BTF (2007) guidelines. Next, the option of decreases in PbtO₂ are addressed, with a treatment threshold of 20mmHg. Therapy and monitoring for this variable are directed at optimizing ICP, CPP, and the specific measures indicated in the literature review and during the patient data assessment, as well as what the BTF (2007) guidelines recommend. Finally, CPP is addressed, with a treatment threshold based on optimal ICP, PbtO₂, and fluid status or blood pressure. The target threshold for treatment of CPP is 60mmHg; however, if ICP is
elevated, treatment is directed at reducing ICP. If the \( \text{PbtO}_2 \) is above 20mmHg, CPP treatment threshold varies based on this value. Monitoring is aimed at measuring volume status via central venous pressure, once the patient has achieved euvolemia, augmentation of blood pressure using specific medications based on assessment with central venous pressure or a pulmonary artery catheter is indicated. As with any treatment algorithm, the goal is that it be used as a guide for treatment options based on standard of practice, and does not replace critical thinking or provider judgment when considering the best therapeutic management pathway (FIGURE 9).

After the literature and data were analyzed, the author gave a presentation of initial results and literature findings to the neurosurgical team in their monthly conference. Each patient was reviewed with the team, and specific data points of each correlate were extrapolated for illustrative purposes and shown in run chart format, as the figures above. Following the presentation, a quick discussion of the next steps, including introduction of the algorithm, completed the meeting.
FIGURE 9. Algorithm for treatment of patients with severe TBI

_Treatment Algorithm for Traumatic Brain Injured Patients_

**Primary Survey**: Airway, Breathing, Circulation, Disability, Exposure

Is patient suspected head injury?  

- Yes

  - **Initial Assessment**: GCS, motor strength/tone, cranial nerve, other systems, vital signs, CT scan head/CTL-spine, labs (ABG w/ lactate, CBC w/ plts, BMP w/ Mg/Phos, PT/PTT/INR)
  - **Initial Management**: Establish airway (RSI for GCS<8), IV access, NGT, Foley w/ temp probe, Mannitol 0.25-1GM/kg IV for posturing or unequal pupils
  - **Implementation of TBI Algorithm for patients with**:
    - GCS 3-8, abnormal CT scan, and abnormal clinical exam; OR
    - GCS 3-8, normal CT scan, and two or more of the following: age>40 years, unilateral or bilateral posturing, SBP<90mmHg
  - **Initial therapy**:
    - OR for patients with lesions/mass effect
    - Placement of Licox and ventriculostomy for ICP/PbtO2 monitoring
    - Placement of arterial/central catheters
    - See algorithm for specific interventions (optimize CPP, ICP, PbtO2)
    - Use electrolyte replacement order set, insulin gtt protocol, ICU sedation/analgiesia w/ propofol/morphine
    - Early enteral nutrition (or TPN consult if pt unstable/on pressors)
    - Phenytoin 100mg TID x 7days for patients with seizure activity
    - Transfer to ICU, q1 hr neuro checks

**Standard Interventions**

- Monitor VS, CPP, ICP, PbtO2
- Assure head and neck alignment
- Re-tape endotracheal tube tape (avoid taping at back of neck)
- Assure sedation and analgesia goals are met
- Position head of bed at 30 to 60° as appropriate for spine status, or in reverse Trendelenburg

**TBI Goals**

- SaO2 > 95%
- PCO2 35-40 mmHg
- CPP > 60 mmHg
- ICP < 20 mm Hg
- PbtO2 >20 mmHg
- Temperature 35-37°C
- Glucose 80-110mg/dL
ICP>20?

Yes

• Assure standard interventions* are in place
• Administer Mannitol 0.25-1 gm/kg or 3% NaCl (250mL x 1) on prn basis as ordered (hold for serum osmo >310, Na+ > 155)
• Notify House Officer for additional Mannitol dosing or for 3% NaCl
• Maintain serum Osmo < 310 mOsm/L and keep patient euvoletic (CVP 4-8 or PAWP 10-14)
• Drain CSF (if ventriculostomy in place)
• Consider repeating a brain CT scan
• Assess ABG. Administer therapeutic hyperventilation (PCO2 30-35) short term (GCS >5), if PbtO2 >20mmHg

No

• Continue to monitor
• Continue standard interventions*
• Ensure TBI goals** are met

PbtO2<20?

Yes

• Assure standard interventions are in place*
• Treat elevated ICP
• FiO2 100% (increase PaO2>100) until PbtO2>20 (assess pulmonary/treat status-CXR, sputum cx, abx, pleural effusions, ARDS)
• Increase PaCO2 if ICP<20
• Determine optimal CPP to increase PbtO2
• Ensure euvoletic (optimize fluids, use pressors)
• Assess H/H, Hct<30%, transfuse PRBC
• Consider hypothermia protocol

No

• Continue to monitor
• Continue standard interventions*
• Ensure TBI goals** are met

ICP>20, sustained after treatment?

Yes

No

ICP>20?

Yes

No

PbtO2<20?
CPP<60?  ICP>20?  Yes  • Treat elevated ICP  • Continue to monitor  • Continue standard interventions*  • Ensure TBI goals** are met  No  If CPP>50mmHg:  • Continue to monitor  • Continue standard interventions*  • Ensure TBI goals** are met  PbtO2>20?  Yes  If CPP<50mmHg  • Continue to monitor  • Continue standard interventions*  • Ensure TBI goals** are met  No  CPP>60 or PbtO2>20?  Yes  • Assure standard interventions are in place*  • Optimize treatments for PbtO2  • Optimize CPP, target >60mmHg; may titrate based on PbtO2:  IF hypovolemic: CVP < 4 - Attempt volume loading*** and attain lactate goals****  - Assess I&O, for goal I=O (polyuria, consider DI, check specific gravity, cc/cc replacement and DDAVP)  If euvolemic: CVP 4-8, insert PAC  - CVP>4, PAWP<8, attempt volume loading** for PAWP 8-12  - CVP>4, PAWP>8, CI<3: Treat any atrial tachy-arrrythmias as necessary, administer Dobutamine  - CVP>4, PAWP>8, CI>3: Administer α-agonist (neosynephrine, norepinephrine)  No  Volume Loading***  • Administer NS 250mL IV bolus every 15 minutes up to 2 liters  • Alternate with 5% albumin 250mL  • Notify house officer if volume goals are unachieved after 2 liters and assess for bleeding diathesis.  Lactate Goals****  • In multitrauma patients, continue with fluid resuscitation until 2 lactates are < 2, R/O new onset of bleeding.  • In isolated head injury patients: If lactate elevated and patient is an isolated head injury, continue to recheck lactate and assess trend. (Goal is trend to lactate < 2, not treat aggressively) Euvolemia is the goal and aggressive fluid resuscitation therapy may not be indicated.
CHAPTER V

Discussion

Aim 1: Structure

Assessment of structure showed the possibility for new technology to improve outcomes in patients with TBI. Although assessment of protocols at this institution did not show a lack in standard of practice, it was somewhat outdated, not always followed by the treating teams, and lacked individualized algorithms of treatment which may help tailor care in this complex patient population. Implementing a new technology which would require education and revision of the current protocol was seen as the primary goal involved in assessing structure. The properties of an organization which are objective are a part of structure. One element of structure that may have become an issue in this study was the fact that there was only one monitor due to the decision to wait to purchase until the initial data was evaluated. One patient received the monitor, was soon declared brain dead, and the same day another patient was admitted who needed it. Although this overlap was not an issue given that PbtO$_2$ was not being treated and the first patient was no longer in need of the monitor, in the future it will be important to obtain enough monitors to use for any patient who comes in with severe TBI, as indicated on the algorithm.

Aim 2: Literature review and patient data correlates

In reviewing the literature, several physiologic parameters that correlate with PbtO$_2$ also showed correlation in the six study patients. Some parameters indicated in the
literature were not monitored closely enough to establish any correlation in the study patients; however, since these variables are important in identifying and treating secondary injury after TBI, they were still presented to the neurosurgical team and were added to the algorithm for treatment. The four correlates observed in both the literature and the patient data included oxygenation, perfusion, intracranial pressure, and temperature. Findings from the literature of the other two correlates, PaCO$_2$ and anemia, are also discussed without a comparative analysis of patient data due to lack of adequate data measurement intervals. Additionally, literature revealed that a PbtO$_2$ of zero was present in patients who were soon after declared brain dead, which was also seen in all study patients who were brain dead (Palmer & Bader, 2005; van Santbrink et al, 1996).

**Oxygenation**

The purpose of ventilation is to exchange oxygen and carbon dioxide, to transport O$_2$ rich blood to the tissues and carry CO$_2$ away from the tissues. Normally, hemoglobin binds easily to O$_2$ and is released to the tissues, where CO$_2$ is picked up and carried back to the lungs. However, certain physiologic conditions can impact the affinity of oxygen to stay bound to hemoglobin, as well as the ability of oxygen to dissociate from hemoglobin at the tissue level. Factors which increase the ability of O$_2$ to be released to the tissues are acidosis, hypercapnia, and increased temperature. Factors which decrease the release of O$_2$ to tissues are alkalosis, hypocapnia, and decreased temperature. These factors also impact brain tissue oxygenation, as oxygen delivery to the brain is dependent on the affinity of O$_2$ to hemoglobin and on the vasculature. As such, it is important to
maintain adequate systemic oxygenation to ensure the brain tissue is receiving the \( \text{O}_2 \), as well as looking at aspects which may impact the affinity of \( \text{O}_2 \) to hemoglobin at the tissue level (Bader, 2006).

Both in previous studies and the current study, oxygenation seems to be correlated with \( \text{PbtO}_2 \) (Gracias et al, 2004; van Santbrink et al, 1996). Several studies have found a high correlation between hypoxia and increased mortality in patients with TBI (Barton et al, 2005; Chestnut et al, 1993; Jeremitsky et al, 2003; Stochetti et al, 1996). Patients with severe TBI also have a high risk of developing pneumonia, as many require mechanical ventilation for airway protection or lung injury. One study found patients with isolated closed head injuries to have a higher incidence of pneumonia, and to have pneumonia occur earlier than in other patient populations (Hsieh, Bishop, Kubilis, Newell, & Pierson, 1992). Additionally, standard treatments which aim to maintain a CPP above 60mmHg, using aggressive fluid resuscitation and pressors to achieve an optimal MAP, also put patients at risk for developing pulmonary edema and adult respiratory distress syndrome (ARDS) (Meixensberger et al, 2003). Recognizing the risk this patient population has for issues of decreased systemic oxygenation, as well as the correlation between hypoxia and mortality, exemplifies the importance of early recognition and treatment of this correlate.

**Perfusion**

Perfusion of tissues is dependent on adequate blood flow. In patients with TBI, changes in CBF can greatly impact outcome (Chestnut et al, 1993; Jeremitsky et al, 2003;
Stochetti et al, 1996). As discussed earlier, it is important to consider the phases of blood flow following injury and correlate treatment with these phases. The hydraulic measure of CPP estimates cerebral perfusion and optimizing this measure to a goal number has been used as variable for treating patients with TBI. Typically, volume resuscitation and medications which augment MAP are standards of practice; however, the addition of PbtO$_2$ as a variable for treatment allows for a direct measure of tissue oxygenation and adds the ability to fine tune treatment. Studies have found that low perfusion is common in patients following TBI, and that even one episode of systolic blood pressure less than 90mmHg leads to a 15% increase in mortality (Jeremitsky et al, 2003). Another study examined cerebral autoregulation after TBI and found that CPP did correlate with PbtO$_2$ when autoregulation was impaired. Adjustments in CPP when autoregulation was intact did not effect the PbtO$_2$ (Artru et al, 1998; Lang et al, 2003; Stiefel et al, 2006). In the current study patients, CPP did seem to correlate with PbtO$_2$, especially in decreases in MAP. Therefore, treatment for CPP in the algorithm is now titrated based on the PbtO$_2$, a more direct measure of oxygenation and perfusion.

**Intracranial pressure**

Elevated ICP is associated with worse outcomes in patients with TBI. Severely elevated ICP leads to impairment of autoregulation and ischemia, as well as risk for brain herniation (Valadka & Robertson, 2007). ICP monitoring has been a standard of practice for this patient population; however, the added information provided by PbtO$_2$ monitoring augments treatment with this variable by adding the perspective of oxygen
delivery. Severe spikes and sustained elevations in ICP have been correlated to decreases in PbtO₂ in the literature, and also seemed to correlate in patients in this study (Bader, 2006; Stiefel et al, 2006; Valadka & Robertson, 2007). Therefore, treatment for ICP elevation above 20mmHg is included in the TBI algorithm, as recommended by the BTF (2007) guidelines.

**Temperature**

Although therapeutic hypothermia has not been added as a recommendation in the BTF (2007) guidelines, mostly due to lack of adequate evidence of improved outcomes, it is important to maintain normothermia in this patient population. Increased temperature is associated with increases in ICP and cerebral metabolism, as well as decreased PbtO₂ (Bader, 2006; Jeremitsky et al, 2003; Tokutomi et al, 2003). Studies which show improved outcome related to temperature have determined the optimal temperature for this patient population is between 35-37°C (Tokutomi et al, 2003). Fluctuations in temperature also seemed to correlate with PbtO₂ in the study patients. As such, one of the goals of treatment for TBI in the algorithm is maintaining normothermia.

**Carbon dioxide**

Changes in carbon dioxide were not measured in the patient population at intervals that allowed determination of PaCO₂ as a correlate of PbtO₂. As discussed previously, changes in PaCO₂ effect both the affinity of oxygen for hemoglobin, as well as systemic pH. Another effect of changes in blood PaCO₂ relates to changes in vascular
diameter in the cerebral blood vessels. CBF is extremely responsive to increases or decreases in PaCO$_2$; elevations lead to vasodilation, which can be dangerous in patient with high ICP. Traditionally, treatment for patients with high ICP included inducing hypocapnia to levels of 25-30mmHg. However, vasoconstriction also reduces CBF and oxygen supply. For every 1mmHg decrease in PaCO$_2$, there is a 2-3% reduction in CBF (Bader, 2006; BTF, 2007; Coles et al, 2002; Valadka & Robertson, 2007; van Santbrink et al, 1996). Considering the phases of CBF following TBI, as well as the increased metabolic rate, it is important to maintain PaCO$_2$ at normal levels. Hyperventilation to reduce ICP is only recommended as a transitory measure during impending herniation. If hyperventilation is considered, cerebral oxygenation should be monitored (BTF, 2007). Given the effect that changes in PaCO$_2$ have in the delivery of oxygen to the brain tissue, it is important to monitor this variable either through assessment of arterial blood gas, or an end-tidal CO$_2$ monitor. Studies have found PaCO$_2$ to be a correlate of PbtO$_2$; therefore, maintaining normocapnia (35-40mmHg) is one of the treatment goals in the algorithm (Bader, 2006; Johnston, Steiner, Gupta, & Menon, 2003).

**Anemia**

Hemoglobin and hematocrit were not included as a correlates in the study population due to lack of frequent monitoring, as it was not standard practice at the institution. Literature reveals that transfusion of fresh packed red blood cells in patients with TBI who have a hematocrit less than 30% or hemoglobin <10 g/dL increases local cerebral tissue oxygenation as measured by a PbtO$_2$ monitor (Bader, 2006; Smith et al,
Given this information, transfusion of packed red blood cells is one of the treatment options on the algorithm that is indicated for a drop in PbtO$_2$.

**Brain tissue oxygenation and brain death**

An important aspect of treating patients is the consideration of when treatment is no longer appropriate. Several methods for the determination of brain death are used, including a clinical exam that is absent of all brain stem reflexes, as well as apnea testing or nuclear studies of cerebral blood flow. One possible future use for the PbtO$_2$ monitor is determination of brain death, as a measure of zero on the monitor seems to be a correlate of brain death. Other studies have found a high correlation between PbtO$_2$ decreases to zero and brain death, as was also evident in the patients in the current study (Palmer & Bader, 2005; van Santbrink et al, 1996). One study by Valadka, Goodman, Gopinath, Uzura, & Robertson (1998) found that changes in levels of biochemical indicators of ischemia associated with brain death occurs after the PbtO$_2$ monitor reads zero.

**Aim 3: Algorithm for treatment of patients with TBI**

The final aim of the study is the development of a treatment algorithm for patients with TBI. Although outcome was not measured in the study patients, the goal of creating a treatment protocol in the form of an algorithm that individualizes and optimizes treatment based on several physiologic parameters impacts process in a way that may directly improve patient outcome. The algorithm that was created addresses several
variables, including PbtO$_2$, and is based on recommendations by the BTF (2007) guidelines. The format of an algorithm allows for individualized treatment based on patient status, rather than an order set which directs care; it includes the new variable PbtO$_2$. Some of the information from the algorithm was translated from the previous order sets, although the template and inclusion of treatment for PbtO$_2$ were both newly designed. As far as the author is aware, use of a treatment algorithm that includes PbtO$_2$ as a variable has not been done previously at this institution, making it a unique addition to quality improvement measures.

Patients with critical brain injuries require care that is optimized by an interdisciplinary team that understands the physiology behind the injury and can work together to achieve the goals established at each phase of this illness. Understanding the physiology of patients with TBI is complex, and some of the factors which are addressed in the algorithm include management of elevations in ICP, titration of CPP to maintain optimal PbtO$_2$, and using euvolemia and medications specific to the patient status as therapeutic interventions for maintaining MAP and CPP. Additionally, establishment of specific interventions aimed at maintaining PbtO$_2$, such as transfusion of blood products and maintaining normothermia and normocapnia, are included as part of the algorithm. As with any treatment algorithm, cooperation between treating teams of providers, interdisciplinary communication, and skilled assessments precluding intervention are keys to success.
Limitations of study

Although the data seemed to correlate with PbtO₂ as suggested by findings in the literature, several limitations in this study must be addressed. As with any retrospective review of patient charts, the lack of control or tested relationships leaves room for confounders to apparent relationships. Since they are only observed and not tested, there is the possibility that other factors may have been equally influential in causing changes in PbtO₂ that were not discussed. This includes mechanism of injury, variations in time lapsed from admission to placement of the monitor, differences in approach to treatment between and within the treatment teams, different skill level in monitor placement by the neurosurgeons, and variations in nursing care. Additionally, a sample size of only six patients is too small to be generalizable to any larger patient population. In depth demographic data were also not addressed, and may impact the patient outcomes beyond what was discussed in this study.

Since the aims of this study did not include testing any hypothesis, but evaluating the quality of standard protocols at the institution by comparing them to current literature on PbtO₂ and introducing new technology to practitioners; the lack of tested relationships in the correlates examined in this patient population does not diminish the value of the aims of the study. Instead, it further supports the use of the technology, the confidence of the practitioners in assessing patient goals, and the creation of a new protocol for treatment of these patients. Additionally, it may help build the ground work for doing a
larger, prospective trial using the PbtO₂ monitor in patients with TBI, not unlike a pilot study.

Summary

Although the study was only the first step in evaluating correlation between a small sample of the TBI patient population and current literature, several correlations emerged among these basic comparisons. This aided in supporting the institution of the PbtO₂ monitor, as well as helping to introduce it to the physicians and nurses who will be using it. Additionally, knowledge about the literature supported the creation of the algorithm for treatment using the PbtO₂ as a treatment end-point. Ultimately, literature supports the use of this technology to further allow individualized treatment of this patient population, and is recommended in the BTF guidelines (2007). These first steps are the initial part of APN guided research on quality assessment of a very complex treatment process of medical care provision. Looking at structure, process, and eventually, outcome for these patients will help to further improve the quality of care for TBI patients at this institution. Although outcome was not specifically assessed for these patients, and with the small sample size, data would be limited in making any inferences, the idea behind Donabedian’s (1966) framework is that outcomes are being improved by directing changes in structure and process. A formal study with a larger number of patients after implementation of the algorithm will further support the idea that outcome has improved, and thus high quality care provided.
REFERENCES


