ANALYSIS OF SEDATION SCALES IN ASSESSING SEDATION LEVELS OF THE
TRAUMATIC BRAIN INJURED PATIENT

BY

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Abstract

The objective of this literature review is to examine current research to support the use of sedation assessment scales for assessing sedation in the traumatic brain injured patient. Sedative administration is necessary in the intensive care unit to facilitate tolerance of interventions and mechanical ventilation. The traumatic brain injured patient is a subset of critical care patients that often require prolonged use of sedatives and are at increased risk for sedative related injury. The use of sedatives in the traumatic brain injured patient can facilitate control of intracranial pressure and decrease neurological injury, however, the consequences of improper sedative administration can be deadly.

In order to minimize the occurrence of improper sedation the use of a sedation assessment scale has become standard practice. Structured assessments of sedation aide medication administration and provide parameters for titration. There are many sedation assessment tools available for use in the critical care but none have been deemed the gold standard. Current research is insufficient in supplying a valid scale for use in the critical care and essentially absent in supplying a scale specific for evaluating sedation in the traumatic brain injured patient. The goal of this paper is to analyze current research and identify a possible sedation assessment scale that could best meets the needs of the neurologically injured.
CHAPTER ONE-INTRODUCTION

Introduction

Sedatives and analgesics are utilized routinely among critical care patients in an effort to minimize the physiological stress response, provide anxiolysis, facilitate ventilation, and expedite care. Patients that suffer a traumatic brain injury (TBI) represent a subset of critical care patients that may require sedation. Many patients with TBI require sedation to provide nursing care, perform invasive medical procedures, optimize mechanical ventilation, and facilitate cerebral perfusion. The improper use of sedatives in this population can have adverse effects and worsen the primary neurological injury (Diawai, Thoyre, & Auyong, 2007).

Evidence based guidelines for the use of sedation has been published by the American College of Critical Care Medicine and Society of Critical Care Medicine to guide therapies (Rhoney & Parker, 2001). The evidence used to create these guidelines was extracted from the diverse populations of critical care with exclusion of patients with neurological injury. The physiological demands of the neurological system, the brain in particular, and its associated injuries require a specialized approach to sedation and analgesia. Therefore the current published guidelines are not safely applicable to the TBI patient, leaving this vulnerable population at risk for sedation related injury (Jacobi et al., 2002). The purpose of this report is to first review TBI, sedation (including history, sedation levels, agents used for sedation), complications of sedation in TBI, sedation guidelines, and sedation scales currently used in practice. Based on this review, the author will critically analyze the use of current sedation scales in assessing sedation levels in the traumatic brain injured patient.
Incidence and Fiscal Impact of Traumatic Brain Injury

Annually 1.7 million people suffer a TBI in the United States. Of these, 275,000 are hospitalized, 1.36 million are treated and released, and 52,000 will die (Faul, Xu, Waid, & Coronado, 2010). 100,000 TBI patients will have long term deficits with 2%, or 5 million, of the U.S population requiring permanent assistance to perform activities of daily living (Faul et al., 2010). Adults 75 years old and greater account for the majority of hospitalizations and deaths as a result of TBI (Faul et al., 2010). Men are more affected than women at a rate of 2:1 and African Americans are at highest risk of TBI associated death (Faul et al., 2010). TBI is most likely to occur in the very young, adolescent, and the old with the majority of injuries occurring at ages 0-4, 15-24, and 60 or greater (Faul et al., 2010).

Falls are the leading cause of TBI accounting for 35% of hospitalizations and motor vehicle crashes are the leading cause of death from TBI accounting for 17% of hospitalizations (Faul et al., 2010). Firearms and assault attribute to 13% of all TBI (Faul et al., 2010). Direct contact occurring with striking or being struck, such as when a tree branch falls on the head, account for 16% and there is 21% of the TBI population with unknown etiology (Faul et al., 2010).

The research produced by Faul et al. (2010) estimates that the United States spends 60 billion dollars annually, 31.7 billion in hospitalization costs and another 16.6 billion in costs associated with fatalities of TBI patients. The total cost associated with acute care and rehabilitation of TBI patients, without consideration for indirect cost to families, is about 9 to 10 billion annually (Faul et al., 2010). A person suffering a TBI can incur over 4 million dollars in medical costs over their lifetime (Faul et al., 2010). Because TBI does not often result in
spontaneous death the cost of dying from a TBI can be exorbitant, reaching 454,000 dollars in medical bills for the surviving family members to endure (Faul et al., 2010).

**Pathophysiology of Traumatic Brain Injury**

The cranium is a non-expandable, hollow, bony structure that encompasses and protects the brain. The cranial vault consists of 10% blood, 10% cerebral spinal fluid (CSF), and 80% brain tissue, each of these components contribute to intracranial pressure (ICP; Porth, 2005). A normal ICP is 0-15mmHg and continually fluctuates with respiration and activity, this fluctuation is possible because of reciprocal compensation of the cranial components; a minimal increase in one component will result in a decrease in one or both of the other components (Porth, 2005). Blood and CSF are the primary compensatory mechanisms, brain tissue has little mechanism for change (Porth, 2005). Increase in ICP is buffered by movement of CSF into the spinal subarachnoid space and by increased absorption (Porth, 2005). The cerebral vasculature constricts in response to rising ICP to move blood out of the venous bed and back into circulation (Porth, 2005). Variations in any of the three components that exceed the compensatory mechanisms ability to balance pressure will elevate the ICP; once these mechanisms have failed small changes produce extensive alterations in ICP (Porth, 2005). For the scope of this paper the discussion related to variations in ICP will be limited to intracranial hemorrhage as a result of trauma but variations in ICP can also occur from tumor, hydrocephalus, metabolic derangement, degenerative disorders and stroke (Porth, 2005).

Cerebral perfusion pressure (CPP) is the pressure gradient created by the flow of blood from the internal carotid arteries into the subarachnoid veins and is the amount of pressure that is required to perfuse the brain (Porth, 2005). Normal CPP is 70-100 mmHg; it is thought that
pressures below 70 mmHg results in decreased brain tissue perfusion, glucose delivery, oxygen delivery, and removal of metabolic waste (Porth, 2005). Science has yet to identify an optimal level of CPP, current recommendations are a CPP between 60-70mmHg, with research emphasizing a more personal approach based on cerebral blood flow and cerebral metabolic oxygen consumption (Brain Trauma Foundation & American Association of Neurological Surgeons, 2008). The cerebral metabolism consumes 20% of the body’s oxygen to function and uses glucose as the primary source of energy (Porth, 2005). Uncompensated increases in ICP will reflect a corresponding decrease in CPP which correlates with ischemia of brain tissue (Porth, 2005). Ischemia will deplete brain tissue glucose in 2-4 minutes with depletion of cellular energy at 4-5 minutes producing lactic acid (Porth, 2005). Energy depletion results in loss of the brain’s ability to maintain ionic gradients, such as the sodium-potassium pump (Porth, 2005). Failure of the ionic gradients results in an influx of sodium, potassium, and calcium ions (Porth, 2005). Excess sodium causes neuronal swelling known as cytotoxic edema, further increasing ICP and decreasing CPP (Porth, 2005). Excessive calcium stimulates a cascade of events that culminate in release of intracellular enzymes that cause cellular destruction (Porth, 2005). The excessive activity of the excitatory neurotransmitter glutamate further contributes to the calcium cascade, glutamate is the primary excitatory neurotransmitter in the brain and functions in memory, cognition, movement, and sensation (Porth, 2005). Over stimulation of the receptor sites for glutamate occur with injury, these receptor sites are paired with receptor operated ion channels, which cause calcium mediated neuronal cell death when over-stimulated (Porth, 2005).

Brain injuries can be separated into 2 categories, primary and secondary injury. Primary injury is the cerebral damage that occurs during the initial impact. The primary injury results in
displacement of cerebral structures related to active hemorrhage, this is known as the core ischemic zone (Corte, n.d.). The core ischemic zone has a blood flow below 10-20% of normal, resulting in rapid tissue ischemia and cellular death (Corte, n.d.). Brain tissue in this zone is generally unsalvageable regardless of intervention; once a neuron has died it can not be regenerated (Corte, n.d.). The area of secondary injury in TBI is known as the penumbra, which lies between well perfused brain tissue and the core ischemic zone (Corte, n.d.). The penumbra is fed by collateral blood vessels and has the potential to maintain viability if adequate perfusion and oxygenation is supplied to the area (Corte, n.d.). The penumbra is very sensitive to fluctuations in cerebral metabolic oxygen consumption (CMRO2) and cerebral blood flow (CBF; Corte, n.d.). Small variations of these parameters will propagate ischemia-induced biochemical events and result in expansion of the penumbra (Corte, n.d.). Ischemia that occurs early in the injury phase is associated with poor outcomes and increased mortality (Corte, n.d.). The following section will discuss sedation and the effects of sedative agents of the pathophysiology of TBI.
CHAPTER TWO-SEDATION

Sedation

Sedation is the process of administering specific drug classes to patients in order to induce a sense of calm, amnesia, or drowsiness (Neuropharmacology, 2000). TBI patients often require the use of sedatives to facilitate care and improve neurological outcomes. The following sections will review the history, levels, medications, guidelines, and influence on cerebral injury.

History of Sedation

The 1800’s brought about the dawn of general anesthesia; Dr. Davey discovered the use of nitrous oxide for painless tooth extractions and Dr. Long was the first documented physician to use ether as an anesthetic prior to surgery (McPhee, 2008). In 1884 Sigmund Freud proposed the use of cocaine to numb specific body parts, bringing about the concept of local anesthesia (McPhee, 2008).

Since its inception, anesthesia has been witness to great pharmaceutical advancements. Multiple drugs and administration techniques have been discovered and implemented with exemplary outcomes. The field of nocioception and anesthesia has grown exponentially bringing with it the need for specialized education and monitoring (McPhee, 2008).

Levels of sedation

There are four levels of sedation used in the treatment of patient’s anxiolysis, conscious sedation, deep sedation, and general anesthesia (American Society of Anesthesiologists, 2004). All levels of sedation can be utilized with the neurologically injured patient and can potentate injury if the medications used are administered inappropriately. Anxiolysis is used for procedures that require the patient to be relaxed but remain responsive. Procedures that use
anxiolytic therapy include minor surgical operations, radiologic exams, bone marrow aspiration, and central line placement. Benzodiazepines, analgesics, and barbiturates are the most frequently used medication to achieve this state (American Society of Anesthesiologists, 2004).

Conscious sedation is defined as a state of diminished level of consciousness arousable by verbal or light tactile stimulus. Cardiovascular stability and spontaneous ventilation are monitored using continuous pulse oximetry and intermittent vital signs (American Society of Anesthesiologists, 2004). This state of depressed consciousness is beneficial in procedures that require the patient to be immobile for short periods. Procedures that use conscious sedation include endoscopy, magnetic response imaging, and various radiological exams. Medications to achieve this state are the same as for anxiolysis but are either used in combination or at higher doses.

Deep sedation is a level of depressed consciousness that requires repeated deep tactile stimulation to achieve a purposeful response. Cardiovascular stability is preserved but spontaneous respirations may become compromised and require the use of a bag-valve mask, nasopharyngeal airway, or laryngeal mask airway to maintain adequate ventilation (American Society of Anesthesiologists, 2004). Procedures that use deep sedation include surgeries that incorporate local anesthesia, airway intubation, and patients requiring mechanical ventilation (American Society of Anesthesiologists, 2004). Medications used are a combination of previously discussed medications as well as sedative-hypnotics and short acting paralytics (Jacobi et al., 2002).

General anesthesia produces a complete loss of consciousness, requiring mechanical ventilation for airway support as well as medications to support cardiovascular constancy.
General anesthesia is used for invasive operations that require complete anesthesia. Analgesics, neuromuscular blockade, and sedative hypnotics are used to induce and maintain sedation (American Society of Anesthesiologists).

**Sedation of Traumatic Brain Injury Patients**

Patients with TBI represent a subset of critical care patients at increased risk for sedation-related complications. Patients suffering TBI present with cognitive, sensory, and motor function impairment that makes the use of sedative agents necessary. They often require intubation for airway protection and have prolonged mechanical ventilation times related to their mechanism of injury (Mirski, Muffelman, Ulatowski, & Hanely, 1995). The goal of sedation in the TBI patient is to prevent secondary neuronal injury related to increased ICP or insufficient CPP and provide comfort while preserving an intact neurological assessment which remains the gold standard for assessing neurological decline (Mirski et al., 1995).

The use of sedative agents in this population facilitates mechanical ventilation and decreases ICP. However, over-sedation causes systemic vasodilation and decreased cerebral blood flow increasing tissue infarction and neurological damage which results in expansion of the secondary injury (Rhoney & Parker, 2001, Mirski et al., 1995). Under-sedation results in increased blood flow and cerebral metabolic oxygen consumption causing increased ICP, CPP, and tissue infarction (Rhoney & Parker, 2001, Mirski et al., 1995). Medication titration must be done cautiously and with the idea of a predetermined sedation level of a sedation assessment scale (Rhoney & Parker, 2001).

Sedation effects cerebral metabolism and intracranial elasticity by changing cerebral vasculature and blood flow which results in decreased CPP, increased ICP, and neuronal death.
Understanding the elaborate interactions of sedatives on cerebral physiology is imperative in guiding the use of these medications in this vulnerable population. The Brain Trauma Foundation and American Association Of Neurological Surgeons (2008) published guidelines regarding sedation and anesthesia of the TBI population stating that the recommendations are considered optimal and can not be applied to all TBI patients related to their unique injuries and physiological requirements. Additionally, because there is limited research of sedation in the neurotrauma patient, the guidelines were based on literature that was produced from healthy volunteers or elective neurosurgical patients (Department of Surgical Education, 2008). The following section will discuss sedative agents and their effects on cerebral physiology.

*Sedation Medications*

The various classes of sedation agents described in this section are commonly used in the TBI population and include narcotics, sedative-hypnotics, and neuromuscular blockades.

**Narcotics** are used to decrease or alleviate the sensation of pain and are frequently used to produce anxiolytic state. Commonly used narcotics include fentanyl, morphine, hydromorphone, and meperidine. Analgesics vary in potency, distribution, duration, and metabolism. They are most often given in conjunction with other sedative medications to alleviate the physical complications of pain which include; tachycardia, elevated myocardial oxygen consumption, hypercoaguability, and immunosuppression (Jacobi et al., 2002). Opioids cause a reduction of CMRO2, CBF, and ICP in normocarbic patients. Transient increases in ICP have been observed with the use of fentanyl, without concomitant cerebral ischemia, and should be used cautiously in the TBI population (Rhoney & Parker, 2001).
Sedative-hypnotics Benzodiazepines are sedative-hypnotics that facilitate retrograde amnesia, blocking the formation of unpleasant memories that occur during traumatic or painful events (Jacobi et al., 2002). This drug class also has anticonvulsant properties and can be used to stop seizure activity. Benzodiazepines have no analgesic property but have a potentiating effect with co-administration of analgesics. Medications in this drug class vary in potency, distribution, duration, and metabolism (Jacobi et al., 2002). Central nervous system effects depend largely on the drugs ability to bind to the benzodiazepine receptor sites (Jacobi et al., 2002). Possible neurological complications associated with benzodiazepine use are decreased CBF with concurrent drop in CPP and decreased CMRO2 (Rhoney & Parker, 2001). ICP is generally not affected by this drug class. Benzodiazepines also cause alpha wave slowing on the electroencephalogram (EEG) and the long half life of intermediate and long acting benzodiazepines impede frequent neurological assessment (Rhoney & Parker, 2001). Drugs included in this category are diazepam, lorazepam, and midazolam. These drugs can be given intermittently or as continuous infusions.

Barbiturates are a sedative-hypnotic that act to increase GABA response in the central nervous system resulting in a sensation of euphoria and relaxation (Dipiro et al., 2005). This class of drug is considered a short acting sedative-hypnotic and is associated with a high addiction rate; they can be given intermittently or as a continuous infusion (Rhoney & Parker, 2001). Barbiturates depress neurological function, including the brain stem, which results in depressed EEG activity and seizure cessation (Rhoney & Parker, 2001). A 50% reduction in CBF and CMRO2 is seen with this drug class along with increased cerebral vascular resistance and a decreased ICP (Rhoney & Parker, 2001). While barbiturates protect against cerebral ischemia
they obliterate the ability to assess neurological status and therefore should be used only after exhausting all medical and surgical resources (Brain Trauma Foundation & American Association of Neurological Surgeons, 2008; Rhoney & Parker, 2001). Phenobarbital, pentobarbital, thiopentol are examples of barbiturates (Dipiro et al., 2005).

Propofol, a sedative-hypnotic, is an alkyphenol that has similar action to barbiturates in that it depresses the central nervous system and produces feelings of well being. The drug has no effect on pain receptors but can be potentiated with concomitant analgesic use. Full recovery from Propofol administration generally takes 10-15 minutes, though recovery may be prolonged with continuous administration beyond 24 hours (Mirski, Muffelman, Ulatowski, & Hanely, 1995). Propofol’s short half life makes it an optimal choice for sedation in the TBI population as it allows for frequent neurological assessments with minimal stress response (Rhoney & Parker, 2001). Decreased CMRO2, CBF, and ICP are seen with the use of this drug and slowing of the EEG is also evident (Rhoney & Parker, 2001). Hypovolemic patients with elevated ICP can have decreased CPP with the use of propofol which is related to systemic hypotension from Propofol induced systemic vascular relaxation (Rhoney & Parker, 2001). Another concern related to this drug is Propofol infusion syndrome (PRIS). PRIS is associated with dosages of >4mcg/kg/hr or use exceeding 48 hours and results in severe metabolic acidosis, rhabdomylosis, hyperkalemia, hypertriglyceridemia, renal failure, hepatomegaly, cardiovascular collapse, and death (Zaccheo & Bucher, 2008). As of 2006 nine TBI cases of PRIS were documented, all resulting in death (Zaccheo & Bucher, 2008). TBI patients are at increased risk for PRIS related to the prolonged use and dosage requirements to maintain sedation and decrease ICP (Corbett, Moore, Rebuck, Rogers, & Greene, 2006).
Ketamine is a short acting sedative-hypnotic-analgesic that induces hypnosis and amnesia; it also acts on opioid binding sites providing analgesia. Ketamine has little cardiac or respiratory effect and can be given safely to hemodynamically unstable patients (Mirski et al., 1995). A side effect of this medication is its effects on the limbic system, which place the patient in a disassociative state with hallucinations lasting for 2-3 hours; effectively ceasing the ability to assess the neurological status (Mirski et al., 1995). Ketamine increases CMRO2, CBF, ICP, CPP and the seizure threshold which places the TBI patient at risk for secondary injury (Rhoney & Parker, 2001).

Dexmedetomidine hydrochloride (Precedex) is a new sedative agent that is an effective sedative and analgesic, with anesthetic- sparing properties. Precedex has been shown to reduce anesthesia and sedation requirements with minimal effect on level of consciousness (Rhoney & Parker, 2001). CBF is decreased without changes in CMRO2 which suggests that Precedex may be unsuccessful as a neuroprotectant; additional research is required for confirmation of current results (Rhoney & Parker, 2001).

**Neuromuscular Blockade** There are two types of neuromuscular blockade, non-depolarizing and depolarizing. Both act on the neurotransmitter acetylcholine, in different ways, to produce paralysis. They require concurrent mechanical ventilation and hemodynamic supporting agents (Dipiro et al. 2005). ICP, CPP, CMRO2 and CBF are minimally affected during neuromuscular blockade (Neuropharmacology, 2000). Examples of neuromuscular blockades include Vecuronium, Rocuronium, and Cisatracurium. Table one summarizes commonly used sedation medications and the effects of the medications on cerebral physiology.
<table>
<thead>
<tr>
<th>Agent</th>
<th>Infusion Dose</th>
<th>Half-Life</th>
<th>Neuro-specific Adverse Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fentanyl</td>
<td>.7-10mcg/kg/hr</td>
<td>1.5-6hrs</td>
<td>R rigidity with high doses can cause increased ICP. Decreased CBF and CMRO2</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>7-15mcg/kg/hr</td>
<td>2-3hrs</td>
<td>Decreased CBF, ICP, CMRO2. Can cause neuroexcitation and seizures</td>
</tr>
<tr>
<td>Meperidine</td>
<td>75-100mg</td>
<td>3-4hrs</td>
<td>Decreased CBF, ICP, CMRO2. Can cause neuroexcitation and seizures</td>
</tr>
<tr>
<td>Morphine</td>
<td>.01-.15 mg/kg/hr</td>
<td>3-7hrs</td>
<td>Decreased CBF, ICP, CMRO2</td>
</tr>
<tr>
<td>Diazepam</td>
<td>.12-.8 mg/kg/24</td>
<td>20-50hrs</td>
<td>Depressed neurological activity Decreased CPP and CMRO2</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>2-5mg/hr</td>
<td>12-15hrs</td>
<td>Depressed neurological activity Decreased CPP and CMRO2</td>
</tr>
<tr>
<td>Midazolam</td>
<td>50-500mcg/kg/hr</td>
<td>.8hrs</td>
<td>Depressed neurological activity Decreased CPP and CMRO2</td>
</tr>
<tr>
<td>Phenobarbital</td>
<td>2-3mcg/kg/24</td>
<td>40-120hrs</td>
<td>Decreased CPP, ICP, CMRO2 Depressed neurological function</td>
</tr>
<tr>
<td>Pentobarbital</td>
<td>1-3mg/hg/hr</td>
<td>15-50hrs</td>
<td>Decreased CPP, ICP, CMRO2 Depressed neurological function</td>
</tr>
<tr>
<td>Thiopentol</td>
<td>3-7mg/kg</td>
<td>12-26hrs</td>
<td>Decreased CPP, ICP, CMRO2 Depressed neurological function</td>
</tr>
<tr>
<td>Propofol</td>
<td>5-50mcg/hg/min</td>
<td>2-60min</td>
<td>Decreased CPP, ICP, CMRO2 Depressed neurological function</td>
</tr>
<tr>
<td>Ketamine</td>
<td>.5-2mg/kg</td>
<td>2.5hrs</td>
<td>Increased CPP, ICP, CMRO2 Increases seizure threshold</td>
</tr>
<tr>
<td>Precedex</td>
<td>.2-.7mcg/kg/hr</td>
<td>2hrs</td>
<td>Decreased CBF</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>.01-.015mg/kg</td>
<td>50-90min</td>
<td>Minimal neurological effect</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>5-2.5mcg/kg/min</td>
<td>14-18min</td>
<td>Minimal neurological effect</td>
</tr>
<tr>
<td>Cisatracurium</td>
<td>.5-10mcg/kg/min</td>
<td>10-30min</td>
<td>Minimal neurological effect</td>
</tr>
</tbody>
</table>

Optimal Sedation and Complications of Sedation in Traumatic Brain Injured Patients

Multiple clinical studies have been performed to determine which medications provide optimal sedation with minimal side effects. Ostermann, Keenan, Seiferling, and Sibbald (2002) reviewed 49 control studies in an effort to identify best sedation practice. The authors concluded that the dosage and drug for optimal sedation remains inconclusive and that current practice guidelines are suggestive rather than absolute (Ostermann et al., 2002). What has been unequivocally determined from the clinical trials is that complications are experienced by patients who are over or under-sedated (Ostermann et al., 2002). Study results have revealed that under-sedation leads to increased potential for self-extubation, catheter removal, ventilator dysynchrony, vital sign instability, and post-traumatic stress disorder (Olsen, Thoyre, Peterson, & Graffagnino, 2009; Ostermann et al., 2002). Under sedation causes an increase in CBF, CPP, and ICP which can result in cerebral edema and propagation of neuronal damage (Rhoney & Parker, 2001). Over-sedation exposes patients to extended hospitalization, muscle weakness, prolonged ventilation, with increased need for tracheostomy, and other complications (Jacobi et al., 2002). Neurologically, over-sedation can lead to decreased CBF and CPP without changes in CMRO2 resulting in neuronal cell death (Rhoney & Parker, 2001).

Sedation of the neurologically injured patient is burdened with additional consequences. Titration of drugs to achieve adequate sedation can mask underlying injury. Agitation can be a result of hypoxia, electrolyte imbalance, temperature dysregulation, hypercarbia, acidosis, infection or hemodynamic shock, all of which result in proliferation of cerebral ischemia (Mirski et al., 1995). The use of sedatives can obscure evidence of a pathophysiologic process that can
lead to secondary injury. Possible physiological contributions to neurological disturbances should always be considered when there is a need to increase sedation.

*Guidelines for Sedation in Traumatic Brain Injured Patients*

The Society of Critical Care Medicine established clinical practice guidelines for the use of sedatives and analgesics based on the premise of optimal sedation with minimal consequence. It is their recommendation that all critically ill patients be assessed regularly for pain and sedation and that all medications be titrated to a pre-approved sedation level (Jacobi et al., 2002). Sedation scales, described below, were developed for this purpose. Evidence based research supporting the effectiveness of a primary sedation scale is scarce and research regarding the application of any scale to TBI patients is essentially nonexistent.

The Brain Trauma Foundation and American Association of Neurological Surgeons (2008) established guidelines regarding sedation and anesthesia of the TBI population stating that while they consider their recommendations optimal they can not be applied to all TBI patients because of the unique physiological requirements of injury. Additionally, because there is limited research of sedation in the neurotrauma patient, the guidelines were based on research of healthy volunteers or elective neurosurgical patients. The following section will discuss the use of sedation assessment scales in the TBI patient and review available literature regarding the use of these scales in evaluating sedation levels.
CHAPTER THREE- LITERATURE REVIEW

Sedation Assessment Scales

Sedation scales are bedside tools used to determine the level of sedation a patient is exhibiting (Delvin et al., 1999). They allow for medication titration to achieve a preset sedation goal. Sedation scales were formulated to improve sedation related complications. The Society of Critical Care Medicine found that sedation scales allow for improved communication of nurse and physician as well as decreased ventilatory times, tracheostomies, shortened length of stay, and lower hospital costs. (Delvin et al., 1999) There are two categories of sedation scales subjective and physiologic.

Subjective scales rely on the examiner to interpret movement, facial expressions, posture and vital signs of non-communicative patients to establish a determined agitation level (Delvin et al., 1999). Agitation is characterized by non-purposeful mental and physical activity that stems from internal anxiety; usually expressed by excessive restlessness and constant movement (Delvin et al., 1999). Based on the examiners analysis medications are titrated to achieve a predetermined sedation level to alleviate the agitated behaviors and internal anxiety.

Physiologic scales rely on external monitors which measure vital signs or neurofunction to display the patient’s level of agitation. Sedation medication is titrated to the predetermined level based on vital signs or electroencephalogram information (Diawai, Thoyre, & Auyong, 2007).

There are currently 25 sedation scales available today, many with similar characteristics and few deemed to have good reliability and validity (Rassin et al. 2007). Research has been scarce to validate these sedation scales, especially their use with the neurotrauma population.
The need for a reliable and valid sedation assessment scale is necessary based on the secondary neurological damage that transpires with improper sedative administration.

**Subjective Scales** Subjective scales rely on examiner interpretation of observed patient behavior to determine pain and agitation levels. Subjective scales are numerically based; the clinician assesses the patient’s response to verbal or tactile stimulus and applies the scales numerical, behavioral correlate. The exams are performed at a predetermined frequency, usually once an hour and the results are influenced by the patient’s sedation level at that exact moment in time. The intermittent approach of subjective scales can not account for the multifactorial influences of baseline consciousness, circadian rhythm, pain, and agitation; nor can they account for changes in sedation levels at non-observed time periods (Daiwai & Thoyre, 2007).

The optimal subjective sedation scale should be easy to implement and document, meticulously exemplify sedation levels within a predefined range, adequately guide treatment, and demonstrate reliability and validity (Jacobi et al., 2002). The goal of treatment is to maintain an adequately sedate patient while maintaining the ability to perform neurological assessments. The sedation level should be determined at the initiation of therapy and be continuously re-evaluated. A pre-determined range of sedation levels should be identified with the knowledge that requirements will fluctuate in accordance to the patient’s circadian rhythm and neurological recovery.

While there are many subjective sedation scales available few have been appraised with good reliability and validity, the majority of the scales lack psychometric testing and none have been chosen as a favorable scale (Diawai et al., 2007). The greatest documented benefit of subjective scales found in the literature is that of improved communication among physicians
and nursing (Diawai et al. 2007). The use of the scale allows the physician to set an optimal level of sedation for their patients and provides the nurses with a goal for medication titration to achieve this level (Diawai et al., 2007). The 4 most common subjective scales used in the critical care setting are the Ramsay sedation scale (RSS), the sedation-agitation scale (SAS), the motor activity assessment scale (MASS), and the Richmond agitation-sedation scale (RASS) (Rassin et al., 2007; Ely et al., 2003). The Ramsay scale appears to be chosen in facilities for use based on its familiarity, as it was the original sedation assessment tool; whereas the other 3 scales have been marginally tested and shown to have good inter-rater reliability (Diawai et al., 2007). The discussion of subjective assessment scales for the purpose of this paper will be limited these 4 scales based on their widespread use in medical facilities. These scales are very similar in construction but vary in the descriptions used to assess sedation levels.

The RSS sedation scale was introduced 36 years ago by Michael E. Ramsay as a means to evaluate the effectiveness of a new sedative-anesthetic and continues to be used today despite the lack of evidence regarding the scale’s efficacy in the critical care setting. The RSS consists of six parameters to define levels of consciousness or agitation. A conscious patient can be given a score of 1-3 and unconscious patients a score of 4-6, most examiners apply the number under 1 of 3 states of sedation: under sedated, sedated, and over sedated (Diawai et al., 2007). Table 2 describes the RSS scale.

<table>
<thead>
<tr>
<th></th>
<th>Ramsay Sedation Assessment Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Patient is anxious and agitated or restless, or both</td>
</tr>
<tr>
<td>2</td>
<td>Patient is cooperative, oriented and tranquil</td>
</tr>
<tr>
<td></td>
<td>Patient responds to commands only</td>
</tr>
<tr>
<td>---</td>
<td>----------------------------------</td>
</tr>
<tr>
<td>4</td>
<td>Patient exhibits brisk response to light glabellar tap or loud auditory stimulus</td>
</tr>
<tr>
<td>5</td>
<td>Patient exhibits a sluggish response to light glabellar tap or loud auditory stimulus</td>
</tr>
<tr>
<td>6</td>
<td>Patient exhibits no response</td>
</tr>
</tbody>
</table>


Published data confirming the reliability and validity of the RSS is scarce; in fact the majority of data consists of comparing the RSS with newly created scales to affirm their validity. The difficulty establishing the validity of the RSS stems from categorical broadness which allows for extensive inter-rater variability and makes comparison studies difficult. This developmental flaw may account for the lack of evidence based supportive data (Hansen-Flaschen, Cowen, & Polomano, 1994). A study by Van Dishoeck, Van Der Hooft, Simoons, Van Der Ent, and Scholte Op Reimer (2008) used a systematic written approach for evaluating sedation in a cardiac and thoracic intensive care unit using the RSS. The premise of the study was to assess the reliability of the RSS by using a written step wise approach to sedation assessment. Their findings support the RSS as a reliable scale, weighted K .86-.92, under the caveat that the RSS must be used following the written instructions (Van Dishoeck et al., 2008). The categorical limitations of the RSS scale may limit its use in the neurologically injured population, based on the variability of agitation in relation to consciousness in these patients.

The lack of constructive measurements of the RSS prompted researchers to develop new observational tools, ones that could be definitively tested for reliability and validity in assessing sedation. The SAS expounded on the RSS by increasing the descriptive detail of sedation levels.
The SAS offered a measurable scale that could be tested for reliability and potentially validate the use of sedation scales in the critically ill patient. The SAS consists of a 7 point scale with level 1 being a highly agitated patient and 7 being a deeply sedated patient (Riker, Picard, & Fraser, 1999). The points in between these two extremes portray the variable levels of consciousness and agitation experienced by the patient during sedative administration. The creators of the SAS scale intentionally created the scale to have few numerical correlates in hopes of establishing a scale that would produce minimal variation among assessors, however this limitation creates some convolution of sedation levels (Riker et al., 1999). The SAS fails to differentiate behavior prior to stimulation and after stimulation as the marker for medication titration leaving room for inter-rater variability (Riker et al., 1999). Table 3 provides an example of the SAS scoring system.

**Table 3**  
_Sedation-Agitation Assessment Scale (SAS)_

<table>
<thead>
<tr>
<th>Score</th>
<th>Level of Sedation-Agitation</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>Dangerous agitation</td>
<td>Pulling at endotracheal tube, thrashing, climbing over bed rails</td>
</tr>
<tr>
<td>6</td>
<td>Very agitated</td>
<td>Does not calm, requires restraints, bites endotracheal tube</td>
</tr>
<tr>
<td>5</td>
<td>Agitated</td>
<td>Attempts to sit up but calms to verbal instructions</td>
</tr>
<tr>
<td>4</td>
<td>Calm and cooperative</td>
<td>Obeys commands</td>
</tr>
<tr>
<td>3</td>
<td>Sedated</td>
<td>Difficult to rouse, obeys simple commands</td>
</tr>
<tr>
<td>2</td>
<td>Very Sedated</td>
<td>Rouses to stimuli. Does not obey commands</td>
</tr>
<tr>
<td>1</td>
<td>Unarousable</td>
<td>Minimal or no response to noxious stimuli</td>
</tr>
</tbody>
</table>

Adapted from Riker, R.R., Fraser, G.L., Cox, P.M. (1994) Continuous Infusion of Haloperidol Controls Agitation in Critically Ill Patients. _Critical Care Medicine_. 22(3), 433-440

Despite the convolution of sedation levels research has shown the SAS to be a reliable scale with easy implementation into critical care practice (Riker et al., 1999). The original verification study of the SAS was compared against the RAMSAY and Harris scale (not discussed here); though neither scale had been tested for reliability nor validity they were the
most frequently used scales at the time. The SAS study was the first of its kind to prove a sedation scale as a dependable form of appraising patient response to medication (Riker et al., 1999). The original study demonstrated an inter-rater agreement of .92 for SAS and .88 for RAMSAY once the scales were quantified along a 3 level scale of sedation which allowed for comparability (Riker et al., 1999). Suggestions for future studies were specific patient population reviews, which continue to be lacking in most subjective scale research (Riker et al., 1999).

The SAS has since been incorporated into many institutions practice and had been used as a verification tool for newly formed scales. A recent study comparing 3 sedation scales in a mixed medical ICU found the SAS to be to a reliable scale but lacking in descriptive verbal and physical detail (Rassin et al. 2007). The depictive imperfections of the SAS make it a suboptimal scale for use in the neuro-ICU as these patients can present with a myriad of agitation and consciousness levels.

The Motor Activity Assessment Scale was created from the SAS scale and is similar in architecture. The MAAS scale was created by intensivists in Utah in an effort to provide a more clearly defined categorical scale that would enhance communication about sedation titration among professionals (Delvin et al., 1999). The MAAS uses 7 concisely defined categories with examples of categorical behavior to describe patient responses while under the influence of sedatives. A MASS score of 0 is given to unresponsive patients with each number up to 6 defining a different degree of agitation and behavioral response (Delvin et al., 1999). Table 4 provides the MAAS scores and associated descriptions.
Table 4
Motor Activity Assessment Scale (MASS)

<table>
<thead>
<tr>
<th>Score</th>
<th>Level of sedation</th>
<th>Response to stimulation</th>
<th>Response to command</th>
<th>Examples of type of complex motor activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>Dangerously agitated and uncooperative.</td>
<td>No external stimulus required to elicit movement.</td>
<td>Does not calm down when asked.</td>
<td>Patient pulling at tubes or catheters or thrashing from side to side or striking at staff or trying to climb out of bed.</td>
</tr>
<tr>
<td>5</td>
<td>Agitated.</td>
<td>No external stimulus required to elicit movement.</td>
<td>Does not consistently obey commands.</td>
<td>Patient attempts to sit up and moves limbs out of bed.</td>
</tr>
<tr>
<td>4</td>
<td>Restless but cooperative</td>
<td>No external stimulus required to elicit movement.</td>
<td>Obeys commands.</td>
<td>Patient is picking at sheets, tubes or uncovering self.</td>
</tr>
<tr>
<td>3</td>
<td>Calm and cooperative</td>
<td>No external stimulus required to elicit movement.</td>
<td>Obeys commands.</td>
<td>Patient adjusts sheets or clothes purposefully.</td>
</tr>
<tr>
<td>2</td>
<td>Responsive to touch or name</td>
<td>Opens eyes or raises eyebrows or turns head toward stimulus or moves limbs when touched or name is loudly spoken.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Responsive only to noxious stimuli</td>
<td>Opens eyes or raises eyebrows or turns head toward stimulus or moves limbs in response to noxious stimulus (tracheal suctioning or 5 secs of vigorous orbital, sternal or nail bed pressure)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>Unresponsive</td>
<td>Does not move with noxious stimulus.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The MAAS is more comprehensive in defining agitation than the previously developed scales in that it provides a more detailed observable response to sedation. There have been two psychometric tests performed to support the use of the MAAS scale in the critically ill; both studies used medical-surgical patients on ventilators with the specific exclusion of the neurologically injured patient. Both studies compared the MAAS to physiologic parameters of agitation and other unsubstantiated scales to confirm validity (Diawai et al., 2007; Delvin et al., 1999). The MAAS was found to have good inter-rater reliability for assessing the level of sedation, with a kappa score of .83 (Diawai et al., 2007). The MAAS scale lacks validity based on the absence of corroborating research and fails to truly delineate itself from the SAS to become a favorable candidate for the optimal tool. The use of this scale in the neurological population has never been tested and the applicability to this population is hindered by the failure of the tool to clearly delineate between consciousness and agitation.

The Richmond Agitation-Sedation Scale was developed by a multidisciplinary team consisting of critical care physicians, nurses and pharmacists in an effort to create an optimal sedation assessment tool (Sessler et al., 2002). The RASS has 10 levels of agitation and sedation; positive numbers define a level of agitation and negative numbers a level of sedation. A person is given a single score following a sequential 3 step approach of observation, response to auditory stimulus, and response to physical stimulus (Sessler et al., 2002). The RASS offers a more precise grading of the level of consciousness than the preceding scales. The scale offers a greater variation for describing the mid-moderate sedation levels, this is beneficial to the assessor in that the majority of medication titration is to a goal of mid-moderate sedation. Table 5 provides an example of the RASS scale’s scoring system and associated descriptive assessments.
<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>+4</td>
<td>Combative Overly combative, violent, immediate danger to staff</td>
</tr>
<tr>
<td>+3</td>
<td>Very agitated Pulls or removes tube(s) or catheter(s); aggressive</td>
</tr>
<tr>
<td>+2</td>
<td>Agitated Frequent non-purposeful movement, fights ventilator</td>
</tr>
<tr>
<td>+1</td>
<td>Restless Anxious but movements not aggressive vigorous</td>
</tr>
<tr>
<td>0</td>
<td>Alert and calm</td>
</tr>
<tr>
<td>-1</td>
<td>Drowsy Not fully alert, but has sustained awakening(eye-opening/eye contact) to voice (&gt;10 seconds)</td>
</tr>
<tr>
<td>-2</td>
<td>Light sedation Briefly awakens with eye contact to voice (&lt;10 seconds)</td>
</tr>
<tr>
<td>-3</td>
<td>Moderate sedation Movement or eye opening to voice (but no eye contact)</td>
</tr>
<tr>
<td>-4</td>
<td>Deep sedation No response to voice, but movement or eye opening to physical stimulation</td>
</tr>
<tr>
<td>-5</td>
<td>Unarousable No response to voice or physical stimulation</td>
</tr>
</tbody>
</table>


The RASS has been evaluated in multiple critical care settings, including neuro-ICU, with the majority of testing occurring in the medical surgical arena. A study out of Israel deemed the RASS to be the most valid and reliable sedation scale of all testable scales (Rassin et al., 2007). This is attributed to the scales unique approach to assessing verbal stimulus response separate from physical stimulus response. This delineation enhances the detection of level of consciousness separate from level of agitation (Ely et al., 2003). Delirium assessment is incorporated into the RASS model allowing for the assessor to determine the patient’s level of cognition and is an additional benefit over other sedation scales. The descriptive abilities provided by the multiply levels of mid-moderate sedation results in a much higher inter-rater reliability, kappa .91, then previously developed scales (Sessler et al., 2002). The RASS has been
shown to have excellent inter-rater validity as well as good validity when compared to the Ramsay and SAS scales (Sessler et al., 2002). The RASS demonstrates promise for use in the neurologically injured patient, it has been sparingly tested in this population and found to have good reliability among interpreters. The multileveled descriptions of mid-moderate sedation and incorporation of delirium assessment are favorable for the neurologically injured patient.

**Objective Scales** Subjective scales measure a moment in time at specified intervals, the presence and interaction of the assessor potentially influences the exam altering the actual assessment of patient sedation (Diawai et al., 2007). The simple act of assessing the patient alters the pre-existing level of consciousness becoming an inherit deficit to all subjective scales. Objective scales were created to bypass this imperfection.

Objective scales are based on physiological measures that are performed on a continuous basis and do not require the assessor to interact with the patient. Theses scales utilize either vital signs or neurofunction monitors to evaluate sedation levels (Diawai et al., 2007). Neurofunction monitors are monitors that utilize the electrical activity of the brain via electroencephalogram monitoring to evaluate the level of sedation. Auditory evoked potential monitors (AEP), electroencephalogram (EEG), and signal processed EEG monitors (BIS) are the most commonly used objective scales (Diawai et al., 2007).

Vital signs are a physiological parameter used for assessing the level of consciousness in a sedated patient. Fluctuations in heart rate, blood pressure, respiratory rate, temperature, and oxygen saturation are used to determine alterations in consciousness (Diawai et al., 2007). The patient’s hemodynamic responses are evaluated for changes that can be attributed to wakefulness.
and it is presumed that tachycardia and hypertension are related to an alert or agitated patient (Diawai et al., 2007). The use of vital signs as a physiological indicator of agitation has never been formally established. There are numerous contributing factors that can cause variations among vital signs making use of these parameters for sedation titration suboptimal (Diawai et al., 2007). This should not discount the use of vital signs in sedation as these can be greatly influenced by medication and the titration of sedatives should have these physiological parameters incorporated (Diawai et al.). Vital signs are not sensitive markers of sedation and therefore should not be used as the sole source of medication titration, however they should be embodied as a source of parameters that allow for hemodynamic stability so as to decrease secondary neurological injury (Diawai et al., 2007).

Electroencephalogram monitoring is performed using multiple scalp electrodes that are placed on the skin of the frontal, temporal, parietal, and occipital areas of the cranium (Diawai et al., 2007). The postsynaptic action potentials that are carried from various areas of the brain to the cerebral cortex give rise to an electrical signal that the scalp electrodes obtain and translate into waveforms (Diawai et al., 2007). The degree of synchronicity or asynchronicity of the waveforms supply us with information regarding level of consciousness; when the waveforms are static and random the patient is awake as they move towards unconsciousness they become more cohesive (Diawai et al., 2007).

Though a specific stable pattern of electrical activity correlating to level of consciousness has not been discerned the full spectrum EEG has been deemed a reliable and valid method for evaluating level of sedation (Guerit et al., 2009). The EEG uses 24 separate leads to observe the
electrical activity of a conscious mind and is therefore not limited by specifically located neurological trauma (Guerit et al., 2009). The exam is sensitive to changes in neurological status and has been found to show neurological decline prior to subjective exams in multiple studies involving TBI patients (Deogaonkar et al., 2004). The limitation of use for the EEG in the neurotrauma patients stems from a practicality issue and not a lack of scientific support; EEG machines are cumbersome, expensive and time consuming (Diawai et al., 2007). The financial resources consumed for purchase, application, maintenance, and trained personnel for each individual patient exceeds the benefit of use and is therefore considered an impractical solution to sedation assessment (Diawai et al., 2007).

Auditory evoked potential (AEP) uses scalp electrodes, similar to EEG, to interpret electrical activity that is evoked by an auditory stimulus. The thalamus and reticular activating system are responsible for regulating levels of consciousness, relying on interactions of neurotransmitters between the pons, midbrain, hypothalamus, and thalamus to create an alert state (Diawai et al., 2007). The interplay that occurs in these areas following stimulus create specific wave patterns on EEG and are known as short latency, middle latency, and long latency evoked potentials (EP; Guerit et al, 2009). Short latency EP involves the brainstems auditory center and upper limb sensory nervous systems action potentials which can be seen approximately 20 milliseconds after a presented stimulus (Guerit et al., 2009). Middle latency follows the primary auditory cortex after an auditory stimulus and produces activity approximately 20-70 milliseconds after auditory stimulus induction (Guerit et al., 2009). Short and middle latency EP are sensitive to changes in the sub-cortical action potential velocity; this is the area that is affected by sedatives, metabolic disequilibrium, and hypothermia (Guerit et al.,
2009). Long latency EP are stimulated by auditory, visual, and somatosensory stimulus to produce EEG activity, long latency conduction loss is associated with irreversible brain damage (Guerit et al., 2009).

The AEP relies on middle latency EP response to determine the level of sedation. A stimulus, usually a click via headphones, enters the brain through the vestibulocochlear cranial nerve and travels along the brainstem to the cortex resulting in transcutaneous electrode conversion to waveforms (Guerit et al., 2009). The use of sedatives delays conduction of the action potential along its pathways coinciding with slowing of the EEG tracing, the degree of slowing indicates the level of cerebral cortex activity which reflects the level of wakefulness (Guerit et al., 2009). Because the AEP uses a limited pathway of consciousness via the auditory nerve, only the temporal cortical response is evaluated (Diawai et al., 2007). The use of AEP has similar scientific support as the EEG monitor with a few exceptions; electrode placement, environmental influence, and individual hearing impairment (Bell, Smith, Allen, & Lutman, 2003). The electrodes must be placed precisely to achieve the least amount of interference possible, as the use of the AEP requires filtering of extraneous stimulus such as postauricle muscle twitch which is also stimulated by sound (Bell et al., 2003). Electrode placement can be hindered in neurotrauma patients based on surgical site interference. The level of extraneous noise that occurs in a busy neurological ICU is problematic, in order to obtain a clear picture of vestibulocochlear stimulation from a specific source other sources that can interfere must be eliminated (Bell et al., 2003). To assist with this problem a filter is applied, which if it is set at inappropriate levels can alter the activity reading providing a falsely elevated or depressed level of consciousness (Bell et al., 2003). Hearing impairment alters the stimulus results in that not the
entire stimulus invokes an action potential, which can appear as a slowed response indicating inaccurate levels of sedation (Bell et al., 2003). These concerns combined with expense make the use of AEP in neurotrauma patients suboptimal.

The bispectral index monitor (BIS) was derived from EEG studies using power spectral and bispectral analysis to note fluctuations and end points of consciousness (Olsen, Chioffi, Macy, Meek, & Cook, 2003). The BIS uses 4 sensors that are applied to the skin of the forehead that extent along the temple and above the eye. The BIS uses processed EEG signals acquired from the frontal cerebral cortex, the brainstem relays activity through the thalamus to the cerebral cortex to regulate consciousness, to determine a number that correlates with wakefulness (Diawai et al., 2007). The BIS monitor displays a number of 0-100 based on the processed EEG signal, with 100 being awake, 70-80 consciously sedated, 60-70 deeply sedated and 40-60 anesthetized (Olsen et al., 2003). The numeric goal for sedation titration is 60-70 in the neurologically injured patient (Olson et al., 2003).

The BIS is greatly subjected to artifact from poor signal quality and electromyographic interference, to account for this the BIS uses a smoothing rate. The smoothing rate determines the amount of artifact free EEG that must be obtained to generate a BIS value and can be set for 10, 15 or 30 second intervals (Diawai et al., 2007). In the ICU setting, where sedatives remain relatively constant, a 30 second interval is optimal because it allows for a greater length of time to obtain artifact free EEG signals (Diawai et al., 2007). The defect to this approach is that abrupt changes in consciousness can be missed or have delayed presentation on the monitor which can result in inappropriate sedation titration (Diawai et al., 2007).
Poor signal quality and electromyographic interference decrease the legitimacy of the BIS number. The interference of these contributing factors can cause numerical devaluation or false elevation allowing for an inadequately sedated patient and the associated injuries (Diawai et al., 2007). Research has shown improvement of artifact conduction with the use of neuromuscular blockades, however this would negate the use of the BIS monitor for the purpose of assessing sedation in neurologically injured patients (Diawai et al., 2007). Introduction of the newest version of the BIS monitor, BIS XP, has shown considerable improvement of signal quality. BIS XP exhibits good correlation with subjective scales in neurologically injured patients where the previous versions had minimally correlated (Deogaonkar et al., 2004).

The BIS monitor has been compared to several different subjective scales in effort to establish the validity of the tool in assessing sedation of ICU patients. The RAMSAY scale has been found to have the least favorable supporting literature to confirm a correlation. Given the lack of supportive research to confirm the validity of the RAMSAY scale the information provided by these studies is obsolete (Hernandez-Gancedo, Pena, Pestana, Perez-Chrzanowska, & Criado, 2006). The SAS and RASS, both of which have been shown to have good reliability and validity, have demonstrated variable outcomes that substantiate the use of the BIS monitor in ICU populations (Deogaonkar et al., 2004). One of the greatest faults in the literature is the lack of identifiable constructs; the make and model of the BIS is often not identified and a gold standard subjective scale has yet to be recognized (Deogaonkar et al., 2004). The difficulty applying the information obtained from these studies emanates from the assumed comparative value of the BIS parametric data and subjective scales ordinal data; to assume that parametric data can be equilibrated to ordinal data is problematic (Diawai et al., 2007).
When the BIS is evaluated as an adjunct to current therapeutic regimens of sedation the results are inconclusively promising. Significant reduction in amount of sedation required and a correlating drop in expenditure has been demonstrated, most prominently seen with Propofol (Olson et al., 2007). The combination of the BIS and a subjective scale has exemplified a reduction in under-sedation (Deogaonkar et al., 2004). The occurrence of over-sedation has been variable and is attributable to the known flaws of the BIS monitor; however the findings demonstrated that the subjective scales alone allowed for an over-sedated patient when compared to the BIS numbers (Deogaonkar et al., 2004). The use of the BIS offers complementary information to sedation assessment, it may not be a candidate as an individual tool but combined with subjective scales shows promise for use in the NICU.
CHAPTER FOUR-ADDRESSING THE PRACTICE PROBLEM

Recommendation of Sedation Scales for Future Studies

Science has made staggering advancements in the management of the neurologically injured. The ability to improve patient outcomes through medical imaging, diagnostic imaging, intravascular interventions and surgery has decreased the mortality rate significantly but morbidity remains high. Our understanding of the devastating effects that secondary injury can have on this populations outcomes has incited researchers to find new ways to identify and manage contributing factors. Recognition of the detrimental effects of improper sedation on secondary injury has prompted health care providers to formulate an optimal protocol for sedation assessment.

Many facilities have adopted one of the currently available subjective sedation assessment tools despite that lack of psychometric testing or proven efficacy. Research regarding subjective scales is outdated and lacking. The outright privation of studies in the neurologically injured patient is concerning. The absence of a reference standard for comparative research of these scales is problematic as is the notion that there is no optimal sedation dose. There is no way to claim a subjective scale has criterion validity without a proven reference standard and there is no available data to suggest optimal sedative dose as sedation requirements are individually based (De Jonghe et al. 2000). Many of the subjective studies have been tested for clinometric properties and found to have good inter-rater reliability; however, these results are compared to other unproven subjective scales and lack verification studies. The MASS and RASS have been
found to have the strongest inter-rater reliability and validity testing but lack confirmatory research.

Subjective sedation assessment scales provide a moment in time for the sedated patient. Only a single study performed by Ely et al. (2003) demonstrated responsiveness to sedation titration. This study provided information regarding the cohesiveness of the RASS exam with the administration of sedatives. No other research has focused on this imperative factor. For the subjective scale to be useful in a neuro-intensive care setting they must demonstrate the ability to correlate with responsiveness.

Subjective sedation assessment scales have primarily been tested in the medical surgical arena. The complexities of neurological injury are stated as the reason for exclusion. This should be the reason for inclusion because the neurologically injured patient is at the greatest risk for secondary injury related to sedation assessment. It is ambitious to assume that the current subjective assessment scales can be generalized for use with this population based on these extreme differences in clinical presentation.

Objective assessment scales have demonstrated good scientific integrity but expense and operational requirements inhibit their usefulness. Of the available technologies the BIS is the most promising for use in the neurologically injured patient. In contrast to the subjective scales the BIS monitor has been tested among the TBI population and has shown promising results. The perpetual monitoring of consciousness that the BIS provides creates a more global assessment of a patient’s response to sedation. BIS has been shown to decrease sedation dosage and drug expenditure without creating an increase in the incidence of under-sedation. Technical
difficulties and the inability to use the equipment in patients with bi-lateral frontal lobe injury have made validating this tool difficult. BIS correlates well with the RAMSAY scale and variably with the more valid scales of SAS and RASS; comparison validity of the two unrelated values is questionable.

There is a clear deficiency in the current knowledge base related to sedation assessment. Future studies should focus on validating a scale for use among the neurologically injured patient. The RASS has shown the most promise in other populations for distinguishing contributing factors of agitation and provides the most detail for sedation and agitation. However, studies utilizing the BIS monitor have shown that the use of subjective scales alone results in higher doses of sedative administration which results in propagation of neuronal injury. A combination of subjective and objective approaches would facilitate the practitioner’s ability to better comprehend the complexity of neurologically injured patient’s presentation. A comparative study utilizing the RASS tool and the RASS plus BIS XP monitor in neurologically injured patients would be beneficial in the process of determining an optimal tool for evaluating sedation in this population and addressing the gap in literature. A randomized control trial that variably assigns the RASS or the BIS XP plus RASS to TBI patients requiring mechanical ventilation and sedation would contribute to the current body of knowledge and possibly direct future studies in identifying best practice for sedation assessment.

Impact of Advanced Practice Nursing

The use of advanced practice nurses (APN) in the critical care arenas has demonstrated improved patient outcomes, decreased length of stay, decreased re-admission rates, and decreased economic consumption (Kleinpell & Gawlinski, 2005). Research has demonstrated
that the APN is instrumental in implementing evidence-based practice and research into care, advocating for guideline adherence, and increasing staff compliance (Kleinpell & Gawlinski). The Doctorate of Nursing Practice (DNP) was created to enhance the current abilities of the APN to impact health care. The DNP encompasses a scientific foundation for practice, analytical methods for evidenced-based practice, diagnostics and prevention, technological and informatic advancement, and patient health advocacy for the improvement and transformation of health care (Rossetter, 2009). Advancement in educational preparation place the DNP in a unique position to actualize an optimal sedation assessment tool in the traumatic brain injured patient.

Conclusion

In the United States 1.7 million people suffer a traumatic brain injury annually, of these 100,000 will endure severe injury and 52,000 will die (Faul et al., 2010). Traumatic brain injuries are a contributing factor in 30.5% of injury related deaths with the estimated medical cost of these injuries reaching 60 billion dollars annually in the year 2000 (Faul et al., 2010). The current economic climate and declining financial medical health reserve emphasizes the need to identify cost saving measures associated with this prominent form of injury.

Science and technology are growing exponentially and as a result knowledge and understanding of how the body responds to medical therapies has increased. Health care providers are only recently beginning to appreciate the complex interactions that sedatives have on the injured brain. The severe physiological implications that improper use of sedatives have on this susceptible population are discerning. Alterations in cerebral vasculature and blood flow secondary to inappropriate sedative administration results in neuronal tissue death and
propagation of cerebral injury (Diawai et al., 2007). Optimizing sedation in this population can minimize secondary brain injury and improve long term outcomes. Identifying a gold standard sedation assessment tool would greatly improve current sedation protocols and reduce the incidence of improper sedative administration and sedation related injury in TBI in patients thus potentially decreasing the complications that increase morbidity and mortality.
References


