EFFECTS OF ENVIRONMENTAL FACTORS ON SLEEP PATTERNS
IN TRAUMATIC BRAIN INJURED ADULTS IN THE REHABILITATION SETTING

A dissertation presented to
the Kent State University College of Nursing
In partial fulfillment of the Requirements
For the degree of Doctor of Philosophy

By

Shelly C. Amato

March 23, 2018
A dissertation written by

Shelly Amato

Approved by

_____________________________, Chair, Doctoral Dissertation Committee
Mary K. Anthony

_____________________________, Member, Doctoral Dissertation Committee
Yea-Jyh Chen

_____________________________, Member, Doctoral Dissertation Committee
Sheau-Huey Chiu

_____________________________, Member, Doctoral Dissertation Committee
Jeffrey Ciesla

_____________________________, Member, Doctoral Dissertation Committee
Mary Beth Spitznagel

Accepted by

_____________________________, Director, Joint Ph.D. in Nursing Program
Patricia Vermeersch

_____________________________, Graduate Dean, College of Nursing
Wendy Umberger
Acknowledgements

As I reach the end of the journey of earning a PhD, I would like to acknowledge all the people in my life that have helped me to achieve this personal and professional goal. Without the support of each these special people, I would not have been able to earn my PhD.

I would like to thank Dr. Mary Anthony for her guidance and expertise throughout the process. Dr. Anthony set the bar high and pushed me to be the best nurse scientist that I could be. I appreciate her time, attention to detail, and support. I would also like to thank my dissertation committee members, Dr. Chen, Dr. Ciesla, and Dr. Chiu. Each of you provided me with expertise that was helpful and appreciated. I would also like to thank Dr. Spitznagel who moderated the dissertation defense. It was an exciting experience presenting my research findings.

Next, I would like to thank my parents (Claire and Bob), sister (Cheryl) and niece (Christine) who have always supported me, and valued education. Without their belief in me, I may not have even considered pursuing my PhD. Their ongoing support and kind words always helped lift me up when I was feeling down—thank you! I would also like to thank my two wonderful, smart, precious, and hardworking daughters, Alexandria and Adriana. You always said the sweetest things to me: “you are smart mom”, “you will be great mom”, and “you were awesome mom”. I can’t even express how much these words meant to me. There have been countless days and nights that all three of us worked on papers and assignments together. I hope that I have been a positive role model for both of you and demonstrated the importance of never giving up, no matter how difficult the road may be. I love you both!

My friends and co-workers have also provided me with words that have lifted my spirits during the good times and tough times. There are so many to thank, including my BUNCO
girlfriends, and my MetroHealth colleagues and friends. Patty, Kathy, Nilda, Terrie, and Iris, thank you for listening to me and giving me a pep talk when I needed it. A special thanks to my dear friends Cheryl Bradas and Wendy Sarver who also walk in my shoes through the process of earning a PhD; I will always share a special bond with you two ladies. Thank you both for being at my dissertation defense to support me and a special thanks to Cheryl Bradas who helped me re-number my table of contents numerous times, edit my dissertation, fix APA format, as well as help Alexandria get me to dissertation defense on time. I will never forget all you have done for me. Also, I would like to thank Molly McNett who inspires me to continue to advance the science of nursing in the clinical setting. Your positive spirit is so appreciated.

Finally, I would like to thank all of the staff at MetroHealth Medical Center who supported me throughout my research. Thank you to Judy Bodrock and Patty Fuller for giving me administrative approval to complete the study on the Brain Injury Unit. Thank you to my wonderful leader, Sandy Esber for always understanding when I needed to take a day off to work on the dissertation and has encouraged me throughout the process. A special thanks to all the staff, especially the night shift staff, who were willing to help with data collection; I would not have been able to complete this important study without your help. Thank you again.
Table of Contents

Chapter 1: Introduction .......................................................................................................................... 1
  Normal Sleep ................................................................................................................................. 3
  Sleep in Traumatic Brain Injury ................................................................................................. 5
  Sleep Measurement .................................................................................................................... 7
  Sleep and Recovery ..................................................................................................................... 8
  Stressful Environments and Health and Recovery ......................................................................... 9
    Intrinsic Factors Affecting Sleep ............................................................................................. 10
    Extrinsic Factors Affecting Sleep ......................................................................................... 10
  Stress Process Model ................................................................................................................ 14
  Application of Stress Process Model to Study ........................................................................... 15
  Summary .................................................................................................................................... 19
  Conceptual Definitions ............................................................................................................. 20
  Research Questions and Hypotheses ......................................................................................... 21

Chapter 2: Literature Review ........................................................................................................ 23
  Prevalence of Sleep Disturbances .............................................................................................. 23
  Objective Measurement of Sleep Architecture: Polysomnography .............................................. 24
  Objective Measurement of Sleep Patterns: Actigraphy ............................................................. 26
  Subjective Measurement of Sleep ............................................................................................. 30
  Predisposing Factors Contributing to Sleep Disturbance in Persons with TBI ......................... 32
    Neurotransmitters .................................................................................................................. 33
    Hormones ............................................................................................................................... 33
    Psychological and Physical Factors ..................................................................................... 34
    Environmental Factors Associated with Changes in Sleep .................................................... 35
  Sleep Outcomes ........................................................................................................................ 43
    Cognitive Outcomes .............................................................................................................. 43
    Functional Performance ......................................................................................................... 45
    Mood Outcomes ..................................................................................................................... 46
  Summary of Sleep in TBI .......................................................................................................... 47

Chapter 3: Methodology .................................................................................................................. 50
  Institutional Review Board Approval ......................................................................................... 50
  Design, Setting and Sample ....................................................................................................... 50
    Setting ..................................................................................................................................... 50
    Sample ..................................................................................................................................... 50
  Inclusion and Exclusion Criteria .............................................................................................. 51
  Sample Size .............................................................................................................................. 51
Chapter 4: Results

Subject Characteristics ................................................................. 75
  Demographic Characteristics..................................................... 75
  Clinical Characteristics............................................................. 75
Preliminary Analysis ................................................................. 78
  Day One and Day Two Data....................................................... 78
Sleep ............................................................................................ 79
  Quantity of Sleep........................................................................ 79
  Quality of Sleep ........................................................................ 80
  Subjective Sleep: Descriptive Statistics .................................... 81
Patient Care Activities: Descriptive Statistics ............................... 82
Light ............................................................................................ 84
Major Study Research Questions and Hypotheses ............................ 85
  Hypothesis H4A ......................................................................... 85
  Hypothesis H4B ......................................................................... 85
  Hypothesis H4C ......................................................................... 85
  Hypothesis H4D ......................................................................... 85
  Hypothesis H4E ......................................................................... 85
  Hypothesis H4F ......................................................................... 85
Appendices

A. MetroHealth IRB Approval Letter .................................................................156
B. Capacity to Consent Checklist ....................................................................160
C. Bland Altman Scatterplot for Total Light Exposure ....................................161
D. Bland Altman Scatterplot for Average Light Exposure ..............................162
E. Bland Altman Scatterplot for Maximum Light Exposure .............................163
F. Patient Care Environmental Stressor Log .....................................................164
G. Training for Completing Patient Care Environmental Stressor Log (PCESL) ..............................................................................................................166
H. Verran and Snyder-Halpern Sleep Scale ......................................................169
I. Data Collection Tool ......................................................................................171
J. Recruitment Flier ..............................................................................................175
K. Number of Injuries Sustained and Glasgow Coma Scale Score ..................176
L. Hierarchical Regression Analysis for Variables Predicting Total Sleep Time Day 1 ..........................................................................................177
M. Hierarchical Regression Analysis for Variables Predicting Total Sleep Time Day 2 ..........................................................178
List of Tables

1. Subjects Screened as Ineligible ..........................................................................................52
2. Sleep Variable Definitions ................................................................................................56
3. Comparisons of Actiwatch on Subject and Headboards .....................................................63
4. Within Subject Analysis for Sleep and Light Characteristics ............................................70
5. Correlations of Covariates and Independent Variables ....................................................71
6. Demographic Characteristics .............................................................................................76
7. Clinical Characteristics ........................................................................................................77
8. Twenty-Four Hour and Daytime Sleep Data ......................................................................79
9. Quantity of Nighttime Sleep ..............................................................................................80
10. Quality of Nighttime Sleep ...............................................................................................81
11. Verran and Snyder-Halpern Sleep Scale Scores (VSH) ....................................................82
12. Patient Care Interruption Data ..........................................................................................83
13. Light Levels .......................................................................................................................84
14. Correlation of Main Study Variables ................................................................................87
15. Hierarchical Regression Analysis for Variables Explaining Total Sleep .........................89
16. Hierarchical Regression Analysis for Variables Explaining Sleep Duration ...................91
17. Hierarchical Regression Analysis for Variables Explaining Wake After Sleep Onset ......93
18. Hierarchical Regression Analysis for Variables Explaining Sleep Efficiency .................95
19. Mann Whitney U Test Comparing Subjects With and Without Constant Supervision .....97
20. Trimmed Model: Regression Analysis for Variables Explaining Total Sleep Time ........100
21. Trimmed Model: Regression Analysis for Variables Explaining Sleep Duration ...........101
22. Trimmed Model: Regression Analysis for Variables Explaining Sleep Efficiency ..........102
23. Trimmed Model: Regression Analysis for Variables Explaining WASO ......................103
24. Regression Analysis for Variables Explaining Sleep: Trimmed and Full Models ..........104
25. Mann-Whitney U Test Comparing Subjects With and Without Roommate ..................105

Figure

1. Study Model .......................................................................................................................17
Abstract

Each year in the United States an estimated 1.7 million people sustain traumatic brain injuries, resulting in impairments in function, cognition, emotion, and behavior. In addition, disrupted sleep patterns are common after traumatic brain injury with prevalence rates between 30% and 84%. A variety of factors contributes to changes in sleep patterns including the underlying illness, neuronal changes that occur with brain injury, and psychological factors. In addition, environmental factors related to the hospital setting can be a stressor for individuals recovering from illness or injury. Altered sleep patterns have been shown to affect cognition, mood, and reported fatigue, which can negatively affect the recovery process. The purposes of this study were to: (a) describe sleep patterns of adults with traumatic brain injury in the acute rehabilitation setting; and (b) examine the effects of environmental stressors (light and patient care activities) on patterns of sleep. The stress process model was used to guide this study.

The descriptive, correlational, explanatory study was conducted on an acute traumatic brain injury rehabilitation unit located in a 67-bed inpatient rehabilitation hospital. A non-probability convenience sample of 64 traumatic brain injured subjects 18 years of age or older was recruited. Subjects wore an actiwatch for 48 hours to collect light and sleep data. Nursing staff documented all patient care activities performed during nighttime hours.

Hierarchical multiple regression analysis was used to determine the effects of light exposure and patient care activities on quantity and quality of sleep. Results indicated that total sleep time was not explained by either direct patient care activities or light. Nighttime sleep duration and sleep efficiency was explained by patient care activities, while the entire model explained wake time after sleep onset.

Findings from this study provide information about the effects of environmental stressors on sleep outcomes in persons with traumatic brain injury in the rehabilitation setting. This
information will serve as the basis for examining the necessity and timing of nursing care activities and associated light exposure during nighttime hours. Findings will be useful when evaluating standard care practices and provide a basis for making policy changes that optimize sleep in the hospital setting.
Chapter 1. Overview

Introduction

Each year in the United States an estimated 1.7 million people sustain traumatic brain injuries (TBI) from motor vehicle crashes, gunshot wounds, falls, and sports-related head trauma. Of those, more than 282,000 require inpatient hospitalization for injuries (Centers for Disease Control and Prevention [CDC], 2017), many requiring rehabilitation (Murphy & Carmine, 2012). Depending on the severity of injury, TBI can result in impairments in function, cognition, emotion, and behavior (Levine & Flanagan, 2010). Research suggests that in addition to these impairments, disrupted sleep patterns are a common problem after TBI (Baumann, 2016; Sandsmark, Elliot, & Lim, 2017). Prevalence rates of sleep disturbance for persons with TBI range between 30% and 84% and are higher than those in the general population (Mathias & Alvaro, 2012).

Sleep is a restorative process that is necessary not only for survival but for recovery. Sleep allows the body time to replenish energy stores in the brain that have been depleted during wakefulness (Scharf, Naidoo, & Zimmerman, 2008) and plays a vital role in a person’s physical, mental, and emotional health as well as quality of life. After trauma, sleep is needed for cell growth and repair, maintaining a healthy immune system and neuronal rest and repair (National Institute of Neurological Disorders and Stroke [NINDS], 2014). The importance of sleep is even more important for persons with TBI in the acute rehabilitation setting since there are intense cognitive and physical demands placed on patients in order to optimize recovery efforts.

A variety of factors contribute to changes in sleep patterns in persons with TBI including the underlying illness, neuronal changes that occur with brain injury, and psychological factors. Trauma to the brain predisposes persons with TBI to sleep disturbance due to damage to areas of
the brain responsible for regulation of sleep. These physiologic changes cannot be controlled; consequently, the importance of managing environmental factors that can disturb sleep is a priority. Nursing and medical care provided to patients in the hospital setting can serve as stressors for individuals recovering from illness or injury (Friese, 2008). Studies in ICU and acute care settings have found that patient care activities and light during nighttime hours can cause sleep disturbance (Le et al., 2012; Bano et al., 2014). Patient care activities can disturb sleep while exposure to light during times of sleep or lack of exposure to light dark contrast can disrupt the circadian rhythms (Bano et al., 2014; Cho, Joo, Koo, & Hong, 2013). These environmental factors conflict with promoting adequate sleep and recovery efforts in this vulnerable population recovering from TBI. Although studies suggest that environmental stressors affect sleep in the acute care settings, little is known about how patient care activities and light affect sleep in the rehabilitation setting.

As persons recover from TBI it is essential to promote effective sleep patterns to maximize physical and cognitive improvements since the most significant gains in recovery occur during acute inpatient rehabilitation (Agrawal & Joshi, 2014). The functional, cognitive, and emotional deficits associated with brain injury and increased need for healthcare services highlight the importance of addressing factors that can affect sleep patterns while in the rehabilitation setting. Additionally, studies have found that sleep disturbance is common initially after the traumatic brain injury occurs and persists in the community setting. What is not known is whether disturbances in sleep patterns precipitated by environmental stressors in the hospital setting, perpetuates sleep disturbances after discharge.
The purposes of this study were to: (a) describe the sleep patterns of adults with traumatic brain injury in the acute rehabilitation setting; and (b) examine the effects of environmental stressors including exposure to light and patient care activities on patterns of sleep.

**Normal Sleep**

Sleep is defined as a temporary and reversible behavioral state of unresponsiveness to the environment as well as perceptual disengagement (Carskadon & Dement, 2011). The quantity of sleep needed by individuals depends on a variety of factors including genetics, age, lifestyle, and health status. The National Sleep Foundation (NSF) recommends seven to nine hours of sleep daily for adults 18 to 64 years and seven to eight hours daily for 65 years and older (Hirshkowitz et al., 2015). Additionally, the NSF has identified good sleep quality as sleep efficiency (SE) 85% or greater, wake after sleep onset (WASO) 20 minutes or less, and one or less awakenings lasting greater than five minutes across all age groups (Ohayon et al., 2017).

Sleep involves a variety of factors including genetic, immunological, endocrinological, neurochemical, psychological, and electrophysiological aspects (Dresler et al., 2014). Two separate states of sleep have been described based on physiologic parameters: non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep.

Non-rapid eye movement (NREM) sleep includes sleep Stages 1 through 4 and is characterized by a gradual decrease in temperature, respiratory rate, heart rate, blood pressure, and muscle activity as well as response to the external environment. During NREM Stage 3 and 4, referred to as slow wave sleep (SWS), information processing and memory consolidation occurs. Memory consolidation is a neurological process that is enhanced by sleep and involves converting short-term memory to long-term memory (Peigneux & Smith, 2011).
Following the NREM stages of sleep, REM sleep is activated by secretion of acetylcholine and inhibited by serotonin. During REM sleep, skeletal muscles become completely paralyzed with the exception of eye movements, heart, and diaphragm muscles. Brain activity, in contrast to NREM sleep, is extremely active similar to brain activity during wakefulness. Core body temperature is not well regulated during REM sleep and breathing becomes irregular and rapid while heart rate and blood pressure increase to near waking levels. Memory consolidation and new learning take place during REM sleep (Carskadon & Dement, 2011).

During each episode of sleep, a person cycles through both NREM and REM sleep with approximately 5 to 6 cycles each night. Sleep begins with entering into Stage 1 NREM sleep and gradually progresses through Stage 2, Stage 3 and Stage 4 NREM sleep. The first episode of REM sleep occurs approximately 90 minutes after initiation of sleep. An average sleep cycle lasts approximately 90-110 minutes with the first cycle lasting the shortest amount of time.

The first cycle of sleep starts with Stage 1 NREM sleep. Stage 1 is the period when sleep begins and is the time between wakefulness and the start of sleep. Stage 1 sleep lasts for only a few minutes during the first cycle and is characterized by a low arousal threshold that allows a person to be awakened easily. It is the transition stage between wake and sleep and accounts for 2% to 5% of total sleep time (Carskadon & Dement, 2011). Stage 2 is considered onset of sleep when the body disengages from the surrounding environment. During Stage 2 NREM sleep the arousal threshold rises resulting in the need for a stronger stimulus for arousal. Stage 2 sleep lasts 10 to 25 minutes during each cycle and constitutes 45% to 55% of total sleep time (Carskadon & Dement, 2011). Non-REM Stage 3 and 4 (Rechtschaffen & Kales, 1968) are considered deep sleep or SWS and the time when muscles become relaxed, energy is restored, growth, and repair
of tissue occurs and is considered restorative sleep. Stage 3 sleep lasts only a few minutes during the cycle and is followed by 20 to 40 minutes of Stage 4 sleep. Stage 3 NREM sleep accounts for 3% to 8% of sleep time while Stage 4 NREM sleep accounts for 10% to 15% of sleep time (Carskadon & Dement, 2011). During NREM Stage 4 sleep, the sleep arousal threshold is the highest for NREM, requiring a larger stimulus for arousal as compared to Stage 1 and 2 NREM sleep.

Following movement from Stage 1 through 4 of NREM sleep is an ascent back through the lighter stages of sleep; initially several minutes of Stage 3 then Stage 2 and finally movement into the first episode of rapid eye movement (REM) sleep. During this first cycle, REM sleep lasts for 1 to 5 minutes. REM sleep accounts for 20% to 25% of total sleep time with four to six episodes occurring during a typical night of sleep. Non-REM and REM sleep continue to cycle throughout the night with time spent in REM sleep increasing while Stage 3 and 4 (SWS) decrease and may disappear with Stage 2 occupying a majority of NREM sleep as the night progresses (Carskadon & Dement, 2011). During REM sleep energy to the brain and body is restored and consolidation of procedural memory occurs (NINDS, 2014). Uninterrupted sleep allows the body to cycle though the stages of NREM and REM sleep allowing restoration and memory consolidation thus presents an opportunity to optimize the recovery process for persons with TBI.

**Sleep in Traumatic Brain Injury**

Traumatic brain injury is a non-congenital, non-degenerative insult to the brain caused by an external force that leads to a temporary or permanent impairment in psychosocial, physical, or cognitive function or other associated brain pathology (Baumann, 2012; Brain Injury Association of America [BIAA], 2011). In addition to these deficits, sleep is a common problem after TBI
both in short (Baumann, Werth, Stocker, Ludwig, & Bassetti, 2007; Ouellet, Beaulieu-Bonneau, & Morin, 2015) and long-term recovery (Kempf, Werth, Kaiser, Bassetti, & Baumann, 2010). Areas in the brain responsible for sleep wake regulation can be impacted by either direct or indirect trauma to the brain. Pathophysiologic mechanisms involving damage to neurotransmitters or the hypothalamus can lead to sleep disturbances (Mollayeva, Colantonio, Mollayeva, & Shapiro, 2013).

Initially after the TBI, time spent in an intensive care unit (ICU) can result in alterations in sleep patterns due to environmental stressors such as frequent awakenings for patient care activities, abnormal fluctuations in lighting, unfamiliar and unwanted noise, and changes in sleep schedules (Bernhofer, Higgins, Daly, Burant, & Hornick, 2014; Le et al., 2012; Tamburri, DiBrenza, Zozula, & Redeker, 2004). Physiological effects of acute sleep pattern changes, specifically effects of sleep deprivation, result in complaints of subjective sleepiness and have been found to affect alertness, mood, and performance. These symptoms are often mild and quickly reversible with recovery sleep (Bonnet, 2011), but often persons with TBI require extended time in the hospital setting for recovery thus perpetuating altered sleep patterns. After extended time in the hospital setting, prolonged changes in sleep patterns can result in additional negative effects associated with sleep loss resulting in impulsivity, mood swings, depression, and fatigue (Duclos, Beauregard, Bottari, Ouellet, & Gosselin, 2015). Minimizing environmental stressors affecting sleep can positively influence effects of rehabilitation efforts.

Chronic alterations quantity and quality of sleep during acute and rehabilitation hospitalizations pose a potential risk for poor outcomes in persons recovering from TBI. When patients advance to inpatient rehabilitation, therapy encompasses a large part of the recovery process thus, the importance of optimizing this time period is essential to achieving the highest
level of function. While studies have shown that physiological stressors related to the brain injury can affect sleep (Baumann et al., 2007; Mollayeva et al., 2013), environmental stressors inherent to the rehabilitation setting are unexplored.

Sleep Measurement

Prevalence rates of sleep disturbance among persons with TBI have been reported using a variety of measurement approaches. Some studies base prevalence rates on a formal sleep disorder diagnosis such as insomnia, obstructive sleep apnea, or hypersomnia (Gardani, Morfiri, Thomson, O’Neill, & McMillan, 2015) while others use more broad definitions that include self-reported sleep complaints (Rao, McCann, Han, Bergey, & Smith, 2014). Other studies have used either subjective or objective measures of sleep disturbance or a combination of both to report prevalence rates. Subjective measures used to assess sleep include self-reported surveys such as the Pittsburg Sleep Quality Index (PSQI) (Mani et al., 2015), Epworth Sleepiness Scale (ESS), or sleep diaries (Fogelberg et al., 2012; Ponsford, Parcell, Sinclair, Roper, & Rajaratnam, 2013; Shekleton et al., 2010). Others have used objective measures including polysomnography, actigraphy, multiple sleep latency tests, or melatonin levels (Makley et al., 2009; Mathias & Alvaro, 2012; Sommerauer, Valko, Werth, & Baumann, 2013).

Although polysomnography is considered the gold standard for measuring sleep conditions such as obstructive sleep apnea (Chervin, 2011), the use of actigraphy is increasingly being used to measure sleep wake cycles and sleep quality (Harvey & Spielman, 2011). Numerous researchers have used actigraphy due to its ease in monitoring sleep-wake cycles outside of a sleep lab setting. Actigraphy has been widely used in community dwelling persons with TBI, although only two studies have used actigraphy in the inpatient rehabilitation setting (Makley et al., 2009; Towns et al., 2016). Regardless of the measurement approach used,
prevalence rates of sleep disturbances are consistent and compelling within TBI literature suggesting the quantity and quality of sleep is affected. This study integrates both objective and subjective sleep data to provide a better understanding of sleep patterns in TBI during the rehabilitation phase of recovery.

**Sleep and Recovery**

Sleep patterns include characteristics such as the quantity and quality of sleep. Quantity of sleep encompasses sleep that occurs during nighttime hours as well as daytime sleep. Quality of sleep includes wake after sleep onset (WASO), often referred to as sleep fragmentation, and sleep efficiency (SE). Deficits in either the quantity or quality of sleep can impact a person’s health and recovery process.

The National Institute of Neurological Disorders and Stroke (NINDS, 2014) reports that recovery time from TBI is variable, although studies suggest that the most gains in improvement occur during the first 6 months after the injury, with the most significant gains occurring during the acute inpatient rehabilitation phase of recovery (Agrawal & Joshi, 2014). Given that time spent in the inpatient rehabilitation setting is limited, ranging from 2 to 6 weeks depending on the severity of injury, the importance of optimizing sleep by managing hospital environmental stressors is essential to the recovery process.

During the recovery period from TBI, particularly during inpatient rehabilitation, the goal is to help persons reintegrate into social, community, and vocational domains by improving both cognitive and functional outcomes (Chua, Ng, Yap, & Bok, 2007). Disruptions in sleep patterns among TBI patients can lead to fatigue during daytime hours or daytime napping resulting in further disruptions in nighttime sleep (Ponsford & Sinclair, 2014). These changes in sleep patterns may impact the course of recovery and have been found to be associated with longer
hospital length of stays (Makley et al., 2008), duration of post-traumatic amnesia (Makley et al., 2009), altered cognition (Sherer, Yablon, & Nakase-Richardson, 2009), changes in mood (Rao et al., 2014; Chaput, Giguere, Chauny, Denis, & Lavigne, 2009), and functioning (Gardani, Morfiri, Thomson, O’Neill, & McMillan, 2015). Given the impact of disturbed sleep on cognitive, mood, behavioral, and functional outcomes and the limited time spent in the inpatient rehabilitation setting, it is necessary to maximize time spent during inpatient rehabilitation to promote recovery.

**Stressful Environments and Health and Recovery**

Researchers and health care professionals have examined the role of the environment on health and recovery for many years (Gabor et al., 2003; Tamburri et al., 2004). Studies suggest that environmental conditions play a role in the stress of an individual, and physical environments have characteristics that can influence whether or not stress is produced (Delvin & Arneill, 2003). Environmental stress occurs when there is an imbalance between the demands of the environment and response capabilities of an individual (Lazarus & Cohen, 1977) and originates from both the reactions to and evaluation of the environment. A person’s adaptive resources used to manage stress produced by environmental conditions depend on the type and degree of disturbance to the individual (Evans, 1982).

Environmental stressors that impact health in the hospital setting are diverse and can potentially affect health and recovery. Common environmental factors within hospitals that can serve as environmental stressors include patient care activities, light, and noise and have been shown to negatively impact sleep and health (Yoder, Staisiunas, Meltzer, Knutson, & Arora, 2012). Since changes in sleep patterns may negatively impact recovery, it is important for nurses
to manage environmental stressors in caring for patients with TBI in the rehabilitation phase of recovery.

**Intrinsic Factors Affecting Sleep**

Studies have shown that factors related to changes in sleep patterns are multifactorial and include both intrinsic and extrinsic factors. Intrinsic factors such as physiological and hormonal changes after the injury can alter sleep wake cycles while age, prior sleep history, and sleep disorders can negatively affect sleep by impacting the amount and timing of NREM and REM sleep (Carskadon & Dement, 2011). Additionally, a person’s underlying illness or psychological stressors may impact sleep. More specifically, studies have found that pain (Ouellet, Beaulieu-Bonneau, & Morin, 2006; Suzuki et al., 2017), depression, anxiety (Parcell, Ponsford, Redman & Rajaratnam, 2008), and fatigue (Ouellet et al., 2006) contribute to changes in quantity and quality of sleep.

**Extrinsic Factors Affecting Sleep**

In contrast to intrinsic factors that cannot be modified, extrinsic factors that serve as stressors in the hospital environment (patient care activities and exposure to light) can be modified. Several studies conducted in intensive care unit (ICU) settings have found that patient care activities (Ding, Redeker, Pisani, Yaggi, & Knauert, 2017; Gabor et al., 2003) and exposure to light during nighttime hours impact sleep patterns (Guardiola-Lemaitre & Quera-Salva, 2011). Since these extrinsic factors can be managed by altering standard care practices as well as the timing of patient care activities, there is potential to positively impact outcomes for patients with TBI in the rehabilitation setting.

**Extrinsic factor: Patient care activities.** Although causes of disruptions in quantity and quality of sleep are multifactorial, limited research has focused on how patient care activities
serve as stressors in the hospital environment, specifically in the inpatient rehabilitation setting (Celik, Oztekin, Akyolcu, & Issever, 2005; Ugras, Babayigit, Tosun, Aksoy, & Turan, 2015).

Patient care activities are a necessary part of hospitalization performed by healthcare providers to assist in the recovery process although hospital routines are frequently not consistent with routines that occur in a home setting. Patient care encompasses a wide range of activities that can be direct or indirect activities, both of which have the potential to negatively impact sleep when implemented during nighttime hours.

In addition to disturbances caused by direct and indirect activities, the timing of these activities can serve as an additional environmental stressor. In the hospital setting, nursing care is frequently performed during times when persons with TBI would normally be sleeping with potential interruptions occurring during phases of sleep that are important to recovery (Tamburri et al., 2004). Celik et al. (2005) reported that in a sample of 60 patients in the ICU, mean patient care interactions were 51 per patient per night. Similarly, Ugras et al. (2015) reported a mean of 42 patient care interactions for patients in the neurosurgical ICU. Although these interactions are performed to minimize the likelihood of developing physical complications associated with the recovery process, they are often conducted according to standardized hospital routines that lead to fragmented sleep patterns, and decreased SE. In a study by Redeker et al. (2000), 48% of post-operative cardiac subjects reported the most disturbing environmental factor that impacted their sleep were interventions performed by health care providers. Conducting patient care activities can have unintended consequences negatively affecting the quantity and quality of sleep.

During a typical sleep cycle, a person transitions from wake to Stages 1 through 4 NREM then back to Stage 2 NREM before transitioning to REM sleep. Frequent arousals can cause changes in sleep architecture thus impacting the duration and timing of a sleep cycle. Periodic
brief arousals have been found to reduce restoration that occurs during sleep and results in deficits similar to total sleep deprivation (Bonnet, 2011). Since Stage 3 and 4 NREM and REM sleep are responsible for brain plasticity, consolidation of memory, new learning, and restoration (Peigneux & Smith, 2011), the importance of supporting uninterrupted periods of sleep to promote improved outcomes of recovery cannot be underestimated. Although studies in medical surgical and ICU settings have reported that patient care activities are frequent and disrupt sleep patterns (Celik et al., 2005; Gabor et al., 2003; Le et al., 2012; Tamburri et al., 2004), there is a gap in the literature related to the impact that patient care activities have on sleep patterns of patients with TBI in the rehabilitation setting.

**Extrinsic factor: Light.** Visible light is the spectrum of electromagnetic waves that can be seen by the human eye (Miller & Burns, 2014). White light is measured in lux and is the combined mixture of all visible wavelengths and can be generated by sources such as the sun and stars as well as artificial sources of light. Sources of light in the hospital setting include overhead lights, monitors, IV pumps, and nightlights used to promote a safe environment. Recommended light levels for bedroom lighting range from 200 to 300 lux (DiLaura, Houser, Mistrick, & Steffy, 2011), although studies have found lighting in hospital rooms to be low (Bernhofer et al., 2014, Bano et al., 2014).

Light serves as an environmental signal that synchronizes the circadian rhythms (Mistiberger & Rusak, 2011). Although natural light and dark cycles serve as the strongest environmental signal, light signals in the hospital setting can also affect sleep-wake patterns (Reid, Chang, & Zee, 2004). When patients are exposed to light during times of sleep or experience a lack of exposure to natural fluctuations in light-dark patterns, light becomes an environmental stressor.
The effects of light on sleep can have both positive and negative outcomes. Bright light therapy has been used as a non-pharmacological mechanism to improve quality of sleep (Akyar & Akdemir, 2013) and reduce fatigue. In a study by Kobayashi et al. (2001) investigating bright light exposure (8000 lux) in the treatment of sleep disturbances in a hospitalized geriatric population, results suggested that improvements occurred with drowsiness and difficulty falling asleep in 8 of 10 subjects. A similar study using high-intensity blue light therapy for treatment of fatigue in persons with TBI resulted in a reduction in daytime sleepiness and fatigue during the treatment phase with a trend toward baseline after completion of treatment (Sinclair et al., 2014b).

While light has been used as a therapy to improve symptoms of fatigue and sleep disturbance, unwanted or unnatural fluctuations in light during time of sleep can result in awakenings and reported poor quality of sleep (Freedman, Kotzer, & Schwab, 1999). Fluctuations can result from lighting during nighttime hours when patient care is being performed, or decreased light levels during the daytime in attempts to provide a restful environment.

In normal sleep patterns, melatonin levels rise in the evening when external light levels are low causing feelings of sleepiness and drop in the early morning hours when light levels begin to rise (Guardiola-Lemaitre & Quera-Salva, 2011). Too little light exposure during daytime hours stimulates the release of the hormone melatonin that results in sleepiness or fatigue during daytime hours; little day-night light contrast can result in alterations in circadian rhythms (Bano et al., 2014). Light dark contrast is needed to maintain circadian rhythms (Rea, Bierman, Figueiro, & Bullough, 2008), which may be influenced by the hospital environment. A study by Bernhofer et al. (2014) investigating hospital lighting and its association with sleep,
mood, and pain in a sample of 40 patients on a medical unit, found light exposure levels to be low and little sleep wake synchronization with light. Similarly, Bano et al. (2014) reported that in a study investigating the relationship between environmental factors and sleep quality, luminance levels were low and hospitalized medical patients who slept near a window exposed to higher levels of morning light reported sleeping better than those who slept far from the window. The amount and patterns related to when lights are on and off can be a source of environmental stress in the hospital setting leading to poor quality or quantity of sleep. This study investigated how exposure to light during nighttime hours affects sleep patterns in the inpatient rehabilitation setting.

**Stress Process Model**

Pearlin’s stress process model provides a framework for understanding how stressors in the hospital environment affect sleep outcomes in the hospital setting for persons recovering from TBI. The stress process model was first developed in 1989 by Leonard Pearlin and identifies the domains of the stress process as stressors, mediators of stress, and outcomes resulting from stress. Pearlin (1989) categorizes stressors as life events or chronic strains and views stressors as circumstances that give rise to stress. Life events are considered changes that occur in an individual’s life but not necessarily stressful unless the change is thought to be undesirable, unscheduled, or uncontrolled. In contrast to life events, chronic life strains are considered potentially stressful enduring threats or problems that occur in a person’s daily life. Neither life events nor chronic strains stand alone in the stress process. Pearlin (1989) proposes that life events and chronic strains converge to become a stressful experience. The stressful experience occurs in one of three ways. Life events can lead to the chronic strains, chronic strains can lead to life events, or life events and chronic strains provide context for one another.
Pearlin (1989) suggests that in order to accurately interpret outcomes, the researcher must take into account multiple stressors that are present in a person’s life that lead to the outcome.

Pearlin (1989) distinguishes between primary and secondary stressors. Primary stressors are conceptualized as stressors that occur first in an individual’s life experience followed by secondary stressors. The secondary stressors are considered to be a consequence of the primary stressor and are capable of being a stronger source of stress than the primary source. Pearlin (1989) offers explanations why some life events and chronic strains are viewed as stressful to some but not others and why the same stressors may not lead to the same outcomes. Explanations include a variety of additional unobserved stressors that can impact the outcome as well as the role of values that serve to regulate the stressor. Pearlin (1989) conceptualizes values as what is defined as socially good or desirable and serves to regulate the effects of the stressor. More importantly, in addition to unobserved stressors and values, Pearlin describes a collective group of constructs he calls mediators as being responsible for explaining how the same stressor produces differences in outcomes. Finally, Pearlin (1989) describes outcomes, which are conceptualized as manifestations of stress. Figure 1 outlines the conceptual model in this study illustrating how environmental stressors impact health outcomes.

**Application of Stress Process Model to Study**

This study examined how direct or indirect environmental stressors including light and patient care activities impact sleep outcomes. Figure 1 illustrates how the stress process model guides the relationships between two stressful environmental factors and sleep outcomes. The
stress process model includes three components including stressors, mediators, and health outcomes. The experience of having a TBI (life event) is a primary stressor for patients. The life event of experiencing a TBI leads to the secondary stressor of hospitalization and an unfamiliar hospital environment, which can be a stronger source of stress for an individual (Pearlin, 1989). The conditions of the hospital environment encompass patient care activities and light that serve as secondary stressors or chronic strains that a patient encounters as a result of being hospitalized. As Pearlin’s (1989) stress process model proposes, life events and chronic strains do not occur in isolation. Additional chronic strains must be considered to accurately understand outcomes that occur as a result of the stressors. In this study, additional factors that may be influencing the primary and secondary stressors are pain, the subject’s age, and the severity of injury. Although Pearlin (1989) suggests mediating factors as constructs that help explain why the same stressors produce different outcomes, this study did not test mediating effects between environmental stressors and the quantity and quality of sleep.
**Covariates:**

- Age
- Severity of Injury
- Pain

**Stressors:**

- **Life Event:** (Primary Stressor)
  - Traumatic brain injury

- **Chronic Strains:** (Secondary Stressor)
  - Characteristics of the Hospital Environment:
    - Light
    - Patient Care Activities

**Outcomes:**

- Alterations in Sleep Patterns:
  - Quantity of sleep
    - Total Sleep Time (TST)
  - Quality of Sleep
    - Wake after Sleep Onset (WASO)
    - Sleep Efficiency (SE)

*Figure 1. Study model*

Age, severity of injury, and pain were used as covariates in the study since these variables are known to be associated with sleep. Age related changes in sleep architecture have been described in the literature (Floyd, Janisse, Jenuwine, & Ager, 2007; Ohayon, Carskadon Guilleminault, & Vitiello, 2004). Slow wave sleep (SWS) decreases markedly with age while the number of arousals and extended wake episodes increase with age. Rapid eye movement (REM) sleep percentage of TST remains relatively constant in healthy adults although declines in REM sleep have been found in elderly with organic brain dysfunctions. Although patterns of change in sleep exist among elderly individuals, substantial inter-individual variability is apparent (Carskadon & Dement, 2011). The relationship between age and sleep has been mixed in the literature. Ponsford et al. (2013) found a significant negative relationship between age and “bedtime sleep” ($r = -23, p = .024$) and TST ($r = -.22, p = .027$). In contrast, Cantor et al. (2012) found age to be non-significant between subjects with and without insomnia at 1-year and 2-years post injury.

Initial severity of TBI measured by the Glasgow Coma Scale (GCS) identifies the level of injury in relation to neurologic injury to the brain (BIAA, 2011). Several studies investigating the association between severity of injury and sleep have had mixed results. In a study by Makley et al. (2008), no significant difference in GCS score was found between rehabilitation patients with sleep wake circadian sleep disorders (SWCD) compared to a non-SWCD group. In contrast, other studies have shown that there is an association between severity of brain injury and sleep. Greater severity of brain injury has been found to be associated with longer daytime sleep time (Chiu, Chen, Chen, Chuang, & Tsai, 2013), longer 24-hour TST (Chiu et al., 2013; Ponsford et al., 2013), greater daytime sleepiness, and post-traumatic hypersomnia (Baumann et al., 2007; Ponsford et al., 2013). Additionally, Hou et al. (2013) found that while severity of injury was
associated with longer sleep time, mild and moderate traumatic brain injury was associated with insomnia.

Pain defined as subjective physical discomfort caused by illness or injury (Butterworth, Mackey & Wasnick, 2013) is common after TBI and may affect the relationship between environmental stressors and quantity and quality of sleep. In a study comparing TBI subjects to a general neurological (non-TBI) control group, subjects with TBI experienced more pain \( p < .001 \) and more insomnia \( p < .001 \) than those in the non-TBI control group. Additionally, for both the TBI and general neurological groups, those with complaints of pain were twice as likely to have sleep complaints (Beetar, Guilmette, & Sparadeo, 1996).

Hou et al. (2013) reported that symptoms of depression or anxiety were risk factors associated with insomnia in community-dwelling persons with TBI while Ponsford et al. (2013) found that in a sample of 153 mild to severe persons with TBI in the community setting, depression and anxiety were associated with poorer sleep quality. Cantor et al. (2012) also investigated the relationship between insomnia, fatigue and sleepiness in TBI subjects during the first 2 years post-injury. Relationships were found between insomnia and fatigue, anxiety, depression, and overall quality of life.

**Summary**

Restorative sleep is important for maintaining health and wellness and even more essential for persons recovering from TBI. Unlike acute care settings, persons with TBI in rehabilitation are expected to perform in therapies that require both mental and physical exertion to improve cognitive and functional abilities. Therefore, the need for patients to be well rested allowing optimal performance is essential.
An abundance of literature has identified the prevalence of alterations in sleep patterns in persons recovering from TBI (Bauman et al., 2007; Kempf et al., 2010; Makley et al., 2008). Persons with TBI are a vulnerable population since they are already predisposed to sleep disturbance due to damage to areas of the brain responsible for sleep regulation. Numerous studies have investigated the impact of environmental factors affecting sleep in acute care settings (Gabor et al., 2003; Pope, 2010; Young, Bourgeois, Hilty & Hardin, 2008), although limited research exists on how environmental factors including patient care activities and light affect sleep in the inpatient rehabilitation setting. Since persons with TBI are already predisposed to altered sleep patterns, it is important to understand how environmental stressors in the hospital setting can further compound sleep disturbance.

**Conceptual Definitions**

Patient care activities are environmental stressors that are direct or indirect patient care activities in the subject’s room during nighttime between 11:00 p.m. and 7:00 a.m. that may interrupt sleep.

Light is a potential or actual environmental stressor involving the amount of brightness or illuminance of a beam of white light occurring between 11:00 p.m. and 7:00 a.m. that may interrupt sleep.

Sleep quantity is defined in two ways. The first dimension is total sleep time (TST) or the amount of sleep during a 24-hour period. Total sleep time includes both nighttime sleep time (11:00 p.m. to 7:00 a.m.) and daytime sleep (7:01 a.m. to 10:59 p.m.). The second dimension is sleep duration (SD) or the amount of time between the start and end of sleep between 11:00 p.m. and 7:00 a.m.
Sleep time (ST) is the amount of actual time scored as sleep between the start and end of sleep.

Sleep quality is defined in two ways. The first dimension is sleep fragmentation or the time awake after the onset of sleep (WASO). The second dimension of sleep quality is sleep efficiency (SE) or the percentage of time asleep after nighttime sleep start time and sleep end time divided by the rest interval (11:00 p.m. to 7:00 a.m.) multiplied by 100.

Research Questions and Hypotheses

The research questions and hypotheses for this descriptive correlational explanatory study are: Among patients with TBI in the rehabilitation setting:

1. What are the sleep patterns for the quantity (TST), and quality of sleep (WASO and SE)?
2. What are the types and number of patient care activities that occur during nighttime?
3. What is the light exposure during nighttime sleep?
4. What are the relationships among the number of nighttime patient care activities, nighttime exposure to light, TST, WASO, and SE?

H4A: An increase in the numbers of patient care activities at nighttime will decrease TST.

H4B: An increase in the numbers of patient care activities at nighttime will increase WASO.

H4C: An increase in the numbers of patient care activities at nighttime will decrease SE.

H4D: An increase in the amount of exposure to light at nighttime will decrease TST.

H4E: An increase in the amount of exposure to light at nighttime will increase WASO.
H4F: An increase in the amount of exposure to light at nighttime will decrease SE.

5. Controlling for age, severity of injury, and pain:

A. What effect do patient care activities and exposure to light during nighttime hours have on TST?

   H5A1: More patient care activities at nighttime will decrease TST.
   H5A2: More exposure to light during nighttime hours will decrease TST.

B. What effect do patient care activities and exposure to light during nighttime hours have on WASO?

   H5B1: More patient care activities at nighttime will increase WASO.
   H5B2: More exposure to light during nighttime hours will increase WASO.

C. What effect do patient care activities and exposure to light during nighttime hours have on SE?

   H5C1: More patient care activities at nighttime will decrease SE.
   H5C2: More exposure to light during nighttime hours will decrease SE.
Chapter 2: Literature Review

This chapter will outline the state of the science related to sleep disturbance in persons with TBI. It will begin by describing a review of the literature addressing the prevalence of sleep disturbances in TBI using objective and self-report measurement tools. Next, predisposing factors that affect quantity and quality of sleep in the TBI population will be discussed. To support the argument for the need for this study, literature related to relationships between environmental stressors in the hospital setting (patient care activities and light) and sleep quantity and quality will be presented. Additionally, effects of sleep disturbance on cognition, functional performance and mood changes will be discussed. Finally, a summary of the literature will be presented.

Prevalence of Sleep Disturbances

Sleep is a natural periodic suspension of consciousness during which the energy balance in the brain and body tissues is restored (Anafi et al., 2013; Scharf et al., 2008). Until recently, sleep was thought to be a passive and dormant part of living (NINDS, 2014). Research has now shown that sleep is a time when the body regenerates and restores the immune system, as well as an activity that affects physical and mental well-being (NINDS, 2014).

Research evidence suggests that sleep disturbances are common among persons with TBI (Duclos et al., 2016). A variety of measurement strategies have been used to measure sleep in persons with TBI including objective and self-report tools. Objective measures of sleep include polysomnography (Cho et al., 2013; Sommerauer et al., 2013; Verma, Anand, & Verma, 2007; Wiseman-Hakes et al., 2016), actigraphy (Bloomfield, Espie, & Evans, 2010; Chiu et al., 2013; Makley et al., 2009; Sinclair, Ponsford, & Rajaratnam, 2014; Sommerauer et al., 2013; Towns et al., 2016; Wallace et al., 2011), multiple sleep latency tests (Sommerauer et al., 2013; Verma &
Verma, 2007), and blood tests measuring melatonin (Shekleton, et al., 2010) or hypocretin levels (Fronczek, Baumann, Lammers, Bassetti, & Overeem, 2009). In addition to objective measures of sleep, self-report tools used to measure sleep have included standardized questionnaires such as the Pittsburgh Sleep Quality Index (Baumann et al., 2007; Fogelberg et al. 2012; Parcel et al., 2006; Ponsford et al., 2013), Insomnia Severity Index (Bloomfield et al., 2010), Epworth Sleepiness Scale (Baumann et al., 2007; Kemp et al., 2010), and diaries or logs (Bloomfield et al., 2010; Ponsford et al., 2013).

**Objective Measurement of Sleep Architecture: Polysomnography**

Polysomnography, also called a sleep study, is a physiologic, noninvasive tool used to measure sleep architecture or the movement through the stages of sleep and is considered the gold standard for measuring sleep (Chervin, 2011). Polysomnography is used for a variety of indications including diagnosis of sleep disorders, therapy effectiveness, and evaluation of sleep patterns (American Association of Sleep Medicine, 2005). Polysomnography is performed in a sleep lab or hospital setting due to equipment and human resources needed to perform the study although is considered painless with minimal risks. Numerous studies have used polysomnography to measure sleep (Kaufman et al., 2001; Schreiber, et al., 2008; Shekleton et al., 2010; Sommerauer et al., 2013) in the community dwelling TBI population.

Schreiber et al. (2008) investigated sleep architecture using polysomnography in a sample of 26 community dwelling mild traumatic brain injured persons 12 months to 21 years post injury compared to healthy controls. Results indicated that the mild TBI patients compared to controls experienced a higher percentage of Stage 2 NREM sleep ($M = 54.5\%, SD =13.4$, and $M = 46.6\%, SD = 10.4$, $p = .03$) and a lower percentage of rapid eye movement (REM) sleep ($M = 21.2\%, SD = 8.4$, and $M = 25.4\%, SD = 4.5$, $p = .05$), respectively. The increase in Stage 2
NREM sleep and decrease in REM sleep suggested that after disruptions in sleep, the persons with TBI cycled back to Stage 2 NREM sleep, which can negatively impact memory consolidation that occurs during REM sleep. Shekleton et al. (2010) also found that a sample of 23 persons with TBI at least six months post injury ($M = 430$ days) compared to 23 age and gender matched controls, experienced an increase in wake after sleep onset ($M = 62.33$ minutes, $SD = 43.76$, and $M = 27.05$ minutes, $SD = 17$, $p = .003$) and a decrease in SE ($M = 82.15\%$, $SD = 9.79$, and $M = 89.71\%$, $SD = 5.80$, $p = .025$), respectively. These results suggest that although the quantity of time spent sleeping was similar to the control group ($M = 6.53$ hours, $SD = 1.22$, and $M = 6.87$ hours, $SD = .68$, $ns$, respectively), the TBI group spent more time a WASO resulting in fragmented sleep patterns. Although not statistically significant, results showed a trend toward more SWS ($p = .091$), and less REM sleep ($p = .075$) in the TBI group. Since REM sleep is considered restorative sleep, achieving adequate sleep time during this stage is important not only for healthy persons, but for those recovering from brain injury.

As previously described, as the night progresses in normal sleep, REM sleep increases with each cycle while Stage 3 and 4 NREM sleep (SWS) decreases significantly, with Stage 2 NREM occupying most of NREM sleep. When persons experience an increased sleep need or sleep deprivation, they often experience “recovery sleep” which is manifested by an increase in SWS. Findings in the literature indicate that when there is an increase in SWS compared to normal sleep architecture, it occurs as the result of sleep deprivation (Dijk, Brunner, Beersma, & Borbely, 1990), evening naps (Werth, Dijk, Achermann, & Borbely, 1996), and after nights involving fragmented sleep (Bonnet, 1987) which may provide one explanation why studies have shown that persons with TBI experience increases in SWS. An increase in SWS may be an indicator of sleep fragmentation experienced by persons with TBI. In a case-control study of 36
community dwelling persons with TBI compared to 36 controls, sleep measured by polysomnography indicated that persons with TBI experienced an increase in slow wave sleep (20.4%, and 13.8%, \( p = .009 \), respectively). These results may represent recovery mechanisms of the body, or consequences of an increased need for sleep (Sommerauer et al., 2013).

Studies using polysomnography have been limited by their small sample sizes, and despite being considered a valid and reliable objective measurement of sleep, it has limitations for use in the rehabilitation setting. Although polysomnography is considered the gold standard for measuring sleep, it is expensive, requires both human and technological resources, and must be performed in a lab or hospital setting, thus limiting the number of days of sleep recordings (Chervin, 2011). Because of limitations of using polysomnography both in the clinical and community settings, alternative tools have been used in research studies measuring sleep, including other objective measures such as actigraphy and multiple sleep latency tests and subjective measures such as sleep diaries/logs and self-report questionnaires. Knowledge about sleep patterns in the acute rehabilitation setting is limited. Additionally, it is not known whether environmental disruptions in the hospital setting that may disturb sleep patterns can perpetuate sleep disturbance long after discharge from the hospital setting.

**Objective Measurement of Sleep Patterns: Actigraphy**

Numerous studies using actigraphy have found that persons with TBI experience changes in the quantity of sleep, efficiency of sleep (Duclos et al., 2016; Makley et al., 2009), and how often awakening occurs throughout the night (Chiu et al., 2013). Actigraphy involves use of a portable device worn on the wrist (actiwatch) that contains an accelerometer used to record movement of a limb that is translated to either sleep or wake through sophisticated algorithms (Ancoli-Israel et al., 2003). Actigraphy has the ability to measure sleep patterns over multiple
sleep cycles including SD, SE, WASO, and sleep onset latency (SOL). Unlike polysomnography, actigraphy is not capable of measuring sleep architecture or movement through the different sleep stages (Morgenthaler et al., 2007) but allows for measurements of sleep patterns, such as the quantity and quality of sleep for numerous days or even weeks in a natural setting thus allowing for a more accurate and representative measure of typical sleep.

Actigraphy has been found to be a reliable and valid measure of sleep in a variety of populations (Ancoli-Israel et al., 2003). Several studies have tested the sensitivity (ability to detect sleep), specificity (ability to detect wake) and accuracy (total agreement) between polysomnography and actigraphy. In a study by Marino et al. (2013), sensitivity, specificity, and accuracy of actigraphy were compared to polysomnography in a group of young, old, healthy, insomniac, and night-shift workers. Sensitivity (.97), and accuracy (.86) were high although specificity was low (.33). In contrast, numerous studies have found low specificity of actigraphy compared to polysomnography possibly related to quiet wakefulness (Paquet, Kawinska, & Carrier, 2007; Sivertsen et al., 2006). In addition to the reliability of actigraphy, Kamdar et al. (2017) also found it to be feasible for use in patients in the intensive care unit setting with 97% of subjects tolerating wearing the actiwatch for 48 consecutive hours and 57% reporting the watch as being comfortable. Similarly, Towns et al. (2016) investigated the feasibility and implementation of an actigraphy protocol to evaluate both light exposure and sleep wake patterns among persons with TBI in an inpatient rehabilitation unit and found that 86% ($n = 19$) of TBI subjects were able to wear the actiwatch for three consecutive nights.

Although the benefits of assessing sleep quantity and quality using actigraphy are reported and well accepted, limitations of actigraphy include low specificity and risks for overestimating sleep quantity and SE if used on limbs with neuromuscular weakness. Actigraphy
has also been found to underestimate wake time and sleep-onset latency (Sivertsen et al., 2006). Despite the limitations of actigraphy, numerous benefits have been identified. The actigraph watch is non-intrusive, allows continuous measurement of sleep patterns for multiple days or weeks in non-laboratory settings, is low cost compared to polysomnography, and is well suited to the rehabilitation setting. This evidence can provide a better understanding of sleep patterns and an opportunity to improve outcomes in this vulnerable population.

Similar to studies using polysomnography, studies using actigraphy support the presence of sleep disturbances in both the quantity and quality of sleep in both hospitalized and community-dwelling persons with TBI. Some studies report that persons with TBI sleep similar lengths of time compared to a healthy control group (Shekleton et al., 2010) while others have found nighttime sleep (Kaufman et al., 2001) and TST (Imbach et al., 2015; Chiu et al., 2013) to be longer. In addition to nighttime sleep, Chiu et al. found 24-hour total sleep time to be longer ($M = 703.3$ minutes) compared to healthy persons (Ohayon et al., 2004). Similarly, Sommerauer et al. (2013) also found that in a case control study comparing 36 persons with TBI to healthy controls, those with TBI required longer TST ($M = 10.8$ hours, and $M = 7.3$ hours, $p = .001$), respectively. Imbach et al. (2015) investigated sleep wake outcomes in first time traumatic brain injury subjects. Potential subjects were excluded for other neurological diseases, history of alcohol or substance abuse, and psychiatric comorbidities. Findings indicated that 42 subjects who were 6 months post TBI experienced increased TST measured by actigraphy ($M = 8.3$ hours, $SD = 1.1$) compared to healthy, age matched controls ($M = 7.1$ hours, $SD = .8$, $p < .0001$).

Despite some studies indicating that persons with TBI sleep longer than healthy controls, persons with TBI report disturbances in sleep quality (Shekleton et al., 2010). This suggests that although persons with TBI who may be obtaining an adequate quantity of sleep, they are
experiencing a poorer quality of sleep. In a prospective longitudinal study investigating changes in sleep patterns of persons with TBI using actigraphy, Chen et al. (2015) found that in a sample of 53 first time mild to severe persons with TBI, mean SE was \((M = 69.77\%, SD = 10.28)\) during the first month following injury but gradually improved at three months \((M = 75.57, SD = 8.23)\), six months \((M = 77.59, SD = 7.60)\) and 12 months \((M = 82.23, SD = 5.83)\) post injury. Study findings did not indicate whether SE scores at 1-month post injury were in the hospital or community setting making it difficult to interpret potential causes of poor SE.

Makley et al. (2009) also investigated SE using actigraphy for 7 days in a sample of nine TBI subjects admitted to a brain injury rehabilitation unit. The study aim was to investigate whether improvements in SE scores correlated with the duration of posttraumatic amnesia. Seven of the nine subjects had mean SE scores less than or equal to 63\%, and compared to subjects admitted with posttraumatic amnesia (PTA), those subjects without PTA had better SE scores \((p = .032)\). While investigating the feasibility of actigraphy in TBI subjects in an inpatient rehabilitation unit (mean time since injury = 85 days), Towns et al. (2016) found the mean SE for three consecutive days of wearing the activwatch to be 80\%, while WASO was 46 minutes and total nighttime sleep time was 388 minutes.

Both quantity and quality of sleep are also disrupted by difficulties in falling asleep, increased awakenings at night, and decreased SE. Chiu et al. (2013) investigated sleep patterns of 52 TBI patients admitted to neurosurgical wards and reported that the mean daytime sleep was \(M = 307.2\) minutes, TST was \(M = 703.3\) minutes, and nighttime sleep was 396.1 minutes. Compared to normative values for healthy persons based on a meta-analysis, TBI subjects had decreased SE \((M = 68.7\%, p < .0001)\) and longer WASO (174 minutes, \(p < .0001\); Ohayon et al., 2004). Similarly, in a study of 19 adolescents three years post mild traumatic brain injury
compared with 13 adolescent controls, results based on sleep measured using actigraphy indicated that TBI adolescents experienced lower SE ($M = 90\%, SD = 5$, and $94\%, SD = 3$, $p < .05$) increased time awake after sleep onset ($M = 49$ minutes, $SD = 21$, and 28 minutes, $SD = 15$, $p < .05$), respectively and a trend toward more awakenings lasting more than five minutes ($M = 1.8$, $SD = .08$, and $M = 1.2$, $SD = .08$, $p = .063$), compared to adolescents without brain injury (Kaufman et al., 2001). Although actigraphy has been found to be a useful tool in measuring sleep patterns in persons with TBI in the community and acute setting, few studies have used actigraphy to assess sleep patterns of persons with TBI in the rehabilitation setting.

**Subjective Measurement of Sleep**

In addition to the objective evidence of sleep disturbance, persons with TBI also self-report a wide variety of sleep complaints including difficulties with initiating or falling asleep (Cantor et al., 2012; Kempf et al., 2010), maintaining sleep (Cantor et al., 2012), excessive daytime sleepiness (Baumann et al., 2007; Cohen, Oksenberg, Snir, Stern, & Groswasser, 1992; Kempf et al., 2010; Rao et al., 2014), hypersomnia (Baumann et al., 2007; Kempf et al., 2010), or disorders of a person’s sleep–wake schedule. A variety of self-report measurement tools have been used both in the hospital and community setting to assess a TBI person’s perception of sleep in isolation (Mahmood, Rapport, Hanks, & Fichtenberg, 2004) or in combination with objective measurements (Bloomfield et al., 2010) with conflicting results. Some studies have found that severe persons with TBI under-report sleep disturbances possibly due to a lack of self-awareness or underestimating the sleep disturbance in relation to other deficits from the injury (Clinchot, Bogner, Mysiw, Fugate, & Corrigan, 1998). In contrast, other studies have found that persons with mild (Beetar et al., 1996) and moderate brain injury (Clinchot et al., 1998) report more complaints of insomnia than severe brain injured persons. Since persons with TBI may lack
the cognitive ability to recall information necessary to accurately complete questionnaires, self-report tools used in isolation, may provide limited evidence in understanding sleep disturbance among persons with traumatic brain injury.

Fichtenberg, Zafontes, Putnam, Mann, and Millard (2002) compared 50 traumatic brain injury survivors with a mean time since injury of 3.8 months with 50 outpatients consisting of persons with spinal cord injury and persons with musculoskeletal injuries. Although all three groups had high Pittsburgh Sleep Quality Index (PSQI) global scores indicating low quality of sleep, the spinal cord injury and musculoskeletal group reported poorer sleep quality scores compared to the post-acute TBI group. Despite this unusual finding, 30% of the TBI group reported suffering from insomnia and problems with sleep initiation, which occurred almost twice as often as problems reported with SD. A single study by Gardani et al. (2015) investigated the presence and types of sleep disturbance among 30 persons with TBI from two sub-acute rehabilitation centers with an average time since injury of 41 months. Twenty subjects (67%) were found to have sleep disturbances based on global scores of greater than 5 on the PSQI. The mean PSQI score for those with sleep disturbances was higher compared to subjects without sleep disturbance \( (M = 8.61, SD = 3.63, \text{ and } M = 1.62, SD = .91, p < .05) \), respectively. Similarly mean ESS scores for those with sleep disturbance were higher than those TBI subjects without sleep disturbance indicating patients complained of sleepiness during the daytime \( (M = 5.44, SD = 4.66, \text{ and } M = 2.25, SD = 4.55, p < .05) \), respectively.

In a prospective study of 51 persons with TBI 6 months after injury, Baumann et al. (2007) found that subjective excessive daytime sleepiness measured as a score greater than 10 on the Epworth Sleepiness Scale (ESS) was reported by 28% of subjects indicating presence of daytime sleepiness. Similarly, Shekleton (2010) found that compared to controls, persons with
TBI at least six months post injury reported more sleep disturbance \((M = 9.2, SD = 5.0, \text{ and } M = 4.3, SD = 2.0, p < .001, \text{ respectively})\), based on PSQI results. Kempf et al. (2010) conducted a study investigating sleep wake disturbances on the same sample three years post TBI. Results indicated that sleep wake disorders persisted in 67% of subjects with hypersomnia being the most common (27%), followed by excessive daytime sleepiness (12%). Fatigue was also reported in 35% of the TBI subjects.

A longitudinal study by Clinchot et al. (1998) investigated sleep patterns of 86 traumatic brain injured subjects 14 years and older one year following discharge from a rehabilitation unit. Subjects were contacted by phone and were interviewed regarding sleep disturbances. Results indicated that 1-year post injury, 45% of the TBI subjects reported difficulty falling asleep, 25% reported sleeping more than usual, 64% reported waking up earlier than usual, and 50% reported difficulty sleeping. Of those subjects who reported sleep disturbance, 80% also reported complaints of fatigue. These findings are important since complaints of fatigue can interfere with rehabilitation efforts.

Findings suggest that persons with TBI report complaints in both quantity and quality of sleep that begin soon after the brain injury occurs and continues in the community setting. Complaints include insomnia, hypersomnia, excessive daytime sleepiness, and fatigue. Sleep disturbances that are not addressed in the hospital setting can lead to poor outcomes during the hospital stay as well as perpetuate continued sleep disturbance in the community setting.

**Predisposing Factors Contributing to Sleep Disturbance in Persons with TBI**

The evidence supporting sleep disturbance in persons with TBI is abundant and consistent in the literature. Predisposing factors that may precipitate sleep disturbance in this compromised population have been studied. A variety of biochemical, hormonal, medical,
pharmacologic, psychological, and environmental stressors have been found to precipitate sleep disturbance resulting in or exacerbating already existing cognitive, emotional and functional deficits associated with brain injury.

**Neurotransmitters**

The hypothalamus, basal forebrain and brainstem are important structures in the brain that regulate sleep (Franco-Perez, Ballesteros-Zebadua, Custodio, & Paz, 2012). Within these structures are neurotransmitters that play a unique role in regulating sleep and controlling wakefulness. Neurotransmitters involved in regulating the sleep wake cycle include histamine, serotonin, norepinephrine, hypocretin (Franco-Perez et al., 2012), acetylcholine and GABAergic cells (Siegel, 2004).

Pathophysiologic mechanisms involving damage to neurotransmitters or the hypothalamus can lead to disturbances in sleep (Mollayeva et al., 2013). When a traumatic brain injury occurs, researchers consistently find that neurotransmitters such as serotonin, dopamine and hypocretin-1 (Baumann et al., 2009) involved in the modulation of the sleep-wake cycle, are altered in the brain. This suggests that chemical changes are partially responsible for sleep disturbances found in traumatic brain injured persons. Baumann et al. (2009) found a reduction in hypocretin levels in the cerebral spinal fluid of TBI patients one day after injury, with low hypocretin levels of persons with TBI persisting six months after injury (Baumann et al., 2007).

**Hormones**

In addition to neurotransmitters, hormones can also affect sleep. Melatonin is a hormone that is converted from serotonin in the pineal gland and causes drowsiness by inhibiting the circadian alerting system. During the daytime hours when it is light, the pineal gland is inactive.
At dusk when darkness begins, the pineal gland is turned on by the suprachiasmatic nucleus (SCN) and begins to secrete melatonin, causing a person to feel sleepy.

Changes in levels of the hormone melatonin, have been found in persons with TBI (Shekleton et al., 2010). A study by Shekleton et al. aiming to investigate underlying mechanisms of sleep disturbances in persons with TBI, found that compared with age and sex matched healthy volunteers, the persons with TBI had significantly lower levels of evening melatonin production \((p = .031)\) and melatonin levels were positively associated with REM sleep \((r = .35, p = .017)\). While studies suggest that melatonin levels are reduced in persons with TBI, light during times of sleep can also impact melatonin levels further impacting sleep for persons recovering from TBI (Drouot & Quentin, 2015).

**Psychological and Physical Factors**

In addition to biochemical changes resulting from brain injury (Baumann et al., 2007), persons with TBI experience a variety of psychological and physical factors that pre-dispose them to sleep disturbance. Researchers have found that depression (Ouellet et al., 2006; Parcell et al., 2008; Ponsford et al., 2013; Tham, Fales, & Palermo, 2015), anxiety (Parcell et al., 2008; Ponsford et al., 2013), pain (Ouellet et al., 2006; Ponsford et al., 2013), and fatigue (Ouellet et al., 2006) contribute to sleep disturbance in the TBI population.

Ponsford et al. (2013) investigated subjective sleep complaints using the ESS, PSQI and a general sleep questionnaire in a sample 153 community dwelling TBI subjects compared to 128 non-injured sex and age matched controls. Based on PSQI scores, compared to non-injured controls, TBI subjects reported poorer sleep quality \((M = 6.39, SD = 4.74, \text{ and } M = 3.74, SD = 2.16, p < .0001)\), and more daytime sleepiness measured by the ESS \((M = 6.74, \text{ and } M = 5.40, p = .01)\), respectively. Subjective quality of sleep was poorer in the TBI group \((M = 5.20, SD = \)
2.17, \( n = 144 \), and \( M = 4.68, SD = 1.84, n = 114, p = .04 \) based on responses by TBI subjects on
the general sleep questionnaire. Additionally, the impact of pain, depression, anxiety, and
employment on sleep complaints was investigated. Increased pain severity \((r = .43)\), depression
\((r = .41)\), and anxiety \((r = .51)\) measured by The Hospital Anxiety and Depression Scale were
associated with subjective sleep complaints measured by the PSQI \((all \ p < .0001)\). Anxiety \((r = .29)\)
and depression \((r = .38)\) were also associated with self-reported daytime sleepiness measure
by the ESS \((p < .0001)\). Linear regression analysis found that pain \((\beta = .268, \ p = .009)\), and
anxiety \((\beta = .369, \ p = .006)\) accounted for 32.8% of the variance in sleep quality measured by the
PSQI \(F (3, 79) = 12.84, p < .001\).

Similarly, Tham et al. (2015) studied objective and subjective sleep disturbances and
correlates of sleep disturbance in a sample of 50 community-dwelling adolescents with mild TBI.
Depression was a significant predictor \((p < .001)\) of self-reported poor sleep quality measured by
the Adolescent Sleep Wake Scale. Finally, Ouellet et al. (2006) studied predictors of insomnia
syndrome based on a questionnaire and diagnostic criteria defining insomnia syndrome in
persons with TBI with a mean time since injury of greater than seven years. Logistic regression
analysis indicated that increased severity of pain \((\beta = .277)\), depressive symptoms \((\beta = .104)\), and
higher levels of fatigue \((\beta = .131, all \ p < .05)\) were identified as predictors of poor sleep quality.

**Environmental Factors Associated with Changes in Sleep**

Beyond the biochemical, hormonal, and psychological factors that may predispose
persons with TBI to sleep disturbance are environmental factors that can negatively impact
quality and quantity of sleep and consequently interfere with the recovery process.
Environmental disruptions have been found to impact the amount of sleep, quality of sleep,
timing of sleep, and sleep architecture (Colten & Altevogt, 2006). One study using actigraphy

35
indicated that sleep in intensive care units is characterized by frequent arousals, fragmentation of sleep, poor SE and prolonged sleep latencies while polysomnography data suggest increases in Stage 1 and 2 NREM sleep and a decrease in Stage 3 and 4 NREM and REM sleep (Friese, 2008). The impact of environmental disruptors on sleep is important since Stage 3 and 4 NREM sleep and REM sleep are believed to be responsible for brain plasticity, consolidation of memory, and new learning (Peigneux & Smith, 2011). Although evidence suggests environmental factors in the ICU setting negatively impact sleep, little is known about how patient care activities, and associated light in a post-acute rehabilitation setting serve as stressors affecting sleep.

**Patient care activities.** Patient care activities conducted by nurses and healthcare staff during nighttime hours, are frequent and impact a patient’s ability to have consolidated times of sleep (Kamdar, Needham, & Collop, 2012). Although many patient care activities are considered essential to preventing adverse outcomes, some are not essential during nighttime hours (Le et al., 2012) and are provided based on hospital routines and convenience of staff. Studies have been done in the ICU setting among a variety of patient populations investigating the types and frequency of patient care activities that may disrupt sleep (Gabor et al., 2003; Ugras et al., 2015). Studies have shown that disturbances in sleep result from a variety of patient care activities performed by nurses including assessments (i.e. vital signs, neurological assessments), direct care activities (i.e. repositioning, bathing, linen changes, wound care) and procedures (Celik et al., 2005; Kamdar et al., 2012; Le et al., 2012; Tamburri et al., 2004; Ugras et al., 2015). Gabor et al. (2003) investigated the effects of patient care activities and noise on sleep disturbance among mechanically ventilated patients in the intensive care unit. The mean number of patient
care activities per hour was 7.8 (SD = 4.2) accounting for 7.1% of awakenings measured by polysomnography.

Le et al. (2012) investigated the quantity and types of nocturnal nursing interventions (NNIs) performed between 10:00 p.m. and 6:00 a.m. among 40 subjects in five different ICUs (N = 200). Nocturnal nursing interventions were categorized as an assessment, intervention, activity, or patient initiated interventions. The amount and types of NNIs varied by unit with a range between 4.7 to 11.8 NNIs per night per patient, with nursing assessment being the most common. The number of actual NNIs during the eight-hour period may have been underestimated since only NNI’s performed by nursing staff were accounted for and excluded ancillary staff interactions. Since NNIs were recorded by nursing staff with no reliability checks, the overall number of NNIs reported may have been over or underestimated which could bias results. In this study, nurses were asked to report interventions that could be safely eliminated without negatively impacting patient care. Nurses from all ICUs combined reported that 13.9% of all NNIs were non-essential thus allowing subjects to have longer periods of uninterrupted sleep. Nurses from the surgical intensive care unit reported 27.2% of assessments could be eliminated while nurses from the neurological intensive care unit reported 33.3% of patient initiated activities were the most frequently activity that could be eliminated.

Freedman et al. (1999) investigated subjects’ (N = 203) perceptions of sleep quality and factors affecting sleep in the ICU setting. At day of discharge, subjects reported through a questionnaire that interruptions by health care providers were perceived as disruptive to sleep quality. Vital signs and phlebotomy were reported to be significantly more disruptive to sleep than noise, diagnostic tests, light, medication administration, and nursing interventions (p = .006). These studies suggest that some nursing care activities performed during nighttime hours
that are driven by nursing standard care protocol result in unintended negative alterations in sleep.

In a retrospective chart review study by Celik et al. (2005) investigating nocturnal patient care interactions for three consecutive nights in the intensive care unit, findings indicated that the mean number of patient interactions per night between 7:00 p.m. and 7:00 a.m. were 51 per patient per night with mouth and eye care, decubitus care, bathing, and dressing changes occurring more frequently between the hours of 12:00 midnight and 5:00 a.m. In a similar study, Tamburri et al. (2004) found that among 50 patients in four different ICU units, mean number of patient care activities per night were 43 with 62% of baths occurring between 9:00 p.m. and 6:00 a.m. Of the 147 days investigated, only nine episodes of 2 - 3 hours of uninterrupted sleep were reported suggesting that most patients were not getting enough uninterrupted sleep to allow a full sleep cycle. Since each sleep cycle lasts approximately 90 minutes, frequent interruptions from patient care activities during nighttime hours can impact the amount of restorative sleep needed for recovery.

Although a majority of studies investigating environmental factors affecting sleep have been in the ICU setting, Thomas et al. (2012) investigated sleep, disrupters of sleep, and the efficacy of a sleep-promoting intervention on a neurological ward. The sleep-promoting intervention consisted of “sleep rounds” in which actions to minimize light and noise, adjust the room temperature, assess pain, and offer bathroom assistance were provided. A study specific sleep survey to evaluate subjects’ perception of quality and quantity of sleep was administered to a total of 253 subjects at four time points. Data was collected before implementation of the sleep intervention (Phase 1), during the sleep intervention (Phase 2), during a washout period when the intervention was no longer implemented (Phase 3), and during a final phase of the study when an
enhanced version of the sleep promoting intervention was implemented (Phase 4). Although results from all four phases of the study related to quantity of sleep, sleep awakenings, and time required to fall asleep were similar, one question asking how much difficulty the patient had sleeping was significantly worse in the washout phase suggesting that the sleep-promoting intervention may have been partly responsible for improvement in subjects’ perceptions of sleep. Across all four phases of the sleep intervention, on average subjects reported sleeping five hours with three awakenings each night. Subjects also reported pain and staff interruptions as being the most disrupting factors that impaired sleep.

Another study of neurologically compromised patients investigated the impact of patient care interventions at nighttime on perceptions of sleep quality, and patient satisfaction among 82 subjects in the neurosurgical intensive care unit (Ugras et al., 2015). The mean number of patient care interventions between 7:00 p.m. and 6:00 a.m. was 42.21 ($SD = 7.45$). More than half (53.7%, $n = 44$) of subjects reported sleep disturbance based on a self-report sleep satisfaction survey. Of those who reported sleep disturbance, 39.1% ($n = 12$) reported that sleep disturbance was related to patient care interventions. The two most commonly occurring patient care interventions were vital signs ($M = 12$, $SD = .00$), and neurological assessments ($M = 11.70$, $SD = 1.47$), with interventions occurring most frequently at 6:00 a.m., 12:00 a.m., and 7:00 p.m.

In summary, studies suggest that patient care activities occur frequently throughout nighttime hours impacting the quantity and quality of sleep in the hospital setting. There is an abundance of evidence investigating the effects of patient care activities on sleep in a variety of ICU settings although little is known about the types and frequency of patient care activities that impact sleep in the rehabilitation setting. This study provides evidence about the types and
frequency of patient care activities in the rehabilitation setting and how these activities serve as stressors that impact both the quality and quantity of sleep.

**Light.** Circadian rhythms are entrained by internal cues such as hormones as well as external cues of naturally occurring light and darkness (Czeisler & Buxton, 2011). Hospital lighting can serve as an environmental stressor by altering circadian rhythms due to abnormal fluctuations in day-night lighting, when elevated exposure to light occurs during nighttime hours, or when light levels are decreased during daytime hours in attempts to promote a restful environment (Drouot & Quentin, 2015; Pisani et al., 2015). Hospital routines that occur over the course of a 24-hour period such as assessments and direct patient care activities (e.g. repositioning, medication administration) have been found to influence lighting practices (Pisani et al., 2015). While the importance of light as an environmental cue in synchronizing the circadian clock has been established in the literature (Mistiberger & Rusak, 2011), maintaining natural day-night fluctuations of light in the hospital setting has been a challenge (Bano et al., 2014). Although day night fluctuation is important, the intrusiveness of nighttime light during times when persons typically sleep is even more disruptive.

Numerous studies investigating light exposure in hospital settings have found light levels to be low. Bernhofer et al. (2014) used actigraphy to investigate the association between lighting levels and sleep, mood, and pain on among 40 subjects on five inpatient medical units over 72 hours. Exposure to light measured by actigraphy was found to be low during daytime \((M = 104.80 \text{ lux})\), and nighttime hours \((M = 7.07 \text{ lux})\) and not sufficient for circadian entrainment. Fifty percent of subjects slept less than 4 hours \((M = 233 \text{ minutes})\) between the hours of 10:01 p.m. and 6:00 a.m. and mean WASO was 112.51 minutes \((SD = 48.67)\) each 8-hour night. Additionally, based on regression analysis with light and opioid use as predictors of fatigue, lack
of light exposure significantly predicted fatigue ($p < .05$) accounting for 9% of the variation in fatigue. In a separate regression analysis, lack of light during daytime hours predicted mood disturbance, accounting for 8% of the variation in mood (Bernhofer et al., 2014). Bernhofer et al. reported reliability of light data measured by actigraphy as a limitation in the study. Although attempts were made to address issues with reliability of light data by asking subjects to keep the actiwatch uncovered and in an upright position, compliance was not addressed in findings of the study. Further investigation of the reliability of light data measured by actigraphy is needed. This study addresses the challenges of measuring light by actigraphy.

Similarly, Bano et al. (2014) investigated the influence of light measured by a luxmeter and noise measured by a phonometer on sleep quality in a sample of 99 subjects admitted to a medical ward. Results indicated that overall, mean light exposure between 11:30 p.m. and 12:30 a.m. was low ($M = 12$ lux, $SD = 24$) although subjects who slept near a window had significantly higher illuminance levels between 7:30 a.m. and 8:30 a.m. than those subjects who slept far from a window ($M = 212$ lux, $SD = 72$, and $163$ lux, $SD = 74$, $p < .001$), respectively. Subjects exposed to higher levels of morning light also reported higher subjective sleep quality ($7.5$, $SD = 1.9$, and $6.1$, $SD = 2.2$ on a scale of 1-10, $p < .05$), respectively. These findings highlight the importance of adequate light during daytime hours while minimizing exposure to light during evening and nighttime hours to assist in synchronizing the circadian clock.

Cho et al. (2013) compared 10 healthy subjects with a mean age of 27 years who were exposed to one night of 40 lux light during sleep and one night of sleep without light. Subjects exposed to light during the night had sleep characterized by increased arousal levels, increased Stage 1 NREM sleep, and decreased Stage 3 and Stage 4 slow wave sleep. This suggests that even low exposure to light during nighttime hours can alter sleep architecture, impacting
restorative sleep. An exploratory study by Missildine, Bergstrom, Meininger, Richards, and Foreman (2010) investigated the relationship between environmental factors (light and noise), and sleep between 11:00 p.m. and 7:00 a.m. in a sample of 48 hospitalized medical patients, age 70 and older. Light was measured with a light meter secured behind the head of the subject’s bed and noise was measured with a dosimeter. Mean nocturnal sleep time was 225 minutes, (SD = 137) with a mean SE of 46%. The mean light level between 11:00 p.m. and 7:00 a.m. was 64 lux indicating subjects were exposed to light during the nighttime hours. Although multiple regression analysis found that age, light, and noise were not predictors of SE (R² = .047, F = .756, p = .524), the authors reported that the study was underpowered. In addition to objective measures of sleep disturbance related to light exposure during nighttime hours, Ehlers, Watson, and Moleki (2013) found that 64.7% (n = 22) of patients in an ICU setting reported nighttime light as contributing to complaints of sleep deprivation. These studies highlight the impact of light exposure during nighttime hours on both sleep architecture, and the quality, and quantity of sleep. A limitation of these studies were small samples sizes further highlighting the need for additional studies to better understand the how light serves as a stressor in the hospital environment affecting sleep patterns.

Studies suggest that light exposure in the hospital setting is altered and does not mimic normal light and dark fluctuations that allow for entrainment of circadian rhythms. Hospital routines that occur throughout nighttime hours are at least partially responsible for exposure to light during times of sleep while attempts to promote a restful environment during daytime hours has been attributed to low light levels during daytime hours. Although the current study is not addressing daytime light, this study highlights how light exposure during nighttime hours affects sleep in the rehabilitation setting.
Sleep Outcomes

The importance of sleep on maintaining health has been well established in the literature, although equally as important is optimal quantity and quality of sleep when person are recovering from injury and or illness. Disturbances in sleep have been linked to cognitive deficits, diminished immune function, impairments in respiratory function, an increased risk of cardiovascular events, and metabolic syndrome (NINDS, 2014). More specifically, sleep disturbances have been shown to effect cognition, mood, and fatigue in the TBI population (Bloomfield et al., 2010; Killgore, Balkin, & Wesensten, 2006; Lundin, de Boussard, Edman, & Borg, 2006; Rao et al., 2014). Rehabilitation goals for persons with TBI are to improve cognitive and physical abilities to promote the highest level of functioning. Rehabilitation requires patients to be well rested to promote significant gains during the acute rehabilitative phase of recovery, thus the importance of optimizing sleep for persons recovering from TBI cannot be underestimated.

Cognitive Outcomes

Cognition encompasses mental activities including thinking, attention, memory, understanding, and learning and have been found to be affected by sleep disturbances (Alapin et al., 2000; Bloomfield et al., 2010; Djonlogic, Saboisky, Carusona, Stickgold, & Malhotra, 2012; Killgore et al., 2006; Makley et al., 2009; Nakase-Richardson et al., 2013; Wiseman-Hakes, Victor, Brandys, & Murray, 2011). In a sample of TBI subjects at least three months post injury, Bloomfield et al. (2010) investigated whether 11 ‘poor’ TBI sleepers defined by actigraphy, PSQI, Insomnia Severity Injury scores, and self-report had poorer sustained attention than 15 ‘good’ TBI sleepers. Indications for ‘poor sleep’ included a total sleep time less than 6.5 hours, sleep onset latency of more than 30 minutes, or SE less than 85% at least three times a week. A
Mann-Whitney test indicated that compared to ‘good’ sleepers, persons with TBI with poor sleep had significantly poorer sustained attention measured by the Sustained Attention to Response Test ($p = .032, r = -.42$).

In a prospective observational study, Nakase-Richardson et al. (2013) investigated the prevalence, course, and impact of sleep disturbance on cognition one-month post TBI. Sleep wake cycle disturbances (SWCD) were measured by a single item on the Delirium Rating Scale (DRS) ranging from zero indicating no SWCD to three indicating severe SWCD. Sleep wake cycle disturbance was found to be common (84%) with 66% of subjects having moderate or severe ratings on the DRS. Results also found that using a general linear model analysis, sleep disturbance one-month post injury predicted post-traumatic amnesia measured by the Galveston Orientation and Amnesia Test ($F = 105.4, p = .01$).

Similarly, in a prospective study of nine TBI patients in an inpatient acute rehabilitation setting, Makley et al. (2009) found that patients admitted to the rehabilitation unit without post traumatic amnesia (PTA) measured by The Orientation Log (O-LOG), which is a quantitative measure of orientation status, had better SE measured by actigraphy than those with post traumatic amnesia ($Mdn = 75\%$, and $51\%, p = .032$), respectively. Additionally, regression analysis demonstrated that for every 10-unit increase in SE, there was a 1-unit increase in O-LOG scores for patients with PTA (slope = 1.05, $p = .056$). Although the sample sizes for both studies were small, the results provide initial evidence of a relationship between sleep quality and memory in TBI subjects that requires further investigation.

The importance of not only sufficient quantity of sleep, but continuous periods of uninterrupted sleep has been found to have an effect on memory consolidation. In a study done by Rolls et al. (2011) investigating the effects of sleep continuity on mice, results indicated that
sleep fragmentation led to reduced performance on object recognition as compared to a control group of mice that were allowed to sleep naturally. This suggests the importance of, and need for quality sleep. Since sleep is needed to regenerate the body and allow the brain to function optimally, promoting sleep is essential to optimizing recovery in those with TBI.

**Functional Performance**

Improving functional performance, similar to improving cognition, is an important goal for persons recovering from TBI. Although no research has been conducted on the effects of sleep on function in the TBI population, sleep disturbance has been linked to changes in functional performance in non-TBI subjects (Dam et al., 2008; Goldman et al., 2007; Patel et al., 2008). In a study of 2,862 community dwelling men, age 65 or older, Dam et al. (2008) investigated sleep quality and the association with physical function. Results indicated that subjects with SE less than 80% and WASO greater than 90 minutes had poorer performance on grip strength, walk speed, narrow walk, and ability to perform chair stand compared with men who had SE scores of greater than 80% and WASO less than 90 minutes. In a similar study of 2,264 community-dwelling subjects over 75 years, Goldman et al. (2008), found that subjects who slept six hours or less during the night had 4.3% higher fatigue scores based on the Revised Piper Fatigue Scale than subjects who slept seven hours or greater.

Although no studies have examined the association between sleep and function in the TBI population, several studies have investigated the relationship between sleep and fatigue. Fatigue is common in person’s post-TBI (Chaput et al., 2009) with prevalence rates as high as 73% (Riggio, 2011). Fatigue has been found to be both a risk factor for (Cantor et al., 2012) and a consequence of sleep disturbance (Gardani et al., 2015). Although the causes of fatigue after TBI are multifactorial, studies have found fatigue to be associated with sleep disturbances. In a
study of 30 severe persons with TBI in a rehabilitation setting, subjects with sleep-wake cycle disorders measured by the International Classification of Sleep Disorders (ICSD-2) had significantly higher levels of fatigue measured by Barrow Neurological Institute Fatigue Scale (Gardani et al., 2015). Fatigue can also occur as a result of neuronal injury to the arousal centers in the brain causing subjective complaints of fatigue (Ponsford & Sinclair, 2014). If fatigue becomes chronic, it can interfere with the rehabilitation process and be a barrier to recovery.

**Mood Outcomes**

In addition to cognitive changes associated with sleep disturbances, changes in mood states have also been reported in persons with TBI with sleep disturbances (Parcell et al., 2008; Rao et al., 2014). Sleep wake disturbances have been linked to depressive symptoms (Cantor et al., 2012), anxiety (Cantor et al., 2012; Rao et al., 2014), mood alterations, irritability (Chaput et al., 2009; Parcell et al., 2008) and apathy (Rao et al., 2014). In a study of 10 moderate to severe brain injured persons, Parcell et al. (2008) evaluated the relationship between changes in sleep quality and mood states. Ten community-based moderate to severe TBI subjects ($M = 516$ days post injury) were compared with 10 age and sex matched controls from the general community. persons with TBI reported significantly poorer sleep quality ($p < .01$) based on the PSQI score, and higher levels of depression, and anxiety measured by The Hospital Anxiety and Depression Scale ($p = .01$; Parcel et al., 2008).

In a similar retrospective study of 443 previously hospitalized patients with mild TBI, Chaput et al. (2009) examined the relationship among subjective sleep complaints, headaches, and mood alterations at 10 days and 6 weeks post injury through a retrospective chart review and documented follow-up phone interviews. Results indicated that sleep complaints were associated with depressive symptoms, feeling irritable, and headaches at both time points. Patients with
sleep complaints were 9.9 times more likely to report feeling depressed at 10 days and 6.3 times at 6 weeks, and 12.2 times more likely to report irritability at 10 days, and 4.8 times at 6 weeks. Reports of sleep complaints were also 2.3 times more likely to experience headaches at 10 days and 3 times more likely to report headaches at 6 weeks. Similarly, in a longitudinal study, Rao et al. (2014) investigated the relationship between sleep disturbance and neuropsychiatric disturbances in a sample of 101 first time brain injured subjects. Results from a univariate regression analysis suggested that sleep disturbance measured within three months of injury by the Medical Outcomes Scale predicted increased symptoms of anxiety, depression ($p < .0001$) and apathy ($p = .032$ and $p = .000$) at 6 and 12 months respectively. Since studies have identified a relationship between anxiety, depression, and sleep disturbances, this study controlled for these potentially confounding variables by excluding persons with TBI with a history of anxiety or depression.

Findings across studies consistently suggest that sleep disturbance can affect both mood, and cognition limiting the ability to maximize recovery from brain injury. Although this study did not investigate the effects of sleep on cognitive, mood or functional outcomes, it is important to understand the impact that sleep has on outcomes in the TBI population in the rehabilitation setting where therapy to improve these outcomes is the most important reason for rehabilitation therapy. This study provides initial evidence regarding sleep patterns in persons with TBI to support future studies of this population.

**Summary of Sleep in TBI**

Findings from the literature indicate as many as 30% to 84% of persons with TBI suffer from sleep disturbance after brain injury and 25% to 29% are diagnosed with a sleep disorder, which is higher than the general population (Mathias & Alvaro, 2012). Less is known about
sleep patterns of persons with TBI in the rehabilitation setting where significant gains in recovery can occur.

Studies have shown that actigraphy has been widely used to measure sleep in a variety of populations including TBI and has been validated in several populations (Van De Water, Holmes, & Hurley, 2010). Actigraphy has been used to measure sleep variables including TST, WASO, SE, and sleep onset latency (SOL). It has been used as an alternative to polysomnography due to its ease in use, ability to measure sleep for extended periods of time, and low cost compared to polysomnography. Although there are many benefits of actigraphy, limitations include low specificity resulting in risks of overestimating sleep time and SE (Sivertsen et al., 2006). Although actigraphy has been used extensively to measure sleep in the TBI population, much of the research has focused on acute inpatient settings and community dwelling settings where both medical acuity and physical, and physiological demands on subjects are different.

The literature also supports that persons with TBI have multiple factors that predispose them to sleep disturbances. Baumann et al. (2009) has identified that neurotransmitters important in regulating the sleep wake cycle are disturbed after a TBI. Wallace et al. (2011) identified mild traumatic brain injury, higher levels of fatigue, depression, and pain to be factors associated with insomnia in the TBI population, while Orff, Ayalon, and Drummond (2009) identified mild traumatic injuries to be more correlated with sleep disturbance than moderate or severe brain injury.

In addition to the literature investigating intrinsic factors that can affect sleep, numerous research studies have focused on exploring the effects of hospital environmental factors on sleep. Many studies conducted in the ICU setting (Freedman et al., 1999, Gabor et al., 2003; Le et al.,
2012; Ugras et al., 2015) have found patient care activities to be disruptive to sleep patterns. Less is known about how patient care activities affects sleep during the rehabilitative phase of recovery. Light has also been studied and has been found to affect sleep patterns (Bernhofer et al., 2014) as well as negatively affect stages of sleep that are considered restorative sleep (Cho et al., 2013). This study examines modifiable environmental factors in the inpatient rehabilitation setting as stressors that negatively impact sleep.

Predisposing factors to sleep disturbance, negative outcomes have also been associated with sleep disturbance. Poor sleep quality has been found to be associated with language processing, attention, memory (Wiseman-Hakes et al., 2011) anxiety, and depression (Parcell et al., 2008). Additionally, Lemola, Ledermann, and Friedman (2013) found that higher day-to-day variability in SD was associated with lower levels of subjective well-being. Sleep wake disturbances have also been associated with fatigue in the TBI population (Gardani et al., 2015). This study will serve as evidence to support evaluation of the necessity of patient care activities and light exposure during nighttime hours and provide a basis for making policy changes that optimize sleep in the hospital setting.
Chapter 3: Methodology

The aims of this study are to: (a) describe the sleep patterns of adults with traumatic brain injury in the acute rehabilitation setting; and, (b) examine the effects of environmental factors (light, and patient care activities) on sleep patterns.

Institutional Review Board Approval

IRB approval was obtained from MetroHealth Medical Center and Kent State University study protocol # IRB15-00861 (see Appendix A).

Design, Setting and Sample

A descriptive, correlational, explanatory design was used for this study.

Setting

Participants were recruited from an acute traumatic brain injury rehabilitation unit located in a 67-bed rehabilitation hospital associated with The MetroHealth System, an urban teaching hospital in the Midwest, United States. The brain injury unit is a 19-bed unit with an average daily census of 13 patients. In 2016 there were approximately five admissions each week with an average length of stay of 17.15 days (MetroHealth Rehabilitation Institute of Ohio Year End Report, 2016). A multidisciplinary staff including physicians, nurses, physical therapists, occupational therapists and speech therapists provide inpatient treatment including medical and nursing care, as well as cognitive, physical and occupational therapy to prepare patients for discharge.

Sample

A convenience sample of 64 subjects with traumatic brain injured was recruited. The target population was adults who suffered a first-time traumatic brain injury who were in the acute rehabilitative phase of recovery regardless of the severity of brain injury.
Inclusion and Exclusion Criteria

Inclusion criteria were: (a) first time traumatic brain injured subjects admitted to the acute inpatient rehabilitation unit with the diagnosis of traumatic brain injury; (b) 18 years of age or older; (c) English speaking, and (d) able to participate in three hours of therapy each day. Exclusion criteria were: (a) subjects who had any prior documented diagnosed sleep disorder including sleep apnea, parasomnia, insomnia, narcolepsy, circadian rhythm disorder, periodic limb movement disorder or hypersomnia; (b) patients with non-traumatic brain injuries, such as a brain tumor, or stroke; (c) patients with a documented diagnosis of pre-existing depression or anxiety disorder, or (d) patients on isolation precautions. If persons with TBI met inclusion and exclusion criteria and were unable to provide consent, the legal authorized representative (LAR) was contacted.

Sample Size

G-power was used to estimate the sample size for both correlation and hierarchical multiple regression analysis. Using correlation for statistical analysis, the recommended sample size was 64 based on alpha of .05, power of .80, and medium effect size of .3. Using hierarchical multiple regression for statistical analysis, 43 subjects were needed to test the amount variance in the dependent variable (sleep) can be explained by the independent variables (patient care activities and light) based on an alpha of .05, power of .80 and medium effect size of .15 with two predictor variables and three covariates (age, severity of injury, and pain). A sample size of 64 subjects was needed for this study.

Recruitment

A total of 204 potential subjects were admitted to the brain injury rehabilitation unit and screened for eligibility between March 2016 and January 2017. Sixty-nine potential subjects met
inclusion criteria. Of the 69, 37 subjects agreed to participate and 27 LARs consented on behalf of the subject. One subject who met eligibility criteria for the study left against medical advice before being approached to participate, three subjects declined enrollment and one daughter of a potential subject declined enrollment. One hundred and thirty-five subjects did not meet eligibility criteria (see Table 1).

Table 1

Subjects Screened as Ineligible

<table>
<thead>
<tr>
<th>Exclusion Criteria</th>
<th>n</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-TBI Diagnosis (Orthopedic etc.)</td>
<td>60</td>
<td>44.44</td>
</tr>
<tr>
<td>Neurological Diagnosis (Stroke, Brain Tumor etc.)</td>
<td>25</td>
<td>18.52</td>
</tr>
<tr>
<td>History of Anxiety or Depression</td>
<td>21</td>
<td>15.56</td>
</tr>
<tr>
<td>Under 18 years</td>
<td>9</td>
<td>6.67</td>
</tr>
<tr>
<td>Non-English Speaking</td>
<td>5</td>
<td>3.70</td>
</tr>
<tr>
<td>Isolation Precautions</td>
<td>5</td>
<td>3.70</td>
</tr>
<tr>
<td>History of Sleep Disorder</td>
<td>4</td>
<td>2.96</td>
</tr>
<tr>
<td>Unable to Contact Family Member (Subject lacked Capacity)</td>
<td>3</td>
<td>2.22</td>
</tr>
<tr>
<td>Prisoners</td>
<td>2</td>
<td>1.48</td>
</tr>
<tr>
<td>Limited Movement of Upper Extremities</td>
<td>1</td>
<td>.75</td>
</tr>
</tbody>
</table>

Three additional subjects were excluded including two prisoners and one subject with limited movement of both upper extremities. Prisoners were excluded since interruptions and
noise may occur more commonly in this population as well as the possibility of an unexpected discharge. Sleep data may have been affected in the subject with limitations in movement of both upper extremities by scoring limited movement as sleep.

During the study, one subject was lost to attrition when unexpectedly discharged to home 7 hours after data collection began. Therefore of the 64 enrolled, data analysis was completed on 63 subjects. No subjects voluntarily withdrew from the study or were withdrawn by the Primary Investigator as a result of adverse effects of wearing the actiwatch. The final sample for the study was 63 subjects.

**Consenting TBI Subjects**

Procedures to determine capacity to consent were based on recommendations by Applebaum and Grisso (1988) which included determining the subject’s ability to clearly verbalize a consistent choice to participate or decline, as well as understand risks, benefits, and relevant information about the study. Additionally, the subject should understand how consequences of risks and benefits apply to their situation. Brain injured subjects may have the capacity to make some decisions but not others based on the complexity of the decision and the risks and benefits of the decision. Decisions involving lower risk have a lower threshold for capacity compared to decisions involving higher risk (Glezer et al., 2011).

If the subject met inclusion and exclusion criteria, cognitive capacity to consent was assessed. First, the Primary Investigator determined whether the potential subject was oriented to person and place through information documented in the physician’s admission history and physical assessment. Disorientation to time did not exclude potential subjects from being approached with information about the study since patients often lose track of time with lengthy hospitalizations. If the potential subject was oriented to person and place, he/she was approached
about participation in the study within 72 hours of admission to the rehabilitation unit. This time frame allowed standard of care associated with the admission process to be completed, including routine admission assessments to be completed by physicians, nurses, and therapists before potential subjects were approached. This also allowed time to approach potential subjects who were admitted on the weekends. After screening procedures were complete, the informed consent form was reviewed with potential subjects.

To determine whether the potential subject understood the study and was able to consent, the potential subject was asked to repeat verbally or in writing why the study was being done, study procedures, risks, and benefits involved. If the subject was able to repeat or document the information back to the Primary Investigator, the subject was given the opportunity to ask additional questions and given time to consider enrolling in the study before written consent was obtained. Information on subjects’ ability to repeat the purpose of the study, procedures involved, risks and benefits and the ability to withdraw from the study at any time were recorded on a checklist and kept with study records (see Appendix B). After written consent was obtained, a copy of the signed informed consent form was given to the subject and the original was kept with study records locked in a file cabinet, in the Primary Investigator’s locked office.

**Consenting Legal Authorized Representatives**

If the subject lacked decisional capacity to consent for the study, based on lack of orientation to person and place through a medical record review, the Primary Investigator determined if the potential subject had a legally authorized representative (LAR) who could consent on behalf of the potential subject. Legal authorized representatives are persons who are able to provide informed consent for the subject and include the following in order of priority: spouse, adult son or daughter at least 18 years of age, parents of the potential subject, adult
siblings of the potential subject, or any other adult relative. If the potential subject was deemed legally incompetent, an appointed guardian may have provided consent (The MetroHealth Informed Consent Policy, 2009). Legal authorized representatives were approached in person if available or contacted by phone within the 72-hour time frame to describe the study and obtain consent. If the LAR was contacted in person, written consent was obtained. If the LAR was unable to meet in person, verbal phone consent was obtained with a witness per hospital policy (The MetroHealth Informed Consent Policy, 2009). Twenty-eight LARs were approached about participation in the study, and 27 consented.

**Measures**

Six measures were performed and described in the study: objective and subjective sleep, DPCAs, light, covariates, and demographics.

**Sleep Patterns**

Sleep patterns including the quantity and quality of sleep were measured. Sleep quantity was measured as TST in minutes, which included both daytime and nighttime sleep. Sleep quality included WASO (fragmentation of sleep) and SE. Wake after sleep onset was defined as the amount of time in minutes scored as wake between the start and end of nighttime sleep, and SE was defined as the percentage of time asleep after nighttime sleep start time and sleep end time divided by the rest interval (480 minutes) multiplied by 100 (Spruyt, Gozal, Dayyat, Roman, & Molfese, 2011). Additional sleep variables and definitions are listed below (see Table 2).

Sleep patterns were measured using the Actiwatch 2 (Philips Respironics, Inc., Murrysville, PA) activity logger. An activwatch unit is a small waterproof device that has the
Table 2

*Sleep Variable Definitions*

<table>
<thead>
<tr>
<th>Variable Name</th>
<th>Operational Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest Interval</td>
<td>Time between 11:00 p.m. and 7:00 a.m. (480 minutes)</td>
</tr>
<tr>
<td>Nighttime Sleep Interval</td>
<td>Sleep between 11:00 p.m. and 7:00 a.m.</td>
</tr>
<tr>
<td>Daytime Sleep (Naps)</td>
<td>Sleep between 7:01 a.m. and 10:59 p.m. Daytime sleep included the sum of all nap minutes. Naps included any episode of sleep during daytime hours lasting 10 minutes or longer.</td>
</tr>
<tr>
<td>Total Sleep Time (TST)</td>
<td>Total sleep including both daytime sleep and nighttime sleep time measured in minutes.</td>
</tr>
<tr>
<td>Sleep Duration&lt;sup&gt;a&lt;/sup&gt;</td>
<td>The total amount of time between the start and end of nighttime sleep measured in minutes (including wake after sleep onset minutes and wake bouts).</td>
</tr>
<tr>
<td>Sleep Time&lt;sup&gt;a&lt;/sup&gt;</td>
<td>The amount of time scored as sleep between the start of nighttime sleep and end of nighttime sleep measured in minutes. Nighttime sleep time is typically shorter than nighttime SD.</td>
</tr>
<tr>
<td>Wake Time after Sleep Onset&lt;sup&gt;a&lt;/sup&gt;</td>
<td>The amount of time scored as wake between the start and end of nighttime sleep measured in minutes regardless of number of wake bouts.</td>
</tr>
<tr>
<td>Sleep Efficiency (SE)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Nighttime sleep time divided by the rest interval (480 minutes) times 100. Measured as a percentage.</td>
</tr>
<tr>
<td>Number of Wake Bouts&lt;sup&gt;a&lt;/sup&gt;</td>
<td>The total number of episodes of wake time during nighttime sleep.</td>
</tr>
</tbody>
</table>

*Note.*<sup>a</sup>Philips Respironics, 2015.

Appearance of a wristwatch that measures movement that is translated into sleep or wake. The actiwatch uses a piezoelectric accelerometer that monitors occurrence and degree of motion. The
degree and speed of motion produce an electrical current that varies in magnitude. As the degree and speed of motion increase, the voltage increases which is stored in the actiwatch as activity counts. Activity counts are measured at a time frame or epoch designated by the investigator and can range from 15 seconds to 2-minute epochs (Philips Respironics, 2015). A 1-minute epoch was used for this study based on similar studies using actigraphy to measure sleep in TBI subjects (Sinclair et al., 2014a; Chui et al., 2013). Activity counts are generated as a weighted average for each epoch and surrounding epochs. Scoring calculations for one-minute sampling epochs were calculated as the activity count of the epoch being scored, plus the activity count before and after the epoch times one fifth, plus the epoch 2 minutes before and after times one twenty-fifth using Actiware 6.0.5 proprietary commercial software (Philips Respironics, 2015).

The Actiwatch 2 uses calculations that score individual epochs as sleep or wake, using activity count threshold values via the Cole-Kripke algorithm (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992). The algorithm is used to calculate all sleep variables. Activity count threshold values can be set at 20, 40, 60 and 80 with 20 being the most sensitive, meaning that smaller movements are scored as wake, compared to higher threshold values (Lichstein et al., 2006). If the activity count for one epoch exceeds the set activity threshold, the data is scored as wake. If the activity count does not meet the threshold, the data is scored as sleep. The activity threshold value for this study was set at 40 since studies have shown that limitations of actigraphy include overestimating TST due to scoring quiet wakeful periods as sleep (Sadeh, 2015).

Actigraphy has been compared to polysomnography in numerous studies using a variety of different populations. In a systematic review by Van De Water et al. (2010), results suggest that the accuracy of actigraphy as compared to polysomnography depends on the variables of interest, population being studied, algorithm and threshold used, and the make and model of the
actigraph unit. In a study of 31 subjects with insomnia, correlations between actigraphy and polysomnography were $r = .92$ for total sleep time, $r = .77$ for SE, and $r = .85$ for WASO (Sanchez-Ortuno, Edinger, Means, & Almirall, 2010).

In this study, inter-device reliability testing was performed to determine agreement between the actiwatch units. The Primary Investigator wore three actiwatches on the same wrist to compare sleep data findings. Sleep duration had 100% agreement across the three watches. Sleep efficiency results had close agreement across watches, with actiwatch A-88.13%, actiwatch B-87.71%, and actiwatch C-88.30%. Wake time after sleep onset also had small differences between the actiwatches with watch A-17 minutes, actiwatch B-19 minutes, and actiwatch C-16 minutes. These small differences could have been related to the sequential timing of applying and removing the watches.

Previous studies have described potential sources of error including the actiwatch interpreting sleep as wake (false positive) or wake as sleep (false negative). Wake periods can be interpreted as sleep when the subject is awake, but without movement. Contrarily, sleep can be interpreted as wake if a subject has movement such as tremor or spasticity when sleeping. To minimize error, information about presence of paresis, contracture, or spasticity was obtained as part of the screening process from either the physician history and physical, or the physical or occupational therapy admission assessments in the electronic medical record. The information was used to determine which wrist the actiwatch was applied based on the recommendations of Zollman, Cyborski, and Duraski (2010) suggesting the actiwatch be placed on the limb with the least paresis, contracture or spasticity to improve accuracy of sleep measurements. If there was no evidence of paresis, contracture, or spasticity the watch was placed on the non-dominant
wrist. Five subjects had paresis of the upper extremity therefore the actiwatch was placed on the opposite wrist.

**Perceptions of Sleep**

The Verran and Snyder-Halpern (VSH) Sleep Scale was used to investigate the subjects’ perception of sleep as well as the relationship between subjective and objective measures of sleep. The VSH Sleep Scale (Appendix H) is an 8-item visual analogue scale used to measure the subjects’ perceptions of sleep. The possible range of total scores is between 0 – 800 with higher scores indicating better perceptions of sleep. The scale is a subjective measure of sleep characteristics including sleep fragmentation ($n = 2$), length ($n = 1$), delay ($n = 1$), and depth of sleep ($n = 4$). Subjects respond to questions based on their previous night’s sleep by placing a vertical mark on a 100-mm response line. Scores for two questions related to sleep fragmentation and one question related to a delay in falling asleep are reversed before summing for a total score. A Cronbach alpha of (.82) has been established for the instrument indicating adequate reliability (Snyder-Halpern & Verran, 1987). The Cronbach alpha for this study was .77 indicating acceptable reliability. When compared to the St. Mary’s Hospital Sleep Questionnaire, which measures a subject’s perception of a previous night’s sleep, and Sleep Log, evidence of convergent construct validity was established (Snyder-Halpern & Verran, 1987). Only subjects who had the capacity to consent ($n = 37$) were asked to complete the VSH scale. Subjects without capacity to consent would not have had the cognitive abilities to reliably answer the questions.

**Patient Care Activities**

The Patient Care Environmental Stressor Log (PCESL) was used to measure patient care activities performed by professional or non-professional staff between the hours of 11:00 p.m.
and 7:00 a.m. (see Appendix F). The PCESL was a tool developed by the investigator based on similar studies investigating the effects of environmental factors on sleep (Celik et al., 2005; Kamdar et al., 2012; Le et al., 2012; Tamburri et al., 2004; Ugras et al., 2015). The tool included patient care activities that could be documented in 15-minute increments and included indirect, and direct patient care activities. Indirect patient care activities included observation, documenting in room, managing equipment, and care of a roommate. Direct patient care activities included vital signs, assessments, medications, dressing changes, restraint application or removal, repositioning, bathing, dressing, toileting, transfers, radiological procedures, tracheostomy care, suctioning, venipuncture’s or other patient care activities not listed on the data collection form that took place in the subject’s room between 11:00 p.m. and 7:00 a.m. If a non-nursing health care provider entered the room to perform a patient care activity such as a radiological procedure or respiratory procedure, the nursing staff was asked to document the patient care activity on the PCESL.

A meeting with all levels of nursing staff was conducted to discuss the purpose and procedures involved in the study. Nursing staff members (five registered nurses and nine unlicensed staff) were educated through group sessions on how to complete the PCESL prior to initiating the study (see Appendix G). Verbal permission to participate in this study was obtained from nursing staff to complete the Patient Care Environmental Stressor Log (PCESL). Informal education including verbal reminders and answering targeted questions pertaining to documentation of patient care activities and the actiwatch occurred several times throughout the study. The Primary Investigator was present on the brain injury unit during the night shift for the first two subjects who were enrolled to answer questions pertaining to documenting on the PCESL.
To minimize risk of systematic or random measurement error, systematic reliability checks were done by the Primary Investigator alternating day one and day two on every fifth subject for a total of 13 subjects comparing percent agreement between patient care activities documented on the PCESL with patient care activities documented in the electronic medical record. If less than 85% agreement was found between the documentation on the PCESL and data abstracted from the electronic medical record, the individual staff was identified and re-educated. Results showed an agreement between the PCESL flowsheet and the electronic medical record documentation to be between 94% and 100%.

**Light**

Light was measured in lux with a photodiode element of the Actiwatch 2 ambient light monitor, Philips Respironics. The optical sensor on the light monitor has sensitivity similar to the human eye with a lumen sensitivity range of .01 lux to 150,000 lux. Lux is a unit of measurement that is equal to one lumen of light per square meter (Philips Respironics, 2015).

The photodiode element measures the amount and duration of illuminance of white light. Measures of illuminance of the Actiwatch 2 were compared to National Institute of Standards and Technology (NIST)-traceable light sources and testing apparatus (Philips Respironics, 2008). A random sample of Actiwatch 2 activity monitors were exposed to four commonly used types of white light. Results indicated agreement between the Actiwatch 2 and the NIST-traceable photometer (Philips Respironics, 2008). Similarly, in a study examining the congruence of a stand-alone light meter versus a wrist worn light meter in hospital rooms of intensive care units, results found the mean 24-hour light levels 31.3 lux ($SD = 21.4$) measured by the light meter and 23.9 lux ($SD = 16.1$) measured by the sleep watch. The difference between the two devices was
within the standard deviation of 1.96 indicating good agreement between devices (Higgins, Winkleman, Lipson, Guo, & Rogers, 2007).

In a similar study, Jardim et al. (2011) compared a wrist-worn light monitor (Actiwatch-L) to an eye level light monitor attached to the forehead of the subject (Daysimeter) in a sample of 12 post-operative cardiac surgery patients. Lux levels were higher for the Daysimeter compared to the Actiwatch-L for both daytime, \((M = 156 \text{ lux}, \text{ and } M = 128 \text{ lux})\), respectively, and nighttime \((M = 28 \text{ lux}, \text{ and } M = 19 \text{ lux})\), respectively. When light levels were less than 5000 lux, a difference of 10 lux was found indicating good agreement between devices. Agreement between devices decreased when light levels rose above 5000 lux to a difference of greater than 100 lux.

In this study, to test the inter-device reliability, the Primary Investigator wore three watches inches apart on the left arm simultaneously during one night between 11:00 p.m. to 7:00 a.m. Total light exposure ranged between 3.53 lux and 3.72 lux indicating good agreement. Similarly, average lux was \(M = .01 \text{ lux} (100\% \text{ agreement})\) and the mean maximum lux exposure ranged from .02 to .07 lux across all three watches. The difference in lux exposure, and maximum lux exposure results could have been attributed to positioning of the arm, small differences in the positioning of the watch or the angle of the light in relation to the watch.

The reliability of light data in this study was also tested by comparing results of watches worn on the wrist by a random sample of seven different subjects on 11 different episodes to watches attached to the headboard of the subject’s bed. Total light exposure, average light exposure, and maximum light exposure results for actiwatches worn on subjects and headboards are presented (see Table 3).
To further investigate level of agreement between actiwatches worn on subjects and actiwatches attached to the headboard, the Bland Altman Test was conducted (Bland & Altman, 2010). First, a one-sample t-test was conducted to test the null hypothesis that the mean difference between measurements was zero. The mean difference for total light exposure was $M = -8440.23$ lux, $SD = 26806.60$, $p = .321$, average light exposure was $M = -17.43$ lux, $SD = 55.36$, $p = .321$, and maximum light exposure was $M = -36.55$ lux, $SD = 59.74$, $p = .070$ indicating some level of agreement between the two measurement methods.

Table 3

Comparisons of Actiwatch on Subject and Headboard

<table>
<thead>
<tr>
<th>Subject</th>
<th>Total Light Subject</th>
<th>Total Light Headboard</th>
<th>Average Light Subject</th>
<th>Average Light Headboard</th>
<th>Maximum Light Subject</th>
<th>Maximum Light Headboard</th>
</tr>
</thead>
<tbody>
<tr>
<td>111b</td>
<td>3.09</td>
<td>4.01</td>
<td>.01</td>
<td>.01</td>
<td>.02</td>
<td>.07</td>
</tr>
<tr>
<td>114b</td>
<td>17.30</td>
<td>5.39</td>
<td>.04</td>
<td>.01</td>
<td>7.83</td>
<td>.22</td>
</tr>
<tr>
<td>149b</td>
<td>10639.13</td>
<td>99865.75</td>
<td>24.23</td>
<td>208.49</td>
<td>250.51</td>
<td>439.97</td>
</tr>
<tr>
<td>150a</td>
<td>606.47</td>
<td>3402.49</td>
<td>1.28</td>
<td>7.10</td>
<td>177.14</td>
<td>273.19</td>
</tr>
<tr>
<td>150b</td>
<td>251.74</td>
<td>772.94</td>
<td>.60</td>
<td>1.61</td>
<td>56.20</td>
<td>84.82</td>
</tr>
<tr>
<td>154a</td>
<td>5.87</td>
<td>13.10</td>
<td>.01</td>
<td>.03</td>
<td>.65</td>
<td>2.54</td>
</tr>
<tr>
<td>154b</td>
<td>42.17</td>
<td>80.08</td>
<td>.09</td>
<td>.17</td>
<td>2.04</td>
<td>48.29</td>
</tr>
<tr>
<td>158a</td>
<td>3.56</td>
<td>3.80</td>
<td>.01</td>
<td>.01</td>
<td>.01</td>
<td>.02</td>
</tr>
<tr>
<td>158b</td>
<td>8.57</td>
<td>3.90</td>
<td>.02</td>
<td>.01</td>
<td>.09</td>
<td>.03</td>
</tr>
<tr>
<td>162a</td>
<td>400.86</td>
<td>669.92</td>
<td>.84</td>
<td>1.40</td>
<td>35.66</td>
<td>83.00</td>
</tr>
<tr>
<td>162b</td>
<td>4.20</td>
<td>4.14</td>
<td>.01</td>
<td>.01</td>
<td>.03</td>
<td>.11</td>
</tr>
</tbody>
</table>

Note. $N = 11$. Light parameters measured in lux. $a =$ night 1, $b =$ night 2

Next, scatterplots were constructed plotting the mean of the two light measurements on the x-axis and the difference between the two light measurements on the y-axis. Upper and lower limits of agreement were constructed representing $+1.96$ standard deviation from the mean difference between measurements (see Appendices C-E). The upper and lower levels of
agreement for total light exposure were 44100.71 and 60981.17 lux, average light exposure 91.07 and 125.94 lux, and maximum light exposure 80.53 and 153.64 lux, respectively.

Finally, a regression analysis was performed to further investigate agreement between the two light measurements. The mean difference was used as the dependent variable and the mean of the two measurements as the independent variable. Findings indicated significant results for total light exposure (B = -1.62, p < .0001), average light exposure (B = -1.59, p < .0001), and maximum light exposure (B = -0.508, p < .0001), suggesting that there were systematic differences between the two measurements suggesting bias and that results should be interpreted with caution. Although findings suggest bias in measurements, there were very only 11 observations and several outliers within the sample fell outside the limits of agreement affecting the results.

One final measure to assess the reliability of the light data from the actiwatch involved asking nursing staff to observe and document whether the actiwatch was visible, or not visible during the hourly rounds conducted between 11:00 p.m. and 7:00 a.m. for the 2 days the actiwatch was worn. Results indicated that the watch was visible 63.9% of the time during 1,893 times observed.

**Age, Pain, and Severity of Injury**

Additional variables included age measured in years at time of admission. Age was obtained in the electronic medical record. Pain was measured as the number of episodes of pain medication given during the 48-hour data collection period. The number of pain medication episodes was obtained from the medication administration record, which is part of the electronic medical record. The severity of injury was measured using the first GCS score after the initial injury. The GCS score was initially developed to describe level of consciousness and provide a
grading system of brain injury severity (Teasdale & Jennett, 1974). The scale assesses eye
tion (1-4), verbal response (1-5), and motor response (1-6), with higher scores indicating
less severe injury. Scores between 3-8 indicate severe brain injury, 9-12 indicates moderate brain
injury and 13-15 indicates mild brain injury (Kortbeek et al., 2008). The GCS has shown
adequate construct validity with the Glasgow Outcome Scale, $r = .557$ (Amirjamshidi et al.,
correlations between the GCS and GOS as low as $r = .21$ and as high as $r = .39$, $p = .011$. The
GCS was obtained in the physician history and physical; if not documented in the physician
history and physical, the GCS was obtained in the nursing assessment documentation for subjects
admitted from MetroHealth or from the medical record sent from the transferring hospital.

**Demographic Variables**

Demographic variables used to describe the sample included the admitting diagnosis,
mechanism of injury, co-morbidities, time since injury, gender, race, marital status, and
employment prior to injury. Additional variables included the time of first therapy appointment,
if subject had a roommate and if the subject was on constant supervision (direct in-person
supervision provided continuously by a staff member for safety purposes; see Appendix I). All
demographic data were obtained from the electronic medical record with the exception of time of
first therapy and whether the patient had a roommate. Demographic data were located in either
the physician history and physical or the nursing admission assessment. Time of first therapy
was located on a daily therapy schedule kept in the central nurses’ station. If patient was in a
semi-private room, the charge nurse was contacted to determine if the subject had a roommate.
Procedures

After IRB approval, verbal permission was obtained to conduct the study from the Medical Director of the Brain Injury Unit, the Director of Rehabilitation Nursing and the Nursing Unit Manager to gain support for the study. Permission to approach potential subjects or LARs was requested from the physician responsible for the medical care of the potential subjects who met inclusion and exclusion criteria.

Recruitment

To assist with recruitment, an informational flier was given to the secretary to place in standardized admission packets given to potential subjects upon admission to the rehabilitation unit (see Appendix J). A partial HIPAA waiver obtained for the study allowed the Primary Investigator to access the electronic medical record to obtain a list of newly admitted potential subjects and to allow screening for inclusion and exclusion criteria. A list of newly admitted potential subjects was obtained each morning, Monday through Friday. If subjects screened as eligible to participate in the study, the potential subject or LAR was approached for consent.

Data Collection

Sleep and light data. Before using actigraphy for measurement of sleep and light exposure, the actiwatch software was installed on the computer. The actiwatch was connected to the communication dock and configured with the subject identification number, epoch length, activity threshold, and start and stop time. This study used a 60 second epoch length, activity threshold of 40 and collected data for 48 consecutive hours based on similar studies measuring sleep (Chiu et al., 2013; Sinclair et al., 2014a).

After the actiwatch was configured, it was applied to the subject’s wrist at 8:00 a.m. between days 3 and 6 of the rehabilitation hospital stay after consent was obtained. Actigraph
sleep and light data were collected during weekdays in order to standardize both environmental factors and sleep measures for the sample of subjects since differences in rehabilitation schedules (therapies), patient care activities (for example, lab testing) and sleep patterns may have differed between weekdays and weekends. Data were collected on which wrist the actiwatch was placed and was documented on the data collection sheet. The watch was placed on the left wrist 56% of the time \( (n = 36) \) and on the right wrist 44% of the time \( (n = 28) \).

In addition to sleep parameters, light exposure was collected using the light sensor on the actiwatch. Light was measured continuously during the 48 consecutive hours that the actiwatch was worn. To decrease systematic or random error due to subjects blocking the light sensor, subjects were asked to keep the arm with the actiwatch outside the blankets during nighttime hours. Additionally, staff were asked to position the subject’s arm to prevent blankets from covering the light sensor if physical hands-on care was provided. The actiwatch was cleaned between subjects with mild soap and a damp cloth per manufacturer’s instructions to minimize buildup of debris on the lens (Philips, Respironics, 2015). After completing the 48 hours of data collection, the watch was removed from the subject’s wrist, placed on the dock allowing sleep and light statistics to be downloaded.

**Subjective sleep data.** Subjects who had the capacity to consent for the study were asked to complete the VSH Sleep Scale by 10:00 a.m. in the morning. If assistance was needed to complete the sleep scale, the Primary Investigator assisted the subject. All subjects who completed VSH scale \( (n = 29) \) required assistance.

**Patient care activity data.** Patient care activities were collected for two consecutive nights between the hours of 11:00 p.m. and 7:00 a.m. using the PCESL (Appendix F). Staff were instructed to place a check mark on each activity that was performed during a specific 15-minute
time period on the PCESL. The PCESL was kept at the nurses’ station to prevent additional disruptions in the subject’s rooms as a result of documentation. The PCESL was kept in the room for subjects who were on constant supervision. The tool was collected daily and data were transferred into the REDCap (Research Electronic Data Capture), a secure, web-based application designed to support data capture for research studies (Harris et al., 2009).

**Data Management and Analysis Plan**

**Data Management**

The Statistical Package for the Social Sciences (SPSS), student version 24 was used as the data software program for analyzing data. Prior to data analysis, steps in the data management plan included preparing a codebook, creating a data file, entering data, cleaning the data, and inspecting data for outliers, errors, and irregularities such as values that may be out of range for possible values for the variable.

**Screening and Cleaning Data**

Data was checked for data entry errors. Categorical variables were checked to determine if they were within a plausible range of scores. Descriptive statistics were used to assess continuous variables. Minimum, maximum, and mean scores were analyzed to determine if they were within the range of scores. Incorrect or missing values were corrected if data entry errors occurred.

**Management of Missing Data**

Eight subjects had missing GCS scores and three subjects had missing sleep and light data on day two of wearing the actiwatch. One additional subject had all patient care activity, light, and sleep data missing for both days; therefore, the final sample for the study was 63 subjects. Of the 37 subjects who were asked to complete the VSH scale, five refused on both
days, one was discharged after seven hours, two subjects completed the scale which was found to have an incorrect length for the visual analogue line, thus requiring an amendment to correct the error. Of the 29 remaining subjects, six had partial missing VSH data for one of the days. Little’s Missing Completely at Random Test was done to determine if patterns of missing data were random or systematic. A significance level of \( p = .421 \) indicated that GCS scores, sleep, and light data were missing at completely at random. A second Little’s Missing Completely at Random Test was conducted on a subset \( (n = 29) \) of subjects who completed the VSH scale. A significance level of \( p = .291 \) indicated that VSH scores were also missing completely at random.

Data imputation consisted of two strategies. First, within subject analysis using general linear model testing was done to determine individual differences between day one and day two sleep and light data (see Table 4). Results indicated no statistically significant individual differences between day one and day two \( \text{SD} \) \( (p = .203) \), \( \text{SE} \) \( (p = .255) \), \( \text{WASO} \) \( (p = .744) \), and maximum light exposure \( (p = .462) \), although results for TST were significant \( (p = .014) \). Since no within subject statistically significant differences were identified, sleep and light exposure values for day one were imputed into day two scores for the three subjects with missing data. Secondly, Expectation Maximization was used as the method for replacing missing GCS and VSH scores with predicted scores.
Table 4

*Within Subject Analysis for Sleep and Light Characteristics*

<table>
<thead>
<tr>
<th>Sleep Characteristic</th>
<th>Day 1</th>
<th>Day 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M   (SD)</td>
<td>M   (SD)</td>
</tr>
<tr>
<td>Total Sleep Time</td>
<td>520.02 (150.81)</td>
<td>561.78 (189.05)</td>
</tr>
<tr>
<td>Sleep Duration</td>
<td>421.07 (81.27)</td>
<td>437.33 (76.17)</td>
</tr>
<tr>
<td>Sleep Efficiency</td>
<td>71.27 (16.74)</td>
<td>74.12 (18.49)</td>
</tr>
<tr>
<td>Wake after Sleep Onset</td>
<td>78.93 (42.06)</td>
<td>81.55 (52.07)</td>
</tr>
<tr>
<td>Maximum Light</td>
<td>33.19 (42.57)</td>
<td>39.44 (63.90)</td>
</tr>
</tbody>
</table>

*Note. N = 60*

**Testing Assumptions**

Testing for assumptions of multiple regression were assessed including multicollinearity, singularity, normality of residuals, linearity of predictor and outcome variables, and homoscedasticity (Munro, 2005).

To test for multicollinearity, correlations between predictor variables were inspected (see Table 5). Bivariate correlations between predictor variables did not exceed .43 therefore there was no violation of multicollinearity (Munro, 2005). Collinearity diagnostics were also assessed to identify multicollinearity that was not identified in the correlation matrix. The minimum tolerance value was .65 and the highest VIF was 1.55 suggesting the assumption of multicollinearity was not violated. Neither, multicollinearity, or singularity was identified between any of the predictor variables.
Table 5

*Correlations of Covariates and Independent Variables*

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. GCS</td>
<td>.43**</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Pain</td>
<td>-.30*</td>
<td>-.22</td>
<td>---</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. DPCA</td>
<td>.04</td>
<td>-.42**</td>
<td>.18</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>5. MaxLux</td>
<td>.10</td>
<td>-.02</td>
<td>-.15</td>
<td>.22</td>
<td>---</td>
</tr>
</tbody>
</table>

*Note.* *N* = 63. GCS = Glasgow coma scale; DPCA = direct patient care activities; MaxLux = maximum lux exposure.

* p < .05, two tailed. ** p < .01, two tailed.

Normality, linearity, and homoscedasticity of residuals was assessed by inspecting Normal Probability Plots of the Regression Standardized Residual (P-P Plots) and scatterplots. Descriptive statistics including skew and kurtosis were also evaluated. The P-P Plots showed scores that were in a relatively straight diagonal line from bottom to top indicating no major deviations of normality. Scatterplots were also inspected for the distribution of the residual scores with scores noted to be in a somewhat rectangular fashion. No systematic patterns such as curvilinear distributions or scores higher on one side or another were noted. No major deviations from normality were identified in the multivariate analyses.

The presence of outliers was assessed using standardized residual values of more than 3.3 or less than -3.3 (Tabachnick & Fidell, 2013) or Mahalanobis distances exceeding the critical values based on five independent variables. No standardized residuals were found to be more than 3.3 or less than -3.3. A Mahalanobis distance of 27.37 exceeded the critical value of 20.52 for one subject. After evaluating Cook’s distance (.021), it was determined that this outlier did
not influence the model as a whole since the value did not exceed 1.0 (Tabachnick & Fidell, 2013).

In the post hoc analysis, normality and homogeneity of variance were assessed prior to conducting independent t-tests to compare differences between subjects with and without constant supervision and subjects with and without a roommate. The assumption of normality and homogeneity of variance were not met, therefore a Mann-Whitney U test was conducted.

**Statistical Analysis**

Descriptive statistics were used to describe characteristics of the study sample, assess for violations of assumptions of the statistical techniques used in the study, and to address research questions 1-3. For research question four, correlation analysis using the Pearson product-moment coefficient was used to determine the strength and direction of the relationship among the continuous variables. Finally, hierarchical multiple regression analyses were used to evaluate the hypotheses associated with research question number five.

**Evaluating the regression model.** A 3-step hierarchical model was used to determine how well direct patient care activities, and maximum light exposure predicted TST, SE, and WASO. Hierarchical multiple regression was used to determine how the independent variables added to the prediction of the sleep after previous independent variables were controlled (Tabachnick & Fidell, 2013). The stress process model that guided this study proposes that primary and secondary stressors converge to lead to an outcome. Direct patient care activities were entered in Block 2 based on numerous studies (Celik et al., 2005; Freedman et al., 1999; Gabor et al., 2003; Kamdar et al., 2012; Le et al., 2012; Tamburri et al., 2004; Ugras et al., 2015) indicating that patient care activities affect sleep. Fewer studied have been conducted on the impact of light on sleep in the hospital setting (Bernhofer et al., 2014; Bano et al., 2014; Cho et
(al., 2013; Missildine et al., 2010), therefore maximum light exposure was entered into Block 3. In each analysis, model 1 included covariates age, severity of injury, and pain. Model 2 included direct patient care activities and model 3 included maximum light exposure. R square indicated the amount of variance in the dependent variable that explained the model, while R square change indicated how much additional variance in the dependent variable was explained by the addition of variables at each step.

The research questions and hypotheses for this study are as follows: Among traumatic brain injured patients in the rehabilitation setting:

1. What are the sleep patterns for the quantity (TST), and quality of sleep (WASO and SE)?
2. What are the types and number of patient care activities that occur during nighttime?
3. What is the light exposure during nighttime sleep?
4. What are the relationships among the number of nighttime patient care activities, nighttime exposure to light, TST, WASO and SE?

H4A: An increase in the numbers of patient care activities at nighttime will decrease TST.
H4B: An increase in the numbers of patient care activities at nighttime will increase WASO.
H4C: An increase in the numbers of patient care activities at nighttime will decrease SE.
H4D: An increase in the amount of exposure to light at nighttime will decrease TST.
H4E: An increase in the amount of exposure to light at nighttime will increase WASO.
H4F: An increase in the amount of exposure to light at nighttime will decrease SE.
5. Controlling for age, severity of injury, and pain:

A. What effect do patient care activities and exposure to light during nighttime hours have on TST?

H5A1: More patient care activities at nighttime will decrease TST.

H5A2: More exposure to light during nighttime hours will decrease TST.

B. What effect do patient care activities and exposure to light during nighttime hours have on WASO?

H5B1: More patient care activities at nighttime will increase WASO.

H5B2: More exposure to light during nighttime hours will increase WASO.

C. What effect do patient care activities and exposure to light during nighttime hours have on SE?

H5C1: More patient care activities at nighttime will decrease SE.

H5C2: More exposure to light during nighttime hours will decrease SE.

Protection of Human Subjects

Although not previously reported, a possible risk was abrasion to the skin from the actiwatch wristband. Subjects’ skin was assessed before application of the actiwatch and each morning that the actiwatch was in place to monitor for early signs of irritation or breakdown. There were no instances of skin redness, irritation or abrasion. If skin irritation or breakdown occurred, the physician managing the medical care of the subject would have been notified and the subject would have been withdrawn from the study.
Chapter 4: Results

This chapter presents findings from this descriptive, correlational, explanatory study. First, a description of subject’s demographic, and clinical characteristics, will be presented. Next preliminary analysis will be discussed followed by sleep, patient care activity, and light descriptive statistics. Hypotheses will be presented followed by findings related to each hypothesis. Finally, post hoc analysis findings will be discussed.

Subject Characteristics

Demographic Characteristics

The mean age for 63 subjects was 50.71 years ($SD = 20.28$). The majority were male ($n = 48, 76.2\%$), and Caucasian ($n = 42, 66.7\%$). A majority of subjects were single ($n = 35, 55.6\%$), and employed ($n = 32, 50.8\%$) at the time of injury (see Table 6).

Clinical Characteristics

Approximately fifty percent ($n = 32, 50.8\%$), of subjects had a subarachnoid hemorrhage. Twenty-nine subjects (46.0%) had a subdural hematoma, while 16 (25.4%) had a cerebral contusion. Twenty-six subjects had one type of brain injury, while 25 subjects had two distinct injuries and 12 subjects had three distinct injuries. Appendix K provides the mean, standard deviation, and range for the number of injuries. Twenty-one subjects (33.3%) had no comorbidities and 22 subjects (34.9%) had hypertension. A fall resulted in the TBI for approximately half of the subjects ($n = 32, 50.8\$), while 13 subject’s TBI (19.0%) was the result of a motor vehicle crash. The mean GCS score was $11.40$ ($SD = 3.34$; see Table 7) representing a moderate brain injury. Thirty-four subjects (54%) were classified as mild TBIs, fourteen (22.2%) were moderate, and 15 (23.8%) were severe.
The mean time since injury was 17.33 days ($SD = 24.44$), with a median of 11 days. Time since injury outliers ($n = 5$) ranged from 35 to 153 days. Of these five subjects, one sustained the TBI out of the country. The remaining four were initially admitted to another acute care hospital. Of the four, two were transferred to a long-term care facility prior to being admitted to

Table 6

Demographic Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>15</td>
<td>(23.8)</td>
</tr>
<tr>
<td>Male</td>
<td>48</td>
<td>(76.2)</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>42</td>
<td>(66.7)</td>
</tr>
<tr>
<td>African American</td>
<td>16</td>
<td>(25.4)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>5</td>
<td>(7.9)</td>
</tr>
<tr>
<td>Marital Status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>35</td>
<td>(55.6)</td>
</tr>
<tr>
<td>Married</td>
<td>20</td>
<td>(31.7)</td>
</tr>
<tr>
<td>Divorced</td>
<td>1</td>
<td>(1.6)</td>
</tr>
<tr>
<td>Widowed</td>
<td>2</td>
<td>(3.2)</td>
</tr>
<tr>
<td>Not documented</td>
<td>5</td>
<td>(7.9)</td>
</tr>
<tr>
<td>Employment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>32</td>
<td>(50.8)</td>
</tr>
<tr>
<td>Unemployed</td>
<td>12</td>
<td>(19.0)</td>
</tr>
<tr>
<td>Other</td>
<td>4</td>
<td>(6.3)</td>
</tr>
<tr>
<td>Not documented</td>
<td>15</td>
<td>(23.8)</td>
</tr>
</tbody>
</table>

Note. $N = 63$
Table 7

Clinical Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n</th>
<th>(%)</th>
<th>M</th>
<th>(SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary Admission Injury</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subarachnoid Hemorrhage</td>
<td>32</td>
<td>(50.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subdural Hematoma</td>
<td>29</td>
<td>(46.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebral Contusion</td>
<td>16</td>
<td>(25.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intracranial Hemorrhage</td>
<td>9</td>
<td>(14.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intra-ventricular Hemorrhage</td>
<td>8</td>
<td>(12.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diffuse Axonal Injury</td>
<td>5</td>
<td>(7.9 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epidural Hematoma</td>
<td>5</td>
<td>(7.9 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intraparenycymal Hemorrhage</td>
<td>3</td>
<td>(4.8 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Comorbidities</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>21</td>
<td>(33.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>22</td>
<td>(34.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>13</td>
<td>(20.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol/Substance Abuse</td>
<td>9</td>
<td>(14.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Orthopedic</td>
<td>9</td>
<td>(14.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAD/MI/CHF</td>
<td>8</td>
<td>(12.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurologic</td>
<td>5</td>
<td>(7.8 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancer</td>
<td>4</td>
<td>(6.3 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychiatric</td>
<td>3</td>
<td>(4.7 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>COPD</td>
<td>2</td>
<td>(3.2 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>20</td>
<td>(31.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not documented</td>
<td>2</td>
<td>(3.1 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mechanism of Injury</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fall</td>
<td>32</td>
<td>(50.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor Vehicle Crash</td>
<td>12</td>
<td>(19.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor Cycle Crash</td>
<td>5</td>
<td>(7.9 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Struck pedestrian</td>
<td>5</td>
<td>(7.9 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assault</td>
<td>3</td>
<td>(4.8 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gun Shot Wound</td>
<td>3</td>
<td>(4.8 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>3</td>
<td>(4.8 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Pain Medication Episodes</strong></td>
<td></td>
<td></td>
<td>3.47</td>
<td>(2.93)</td>
</tr>
<tr>
<td><strong>Glasgow Coma Scale Score</strong></td>
<td></td>
<td></td>
<td>11.40</td>
<td>(3.34)</td>
</tr>
</tbody>
</table>

*Note. N = 63. Some subjects had more than one primary admission injury, and comorbidity.*
MetroHealth for rehabilitation. The remaining two subjects were initially admitted to another acute care hospital then transferred to MetroHealth for rehabilitation. Both subjects were consented for the study but required readmission back to acute care unit for deterioration in their medical condition prior to the actiwatch being applied. When these subjects were readmitted to rehabilitation, they were re-consented and completed study procedures. The mean number of pain medication episodes during the study period was 3.47 (SD = 2.93). Although therapy start times varied for participants between day one and day two, therapy began at 9:00 a.m. or earlier on 52% of the days, and 58.7% (n = 37) of the subjects did not have a roommate. Constant supervision (CS) was provided for 23 subjects on both days of wearing the actiwatch, while 39 subjects did not require CS. One subject required CS on day one, but not on day two.

**Preliminary Analysis**

**Day One and Day Two Data**

Preliminary analyses were conducted to determine the strategy to analyze two days of data for independent variables (DPCAs and maximum light exposure) and dependent sleep variables (TST, WASO and SE). Within subject analysis was conducted using a general linear model to assess individual differences between day one and day two for direct patient care activities, maximum light exposure, total sleep time, SD, SE, WASO and VSH scores. Results indicated no statistically significant individual differences for direct patient care activities (p = .611), maximum light exposure, (p = .462), SD (p = .203), SE (p = .255), WASO (p = .744), and VSH scores (p = .538) therefore a decision was made to sum and average day one and day two for variables analyzed in the study. There was a statistically significant individual difference between day one and day two for total sleep time (p = .014). To address this difference, analyses for TST was conducted using two approaches. First, individual analyses were conducted using
day one and day two separately. Second, day one and day two were combined. Results will be
described in chapter 4 with research question five.

**Sleep**

In addition to the sleep variables TST, WASO, and SE, other sleep variables including
SD, daytime sleep, and subjective sleep will be included in specified analyses.

**Research Question One:** What are the sleep patterns for the quantity (TST), and quality of sleep
(WASO and SE)?

**Quantity of Sleep**

**Total sleep time and daytime sleep.** The mean TST was 539.10 minutes \((SD = 155.83)\).
Total daytime sleep quantity was 194.95 minutes \((SD = 118.32)\). On average, subjects took
frequent naps during daytime hours \((M = 4.30, SD = 1.84)\). On average naps were \(M = 45.70\)
minutes \((SD = 40.97\); see Table 8).

Table 8

**Twenty-Four Hour and Daytime Sleep**

<table>
<thead>
<tr>
<th>Variable</th>
<th>(M)</th>
<th>((SD))</th>
<th>Range</th>
<th>Median</th>
<th>Skew</th>
<th>(LL)</th>
<th>(UL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TST</td>
<td>539.10</td>
<td>(155.83)</td>
<td>242.50-946</td>
<td>507.00</td>
<td>0.63</td>
<td>499.85</td>
<td>578.34</td>
</tr>
<tr>
<td>DS</td>
<td>194.95</td>
<td>(118.32)</td>
<td>28.50-525</td>
<td>184.50</td>
<td>0.87</td>
<td>165.15</td>
<td>224.75</td>
</tr>
<tr>
<td>Nap #</td>
<td>4.30</td>
<td>(1.84)</td>
<td>1-9</td>
<td>4.00</td>
<td>0.46</td>
<td>3.84</td>
<td>4.77</td>
</tr>
<tr>
<td>ND</td>
<td>45.70</td>
<td>(40.97)</td>
<td>10-266</td>
<td>33.00</td>
<td>2.32</td>
<td>42.17</td>
<td>49.22</td>
</tr>
</tbody>
</table>

*Note. N = 63. CI = confidence interval; \(LL\) = lower limit, \(UL\) = upper limit; DS = daytime sleep;
ND = nap duration; Nap # = number of naps; TST = total sleep time. Nap duration, daytime sleep and TST sleep measured in minutes.*
Nighttime sleep. Mean nighttime SD (time between the start and end of sleep) was $M = 428.94$ minutes ($SD = 60.44$), and slightly skewed. Subjects’ mean nighttime sleep time (time scored as sleep between the start and end of nighttime sleep) was $M = 346.48$ minutes ($SD = 70.62$), thus reflecting the time subjects were awake between the start and end of sleep (see Table 9).

Table 9

**Quantity of Nighttime Sleep**

<table>
<thead>
<tr>
<th>Variable</th>
<th>$M$ ($SD$)</th>
<th>Range</th>
<th>Median</th>
<th>Skew</th>
<th>$LL$</th>
<th>$UL$</th>
</tr>
</thead>
<tbody>
<tr>
<td>SD</td>
<td>428.94 (60.44)</td>
<td>177-479</td>
<td>449.00</td>
<td>-2.25</td>
<td>413.72</td>
<td>444.17</td>
</tr>
<tr>
<td>ST</td>
<td>346.48 (70.62)</td>
<td>65.50-441</td>
<td>360.00</td>
<td>-1.35</td>
<td>328.69</td>
<td>364.26</td>
</tr>
</tbody>
</table>

*Note. N = 63. CI = confidence interval; LL = lower limit, UL = upper limit; SD = Sleep duration, ST = Sleep time. Sleep duration and sleep time measured in minutes.*

**Quality of Sleep**

Sleep efficiency was $M = 72.18\%$, ($SD = 14.71$). Overall subjects’ average time awake after sleep onset (WASO) was 82.47 minutes ($SD = 38.56$). The average number of wake bouts was 20.46 ($SD = 7.40$), and the mean time of each wake bout was 4.21 minutes ($SD = 2.08$; see Table 10).

Of note, in examining intercorrelations between sleep variables, nighttime sleep time (amount of time sleeping between the start and end of sleep), and SE were found to be perfectly correlated ($r = 1.00, p < .01$) therefore nighttime sleep time was only reported in the descriptive
statistics. Nighttime sleep time was used to calculate total sleep time (daytime sleep + nighttime sleep = total sleep time).

Table 10

Quality of Nighttime Sleep

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>(SD)</th>
<th>Range</th>
<th>Median</th>
<th>Skew</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>SE (%)</td>
<td>72.18</td>
<td>(14.71)</td>
<td>13.65-91.8</td>
<td>75.00</td>
<td>-1.35</td>
<td>68.48 - 75.89</td>
</tr>
<tr>
<td>WASO</td>
<td>82.47</td>
<td>(38.56)</td>
<td>10.50-207</td>
<td>79.00</td>
<td>0.66</td>
<td>72.76 - 92.18</td>
</tr>
<tr>
<td>Number WB</td>
<td>20.46</td>
<td>(7.40)</td>
<td>1.50-35</td>
<td>21.50</td>
<td>-.13</td>
<td>18.60 - 22.32</td>
</tr>
<tr>
<td>WB min.</td>
<td>4.21</td>
<td>(2.08)</td>
<td>1.52-12.32</td>
<td>3.92</td>
<td>1.56</td>
<td>3.69 - 4.74</td>
</tr>
</tbody>
</table>

Note. N = 63. CI = confidence interval; LL = lower limit, UL = upper limit; SE (%) = sleep efficiency percentage; WASO = wake time after sleep onset minutes; Number WB = number of wake bouts; WB min. = wake bout minutes.

Subjective Sleep: Descriptive Statistics

For the 29 subjects who were able to complete the Verran and Snyder-Halpern Sleep Scale, the mean total scale score was 415.05 (SD = 127.27). The time needed to fall asleep represented the highest mean score (M = 61.16, SD = 22.43), while fragmentation of sleep and sleep depth represented lower scores (see Table 11).
### Verran and Snyder-Halpern (VSH) Sleep Scale Scores

<table>
<thead>
<tr>
<th>Factor/Characteristic</th>
<th>M</th>
<th>(SD)</th>
<th>Range</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fragmentation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waking at Night</td>
<td>48.38</td>
<td>(29.67)</td>
<td>7-100</td>
<td>43.00</td>
</tr>
<tr>
<td>Moving at Night</td>
<td>49.91</td>
<td>(26.86)</td>
<td>3-100</td>
<td>49.00</td>
</tr>
<tr>
<td><strong>Length</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amount of Sleep</td>
<td>56.29</td>
<td>(19.10)</td>
<td></td>
<td>53.50</td>
</tr>
<tr>
<td><strong>Delay</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time to Fall Asleep</td>
<td>61.16</td>
<td>(22.43)</td>
<td>11-96</td>
<td>60.00</td>
</tr>
<tr>
<td><strong>Depth</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slept Lightly/Deeply</td>
<td>41.41</td>
<td>(28.50)</td>
<td>1-97</td>
<td>34.50</td>
</tr>
<tr>
<td>Awoke Exhausted/Refreshed</td>
<td>52.50</td>
<td>(25.94)</td>
<td>8-99</td>
<td>50.00</td>
</tr>
<tr>
<td>Awake Abrupt/Spontaneous</td>
<td>53.40</td>
<td>(28.76)</td>
<td>2-93</td>
<td>57.50</td>
</tr>
<tr>
<td>Quality of Sleep</td>
<td>52.00</td>
<td>(26.16)</td>
<td>1-96</td>
<td>52.00</td>
</tr>
<tr>
<td><strong>Total VSH Score</strong></td>
<td>415.05</td>
<td>(127.27)</td>
<td>148-658</td>
<td>410.00</td>
</tr>
</tbody>
</table>

*Note. n = 29. VSH scores reported from 29 subjects. Range of total scores for VSH Sleep Scale 0-800. Higher scores indicate better sleep.*

**Research Question Two:** What are the types and number of patient care activities that occur during nighttime?

**Patient Care Activities: Descriptive Statistics**

The number of total patient care activities occurring between 11:00 p.m. and 7:00 a.m. were summed and averaged for two nights. More indirect patient care activities occurred ($M = 18.24$, $SD = 12.14$), compared to direct care activities ($M = 5.43$, $SD = 3.25$; see Table 12). The two most common indirect patient care activities were documentation episodes in the subjects’ room, ($M = 11.52$, $SD = 14.81$), and observations ($M = 5.55$, $SD = 4.65$).
The five most common direct patient care activities were toileting, \((M = 1.38, SD = 1.09)\), vital signs, \((M = 1.10, SD = .42)\), assessment, \((M = .83, SD = 1.40)\), medications, \((M = .79, SD = .85)\), and repositioning, \((M = .38, SD = 2.10)\). The mean number of direct patient care activities

Table 12

Patient Care Interruption Data

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Sum</th>
<th>(M) (SD)</th>
<th>Range</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Indirect Patient Care Activities</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Documentation</td>
<td>1451</td>
<td>11.52 (14.81)</td>
<td>0-32</td>
</tr>
<tr>
<td>Observation</td>
<td>699</td>
<td>5.55 (4.65)</td>
<td>0-19</td>
</tr>
<tr>
<td>Care of roommate</td>
<td>129</td>
<td>1.02 (2.18)</td>
<td>0-14</td>
</tr>
<tr>
<td>Manage equipment</td>
<td>19</td>
<td>0.15 (0.42)</td>
<td>0-2</td>
</tr>
<tr>
<td>Total Indirect Patient Care Activities</td>
<td>2298</td>
<td>18.24 (12.14)</td>
<td>0-46</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Direct Patient Care Activities</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Toileting</td>
<td>174</td>
<td>1.38 (1.09)</td>
<td>0-5</td>
</tr>
<tr>
<td>Vital signs</td>
<td>139</td>
<td>1.10 (0.42)</td>
<td>0-10</td>
</tr>
<tr>
<td>Assessment</td>
<td>104</td>
<td>0.83 (1.40)</td>
<td>0-10</td>
</tr>
<tr>
<td>Medications</td>
<td>100</td>
<td>0.79 (0.85)</td>
<td>0-5</td>
</tr>
<tr>
<td>Repositioning</td>
<td>48</td>
<td>0.38 (2.10)</td>
<td>0-23</td>
</tr>
<tr>
<td>Dressing</td>
<td>22</td>
<td>0.17 (0.52)</td>
<td>0-4</td>
</tr>
<tr>
<td>Enteral feed</td>
<td>21</td>
<td>0.17 (0.52)</td>
<td>0-2</td>
</tr>
<tr>
<td>Transfers</td>
<td>15</td>
<td>0.12 (0.39)</td>
<td>0-2</td>
</tr>
<tr>
<td>Venipuncture</td>
<td>13</td>
<td>0.10 (0.33)</td>
<td>0-2</td>
</tr>
<tr>
<td>Bathing</td>
<td>5</td>
<td>0.04 (0.32)</td>
<td>0-3</td>
</tr>
<tr>
<td>Tracheostomy Care</td>
<td>5</td>
<td>0.04 (0.20)</td>
<td>0-1</td>
</tr>
<tr>
<td>Dressing Change</td>
<td>5</td>
<td>0.04 (0.23)</td>
<td>0-2</td>
</tr>
<tr>
<td>Respiratory Care</td>
<td>3</td>
<td>0.02 (0.15)</td>
<td>0-1</td>
</tr>
<tr>
<td>Suctioning</td>
<td>3</td>
<td>0.02 (0.15)</td>
<td>0-2</td>
</tr>
<tr>
<td>Restraint application/removal</td>
<td>2</td>
<td>0.02 (0.13)</td>
<td>0-1</td>
</tr>
<tr>
<td>Radiologic</td>
<td>0</td>
<td>0.00 (0.00)</td>
<td>0</td>
</tr>
<tr>
<td>Other</td>
<td>25</td>
<td>0.20 (0.55)</td>
<td>0-4</td>
</tr>
<tr>
<td>Total Direct Patient Care Activities</td>
<td>684</td>
<td>5.43 (3.25)</td>
<td>1-29</td>
</tr>
</tbody>
</table>

Note. \(N = 63\). Sum = the sum of patient care activities for two nights combined between 11:00 p.m. and 7:00 a.m.; \(M\) = the summed and averaged number of patient care activities performed for two nights between 11:00 p.m. and 7:00 a.m.
between 11:00 p.m. and 1:00 a.m. was 1.22 (SD = 1.65) compared to 2.19 (SD = 2.33) between 1:00 a.m. and 5:00 a.m. and 2.30 (SD = 1.74) between 5:00 a.m. and 7:00 a.m.

**Research Question Three:** What is the light exposure during nighttime sleep?

**Light**

The three light parameters that will be presented in the descriptive statistics include average light exposure, total light exposure, and maximum light exposure between 11:00 p.m. and 7:00 a.m., although only maximum light exposure will be reported in subsequent analyses because maximum light exposure represented the light parameter that would serve as the strongest environmental stressor for subjects. Maximum light exposure had a skew less than two reflecting the most normally distributed light variable. During nighttime hours, the mean light exposure was 1.74 lux (SD = 5.03). Total light exposure was 760.83 lux (SD = 2222.10). The maximum amount of light exposure was 38.57 lux (SD = 45.69; see Table 13).

Table 13

*Light Levels*

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>(SD)</th>
<th>Median</th>
<th>Range</th>
<th>Skew</th>
<th>LL</th>
<th>UL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average Light</td>
<td>1.74</td>
<td>(5.03)</td>
<td>0.36</td>
<td>.01-36.06</td>
<td>5.65</td>
<td>0.47</td>
<td>3.00</td>
</tr>
<tr>
<td>Total Light</td>
<td>760.83</td>
<td>(2222.10)</td>
<td>159.15</td>
<td>2.99-16110.45</td>
<td>5.78</td>
<td>201.20</td>
<td>1320.45</td>
</tr>
<tr>
<td>Maximum Light</td>
<td>38.57</td>
<td>(45.69)</td>
<td>23.66</td>
<td>.01-190.97</td>
<td>1.61</td>
<td>27.07</td>
<td>50.08</td>
</tr>
</tbody>
</table>

*Note.* CI = confidence interval; LL = lower limit, UL = upper limit. Light levels measured in lux per night.
Major Study Research Questions and Hypotheses

**Research Question Four:** What are the relationships among the number of nighttime patient care activities, nighttime exposure to light, TST, WASO and SE?

A family of hypotheses was tested to evaluate the relationship between patient care activities and sleep including:

4A. *An increase in the numbers of patient care activities at nighttime will decrease TST.*

4B. *An increase in the numbers of patient care activities at nighttime will increase WASO.*

4C. *An increase in the numbers of patient care activities at nighttime will decrease SE.*

Using Pearson’s product-moment coefficient there was a small and non-significant relationship between direct patient care activities and total sleep time ($r = -.13, p = .151$). Conversely, there was a significant moderate negative relationship between the number of direct patient care activities and sleep efficiency ($r = -.38, p < .001$), while there was a significant positive relationship between patient care activities and wake time after sleep onset ($r = .28, p < .014$). Hypothesis 4A was not supported. Hypotheses 4B and 4C were supported by the findings (see Table 14). Of note, there was a significant negative relationship between direct patient care activities and nighttime SD ($r = -.27, p = .018$).

A family of hypotheses was tested to evaluate the relationship between exposure to light and sleep including:

4D: *An increase in the amount of exposure to light at nighttime will decrease TST.*

4E: *An increase in the amount of exposure to light at nighttime will increase WASO.*

4F: *An increase in the amount of exposure to light at nighttime will decrease SE.*

Using Pearson’s product-moment coefficient there were no statistically significant relationships between maximum exposure to light between 11:00 p.m. and 7:00 a.m. and TST ($r$
There was a moderate positive relationship found between maximum exposure to light during nighttime hours and wake time after sleep onset ($r = .36, p < .002$), thus hypothesis 4E was supported. Hypotheses 4D and 4F were not supported by the findings (see Table 14).

In addition to these hypotheses, the correlation among subjective sleep with other sleep variables (TST, SD, WASO and SE), direct patient care activities, and light was explored. There was a statistically significant negative correlation between total VSH score and pain episodes ($r = -.39, p = .035$) as well as maximum light exposure ($r = -.37, p = .046$) between 11:00 p.m. and 7:00 a.m. No statistically significant correlation was found between total VSH scores and sleep variables (see Table 14). The relationship between sleep variables, and VSH subscales including fragmentation, length, delay, and depth were also explored with no significant findings.
Table 14

*Correlations of Main Study Variables*

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. TST</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. SD</td>
<td></td>
<td>.56**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. SE</td>
<td></td>
<td></td>
<td>.68**</td>
<td>.84**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. WASO</td>
<td></td>
<td></td>
<td></td>
<td>.38**</td>
<td>.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. VSH&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.09</td>
<td>.14</td>
<td>.23</td>
<td>-.19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. DPCA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.13</td>
<td>-.27*</td>
<td>-.38**</td>
<td>.28*</td>
<td>-.16</td>
</tr>
<tr>
<td>7. MaxLux</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.11</td>
<td>.14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.14</td>
<td>-.07</td>
<td>-.04</td>
</tr>
<tr>
<td>9. Pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.14</td>
<td>-.03</td>
</tr>
<tr>
<td>10. GCS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.05</td>
</tr>
</tbody>
</table>

Note. *N* = 63. <sup>a</sup>*n* = 29. TST = total sleep time; SD = sleep duration; SE = sleep efficiency; WASO = wake after sleep onset; VSH = Verran-Snyder Sleep Scale; DPCA = direct patient care activities; MaxLux = maximum lux exposure; GCS = Glasgow coma scale. *p < .05, two tailed. **p < .01, two tailed.
Research Question Five: Controlling for age, severity of injury, and pain:

What effect do patient care activities and exposure to light during nighttime hours have on TST?

5A1: More patient care activities at nighttime will decrease TST.

5A2: More exposure to light during nighttime hours will decrease TST.

Hierarchical multiple regression analyses were used to determine which independent variables (patient care activities, and light exposure) explained sleep quantity TST and sleep quality (wake time after sleep onset and SE). In each set of analysis, the covariates including age, severity of injury, and pain were entered into Block 1, direct patient care activities were entered into Block 2 and maximum light exposure was entered into Block 3.

Age, severity of injury, and pain were entered into Block 1 and did not explain any statistically significant variation in TST. After entering direct patient care activities in Block 2, the total variance in total sleep time explained by the model was 3%, $F(4, 58) = 1.48, p = .222$. After entering maximum light exposure in Block 3, total variance explained by the entire model was 3%, $F(5, 57) = 1.39, p = .242$. In the final model, neither direct patient care activities ($\beta = -.28, p = .067$), nor maximum light ($\beta = .13, p = .311$), significantly contributed to the model (see Table 15). Neither hypothesis (5A1 or 5A2) was supported by the findings from the analysis.

Since TST was different for day one and day two, a second set of regression analyses was conducted analyzing each day separately. Findings from day one were similar to findings for both days combined. Total variance explained by the entire model was 4.1%, $F(5, 57) = .50, p = .768$. In the final model, neither direct patient care activities ($\beta = -.13, p = .370$), nor maximum light ($\beta = .11, p = .933$), statistically contributed to the model (see Appendix L).
Table 15

_Hierarchical Regression Analysis for Variables Explaining Total Sleep Time_

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>1.31</td>
<td>1.11</td>
<td>.17</td>
</tr>
<tr>
<td>GCS</td>
<td>-6.97</td>
<td>6.61</td>
<td>-.15</td>
</tr>
<tr>
<td>Pain</td>
<td>-6.32</td>
<td>7.12</td>
<td>-.12</td>
</tr>
<tr>
<td><strong>Step 1</strong></td>
<td>Adjusted R^2</td>
<td>.000</td>
<td></td>
</tr>
<tr>
<td><strong>Model 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>1.88</td>
<td>1.14</td>
<td>.25</td>
</tr>
<tr>
<td>GCS</td>
<td>-12.82</td>
<td>7.39</td>
<td>-.27</td>
</tr>
<tr>
<td>Pain</td>
<td>-4.18</td>
<td>7.13</td>
<td>-.08</td>
</tr>
<tr>
<td>DPCA</td>
<td>-11.63</td>
<td>6.96</td>
<td>-.24</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td>Adjusted R^2</td>
<td>.030</td>
<td></td>
</tr>
<tr>
<td>R^2 Δ</td>
<td>.044</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Model 3</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>1.87</td>
<td>1.14</td>
<td>.25</td>
</tr>
<tr>
<td>GCS</td>
<td>-13.10</td>
<td>7.40</td>
<td>-.28</td>
</tr>
<tr>
<td>Pain</td>
<td>-2.88</td>
<td>7.24</td>
<td>-.05</td>
</tr>
<tr>
<td>DPCA</td>
<td>-13.37</td>
<td>7.17</td>
<td>-.28</td>
</tr>
<tr>
<td>MaxLux</td>
<td>.46</td>
<td>.45</td>
<td>.13</td>
</tr>
<tr>
<td><strong>Step 3</strong></td>
<td>Adjusted R^2</td>
<td>.030</td>
<td></td>
</tr>
<tr>
<td>R^2 Δ</td>
<td>.016</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. N = 63. GCS = Glasgow coma scale; DPCA = direct patient care activities; Max Lux = maximum lux exposure.*

* p < .05. ** p < .01.
Finally, a regression analysis was conducted on day two. Similar to the previous analyses, the final model did not explain total sleep time. Total variance explained by the model was 8%, $F(5, 57) = 2.08, p = .082$. One finding that was different from the previous analyses was that in the final model both age ($\beta = .30, p = .043$), and direct patient care activities ($\beta = -.35, p = .018$) significantly contributed to the model, although the model remained non-significant (see Appendix M).

**Regression Analysis for Sleep Duration**

An additional regression analysis was performed to determine if direct patient care activities and light exposure at nighttime explained nighttime SD (see Table 16). Age, severity of injury, and pain were entered into Block 1, and did not explain any statistically significant variation in SD. After entering direct patient care activities in Block 2, the total variance explained by the model was 2.2%, $F(4, 58) = 1.34, p = .265$. After entering maximum light exposure in Block 3, total variance in SD explained by the entire model was 5.6%, $F(5, 57) = 1.74, p = .141$, which was a non-significant finding. In the final model, only direct patient care activities made a statistically significant contribution to explaining SD ($\beta = -.39, p = .012$).
Table 16

Hierarchical Regression Analysis for Variables Explaining Sleep Duration

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-.28</td>
<td>.44</td>
<td>-.09</td>
</tr>
<tr>
<td>GCS</td>
<td>.59</td>
<td>2.62</td>
<td>.03</td>
</tr>
<tr>
<td>Pain</td>
<td>-.97</td>
<td>2.82</td>
<td>-.05</td>
</tr>
<tr>
<td><strong>Step 1</strong></td>
<td>Adjusted R²</td>
<td>-.043</td>
<td></td>
</tr>
<tr>
<td><strong>Model 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.02</td>
<td>.45</td>
<td>-.01</td>
</tr>
<tr>
<td>GCS</td>
<td>-2.43</td>
<td>2.88</td>
<td>-.13</td>
</tr>
<tr>
<td>Pain</td>
<td>.14</td>
<td>2.78</td>
<td>.01</td>
</tr>
<tr>
<td>DPCA</td>
<td>-6.01</td>
<td>2.71</td>
<td>-.32*</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td>Adjusted R²</td>
<td>.022</td>
<td>R² Δ .077*</td>
</tr>
<tr>
<td><strong>Model 3</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.01</td>
<td>.44</td>
<td>.01</td>
</tr>
<tr>
<td>GCS</td>
<td>-2.63</td>
<td>2.83</td>
<td>-.14</td>
</tr>
<tr>
<td>Pain</td>
<td>.10</td>
<td>2.77</td>
<td>.05</td>
</tr>
<tr>
<td>DPCA</td>
<td>-7.16</td>
<td>2.74</td>
<td>-.39**</td>
</tr>
<tr>
<td>MaxLux</td>
<td>.30</td>
<td>.17</td>
<td>.23</td>
</tr>
<tr>
<td><strong>Step 3</strong></td>
<td>Adjusted R²</td>
<td>.056</td>
<td>R² Δ .047</td>
</tr>
</tbody>
</table>

*Note. N = 63. GCS = Glasgow coma scale; DPCA = direct patient care activities; Max Lux = maximum lux exposure. * p < .05. ** p < .01.

5B. Controlling for age, severity of injury and pain:

What effect do patient care activities and exposure to light during nighttime hours have on WASO?

5B1: More patient care activities at nighttime will increase WASO.
5B2: *More exposure to light during nighttime hours will increase WASO.*

Age, severity of injury, and pain were entered into Block 1 and did not explain any statistically significant variance in WASO. After entering direct patient care activities in Block 2, the total variance in WASO explained by the model was 2%, $F(4, 58) = 1.31, p = .276$, which was a non-significant finding. After entering maximum light exposure in Block 3, total variance explained by the entire model was 10.6%, $F(5, 57) = 2.47, p = .043$, which was significant although only maximum light made a significant contribution to explaining WASO ($\beta = .32, p = .013$). In Step 2 of the model, direct patient care activities made a statistically significant contribution to explaining WASO ($\beta = .32, p = .034$), although in the final model (Step 3), when maximum light exposure was added, direct patient care activities no longer made a significant contribution in explaining WASO ($\beta = .23, p = .116$). Maximum light exposure did make a statistically significant contribution to explaining WASO ($\beta = .32, p = .013$), thus hypothesis 5B2 was supported. Hypothesis 5B1 was not supported based on the final model (see Table 17).
Table 17

Hierarchical Regression Analysis for Variables Explaining WASO

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.02</td>
<td>.28</td>
<td>.01</td>
</tr>
<tr>
<td>GCS</td>
<td>-1.11</td>
<td>1.67</td>
<td>-.10</td>
</tr>
<tr>
<td>Pain</td>
<td>.14</td>
<td>1.80</td>
<td>.01</td>
</tr>
</tbody>
</table>

Step 1
Adjust R²   - .042

| **Model 2** |    |     |     |
| Age        | -.16| .28  | -.09|
| GCS        | .78 | 1.84 | .07 |
| Pain       | -.55| 1.77 | -.04|
| DPCA       | 3.75| 1.73 | .32*|

Step 2
Adjust R²   .020
R² Δ  .074*

| **Model 3** |    |     |     |
| Age        | -.17| .27  | -.09|
| GCS        | .61 | 1.76 | .05 |
| Pain       | .23 | 1.72 | .02 |
| DPCA       | 2.72| 1.70 | .23 |
| MaxLux     | .27 | .11  | .32*|

Step 3
Adjust R²   .106*
R² Δ  .095**

Note. N = 63. GCS = Glasgow coma scale; DPCA = direct patient care activities; Max Lux = maximum lux exposure.
* p < .05. ** p < .01.

5C. Controlling for age, severity of injury and pain:

What effect do patient care activities and exposure to light during nighttime hours have on SE?

5C1: More patient care activities at nighttime will decrease SE.

5C2: More exposure to light during nighttime hours will decrease SE.
Age, severity of injury, and pain were entered into Block 1 and did not explain and statistically significant variation in SE. After entering direct patient care activities in Block 2, the total variance in SE explained by the model was 10.1%, $F(4, 58) = 2.74, p = .037$. After entering maximum light in Block 3, total variance explained by the entire model was 8.6%, $F(5, 57) = 2.16, p = .071$. In the final model, only direct patient care activities made a statistically significant contribution to explaining SE ($\beta = -.45, p = .003$). Hypothesis 5C1 was supported, while hypothesis 5C2 was not supported by the findings (see Table 18).
Table 18

*Hierarchical Regression Analysis for Variables Explaining Sleep Efficiency*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-.06</td>
<td>.11</td>
<td>-.09</td>
</tr>
<tr>
<td>GCS</td>
<td>.35</td>
<td>.64</td>
<td>-.08</td>
</tr>
<tr>
<td>Pain</td>
<td>-.23</td>
<td>.69</td>
<td>-.05</td>
</tr>
<tr>
<td><strong>Step 1</strong></td>
<td>Adjusted R²</td>
<td>-.041</td>
<td></td>
</tr>
<tr>
<td><strong>Model 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.04</td>
<td>.10</td>
<td>.05</td>
</tr>
<tr>
<td>GCS</td>
<td>-.67</td>
<td>.67</td>
<td>-.15</td>
</tr>
<tr>
<td>Pain</td>
<td>.14</td>
<td>.65</td>
<td>.03</td>
</tr>
<tr>
<td>DPCA</td>
<td>-2.03</td>
<td>.63</td>
<td>-.45**</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td>Adjusted R²</td>
<td>.101*</td>
<td>R² Δ</td>
</tr>
<tr>
<td><strong>Model 3</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.04</td>
<td>.11</td>
<td>.05</td>
</tr>
<tr>
<td>GCS</td>
<td>-.67</td>
<td>.68</td>
<td>-.15</td>
</tr>
<tr>
<td>Pain</td>
<td>.16</td>
<td>.66</td>
<td>.03</td>
</tr>
<tr>
<td>DPCA</td>
<td>-2.06</td>
<td>.66</td>
<td>-.45**</td>
</tr>
<tr>
<td>MaxLux</td>
<td>.01</td>
<td>.04</td>
<td>.02</td>
</tr>
<tr>
<td><strong>Step 3</strong></td>
<td>Adjusted R²</td>
<td>.086</td>
<td>R² Δ</td>
</tr>
</tbody>
</table>

*Note. N = 63. GCS = Glasgow coma scale; DPCA = direct patient care activities; MaxLux = maximum lux exposure.*

* p < .05. ** p < .01.
Post Hoc Analysis

To further explore findings from this study, additional post hoc analyses were performed.

Comparison Between Constant Supervision Groups

To explore differences between subjects with and without constant supervision, a Mann-Whitney U Test was conducted. The Mann-Whitney U Test was conducted since the Kolmogorov-Smirnov test of univariate normality indicated non-normal distributions for age ($p = .011$), GCS ($p < .0001$), pain ($p < .0001$), maximum lux ($p < .0001$), SD ($p < .0001$), SE ($p = .036$), and direct patient care activities ($p < .0001$). Sixty-two subjects were included in the analysis. One subject was excluded since the subject was on constant supervision on day one and off constant supervision on day two. Findings indicated significant differences between groups on direct patient care activities ($p = .012$) and severity of injury ($p = .009$; see Table 19).
Table 19

*Mann-Whitney U Test Comparing Subjects With and Without Constant Supervision*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Constant Supervision(^a)</th>
<th>No Constant Supervision(^b)</th>
<th>Z</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>54.00</td>
<td>56.00</td>
<td>-.10</td>
<td>.925</td>
</tr>
<tr>
<td>GCS</td>
<td>11.39</td>
<td>13.00</td>
<td>-1.65</td>
<td>.009**</td>
</tr>
<tr>
<td>Pain</td>
<td>1.50</td>
<td>3.00</td>
<td>-1.29</td>
<td>.196</td>
</tr>
<tr>
<td>Max Lux</td>
<td>26.28</td>
<td>21.23</td>
<td>-1.42</td>
<td>.155</td>
</tr>
<tr>
<td>DPCA</td>
<td>6.00</td>
<td>4.50</td>
<td>-2.51</td>
<td>.012*</td>
</tr>
<tr>
<td>TST</td>
<td>507.00</td>
<td>521.00</td>
<td>-.49</td>
<td>.625</td>
</tr>
<tr>
<td>SD</td>
<td>447.00</td>
<td>454.50</td>
<td>-1.56</td>
<td>.119</td>
</tr>
<tr>
<td>SE</td>
<td>72.50</td>
<td>76.46</td>
<td>-1.57</td>
<td>.116</td>
</tr>
<tr>
<td>WASO</td>
<td>83.00</td>
<td>74.50</td>
<td>-.93</td>
<td>.351</td>
</tr>
</tbody>
</table>

*Note.* Constant Sup = Constant Supervision; GCS = Glasgow coma scale; MaxLux = maximum light exposure; TST = total sleep time; SD = sleep duration; SE = sleep efficiency; WASO = wake time after sleep onset; DPCA = direct patient care activities. 

\(^a\)n = 23. \(^b\)n = 39.

\(* p < .05, \** p < .01, two tailed.\)*

**Light Data Analysis**

To better understand the relationship between light data and sleep variables, additional post hoc analyses were conducted. Concerns regarding systematic error were identified based on only 63.9% of staff recording that the light monitor (actiwatch) was visible during rounding. In addition, 16 subjects had maximum light levels of .01 lux. Since all subjects had at least one
direct patient care activity performed during nighttime hours further investigation was needed to
determine whether light levels were valid or reflected systematic error.

Multiple strategies were performed to further investigate the validity of the light data. A
frequency distribution analysis was conducted to examine the frequency of subjects with
maximum lux levels of less than 5 lux. This lux level was determined based on an assessment of
the frequency data and levels of 5 lux indicating very low levels of light. Results identified 16
subjects (25.4%) with maximum lux levels of less than 5 lux during nighttime hours (11:00 p.m.
and 7:00 a.m.). Of those 16 subjects, 13 had two or more direct patient care activities performed
during nighttime hours causing concern regarding validity of the light data.

Next, results of reliability testing were checked to determine if any subjects with
maximum light levels of less than 5 lux were included in the reliability testing procedures
comparing the actiwatch on the wrist and the actiwatch on the headboard. Of the 16 subjects with
maximum lux levels less than 5 lux, reliability testing had been conducted on two of the subjects.
For these two subjects the mean number of DPCAs that occurred during the night was one and
1.5 possibly suggesting that low light levels may be plausible depending on the type of activity
and whether the light was turned on. To further assess the possibility of measurement error,
serial numbers for the actiwatches were checked for the 16 subjects with low maximum lux
levels to determine if there was a malfunction of one of the watches. Data demonstrated that all
three actiwatches were used for study procedures (25%, 37.5%, and 35%) suggesting no single
watch was responsible for low light levels.

To further understand the light data, results of the initial regression analyses were
inspected identifying two unexpected findings. In the regression analysis examining the effects
of DPCAs and maximum light exposure on WASO, the Beta for DPCAs was found to be
significant in Step 2 \((p = .034)\), but became non-significant \((p = .116)\) when maximum lux was added to the model. In addition, in the regression analysis conducted to explain the effects of DPCA and maximum light on SD and SE, in the final step of the model the Beta for maximum light exposure was positive, when theoretically it was expected to be negative.

To further explore these unexpected findings a trimmed model \((n = 47)\) excluding subjects with maximum light levels below 5 lux was created. This trimmed model excluded the covariates of age, pain, and severity of injury. The covariates were eliminated from the model for several reasons. First, age, pain, and severity of injury were not correlated with total sleep time, SD, SE, or WASO. Additionally, these three covariates were not contributing to explanatory power of the model thus decreasing power by using error degrees of freedom.

Similar to the full model, DPCAs and maximum light exposure did not explain 24-hour sleep time, \(F(2, 44) = 1.09, p = .344\), adjusted \(R^2 = .004\). No significant Betas were found in this model. Results from the trimmed model were similar to the full model \((n = 63)\) including covariates of age, pain, and severity of injury (see Table 20).
### Table 20

**Trimmed Model: Regression Analysis for Variables Explaining Total Sleep Time**

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPCA</td>
<td>-10.80</td>
<td>7.32</td>
<td>-.22</td>
</tr>
<tr>
<td>Step 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted R(^2)</td>
<td>-.025</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPCA</td>
<td>-10.47</td>
<td>7.53</td>
<td>-.21</td>
</tr>
<tr>
<td>MaxLux</td>
<td>-0.12</td>
<td>.52</td>
<td>-.04</td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted R(^2)</td>
<td>.004</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R(^2) Δ</td>
<td>.001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. N = 47. DPCA = direct care activities; MaxLux = maximum lux exposure.  
* p < .05. ** p < .01.*

In the trimmed model, DPCAs and maximum light exposure did not explain SD, \( F(2, 44) = 2.34, p = .108 \), adjusted \( R^2 = .055 \). A significant Beta for DPCAs was found in the trimmed model (\( β = -.32, p = .037 \)). These finding are similar to the full model (\( n = 63 \)). An unexpected and consistent finding with the full model was a positive Beta for maximum light exposure when theoretically it should have been negative, further supporting the possibility of error in the light data (see Table 21).
Table 21

Trimmed Model: Regression Analysis for Variables Explaining Sleep Duration

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPCA</td>
<td>-4.61</td>
<td>2.20</td>
<td>-.30*</td>
</tr>
<tr>
<td>Step 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted R²</td>
<td></td>
<td></td>
<td>.069*</td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPCA</td>
<td>-4.86</td>
<td>2.25</td>
<td>-.32*</td>
</tr>
<tr>
<td>Max Lux</td>
<td>.09</td>
<td>.16</td>
<td>.09</td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted R²</td>
<td></td>
<td></td>
<td>.055</td>
</tr>
<tr>
<td>R² Δ</td>
<td></td>
<td></td>
<td>.007</td>
</tr>
</tbody>
</table>

*Note. N = 47. GCS = DPCA = direct care activities; MaxLux = maximum lux exposure. * p < .05. ** p < .01.

Direct patient care activities and maximum light exposure explained 16.3% of WASO, $F(2, 44) = 5.48, p = .007$, adjusted $R^2 .163$ which was significant although only maximum light made a significant contribution to explaining WASO ($β = .39, p = .007$). These findings are similar to the full model ($n = 63$). In contrast to the full model where the Beta was significant in Step 2 but became non-significant in Step 3, the Beta for DPCA remained non-significant in both steps of the trimmed model (see Table 22).
Table 22

Trimmed Model: Regression Analysis for Variables Explaining WASO

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPCA</td>
<td>2.87</td>
<td>1.78</td>
<td>.23</td>
</tr>
<tr>
<td>Step 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted R²</td>
<td>.034</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPCA</td>
<td>1.98</td>
<td>1.69</td>
<td>.16</td>
</tr>
<tr>
<td>MaxLux</td>
<td>.33</td>
<td>.12</td>
<td>.39**</td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted R²</td>
<td>.163**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R² Δ</td>
<td>.145**</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. N = 47. GCS = DPCA = direct care activities; MaxLux = maximum lux exposure. * p < .05. ** p < .01.

Lastly, unlike the full model, in the trimmed model, direct patient care activities and maximum light exposure explained 14.4% of SE, $F(2, 44) = 4.86, p = .012$, adjusted $R^2 .144$. A significant Beta was found for direct patient care activities ($\beta = -.354, p = .014$), which is consistent with the full model. In addition, in the trimmed model, the Beta for maximum light exposure became negative which is consistent with what was theoretically expected, although the Beta remained non-significant similar to the full model (see Table 23).
Table 23

Trimmed Model: Regression Analysis for Variables Explaining Sleep Efficiency

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPCA</td>
<td>-1.56</td>
<td>.55</td>
<td>-.39**</td>
</tr>
</tbody>
</table>

Step 1  
Adjusted R²  
.131**

Model 2  

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>DPCA</td>
<td>-1.42</td>
<td>.56</td>
<td>-.35**</td>
</tr>
<tr>
<td>MaxLux</td>
<td>-.05</td>
<td>.04</td>
<td>-.18</td>
</tr>
</tbody>
</table>

Step 2  
Adjusted R²  
.144**
R² Δ  
.031

Note. N = 47. GCS = DPCA = direct care activities; MaxLux = maximum lux exposure.  
* p < .05. ** p < .01.
Table 24

Regression Analysis for Variables Explaining Sleep: Trimmed and Full Models

<table>
<thead>
<tr>
<th>Predictor</th>
<th>TST β</th>
<th>SD β</th>
<th>WASO β</th>
<th>SE β</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Full(^a) Trimmed(^b)</td>
<td>Full(^a) Trimmed(^b)</td>
<td>Full(^a) Trimmed(^b)</td>
<td>Full(^a) Trimmed(^b)</td>
</tr>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Covariates</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPCA</td>
<td>-.24</td>
<td>-.22</td>
<td>-.32*</td>
<td>-.30*</td>
</tr>
<tr>
<td>Step 2</td>
<td>Adjusted R(^2)</td>
<td>.030</td>
<td>.025</td>
<td>.022</td>
</tr>
<tr>
<td>Model 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPCA</td>
<td>-.28</td>
<td>-.21</td>
<td>-.39**</td>
<td>-.32*</td>
</tr>
<tr>
<td>MaxLux</td>
<td>.13</td>
<td>-.04</td>
<td>.23</td>
<td>.09</td>
</tr>
<tr>
<td>Step 3</td>
<td>Adjusted R(^2)</td>
<td>.030</td>
<td>.004</td>
<td>.056</td>
</tr>
</tbody>
</table>

Note. TST = total sleep time; SD = sleep duration; WASO = wake after sleep onset; SE = sleep efficiency; DPCA = direct patient care activities; MaxLux = maximum lux exposure.

\(^a\)n = 63. \(^b\)n = 47

\(^*\)p < .05. \(^**\)p < .01
Comparison Between Subjects With and Without a Roommate

To explore differences between subjects with and without a roommate, a Mann-Whitney U Test was conducted. Fifty-nine subjects were included in the analysis. Four subjects were excluded since the subjects had a roommate one of the days and did not have a roommate the other day. Findings indicated significant differences between groups on severity of injury ($p = .042; p = .009$; see Table 25).

Table 25

*Mann-Whitney U Test Comparing Subjects With and Without Roommate*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Roommate$^a$</th>
<th>No Roommate$^b$</th>
<th>Z</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>53.50</td>
<td>56.00</td>
<td>-.66</td>
<td>.510</td>
</tr>
<tr>
<td>GCS</td>
<td>11.70</td>
<td>13.00</td>
<td>-2.04</td>
<td>.042*</td>
</tr>
<tr>
<td>Pain</td>
<td>1.50</td>
<td>3.00</td>
<td>-1.89</td>
<td>.059</td>
</tr>
<tr>
<td>DPCA</td>
<td>5.00</td>
<td>5.00</td>
<td>-1.12</td>
<td>.264</td>
</tr>
<tr>
<td>Max Lux</td>
<td>17.04</td>
<td>24.68</td>
<td>-.25</td>
<td>.802</td>
</tr>
<tr>
<td>TST</td>
<td>507.00</td>
<td>521.00</td>
<td>-.16</td>
<td>.875</td>
</tr>
<tr>
<td>SD</td>
<td>437.00</td>
<td>453.00</td>
<td>-1.84</td>
<td>.067</td>
</tr>
<tr>
<td>WASO</td>
<td>82.50</td>
<td>76.50</td>
<td>-.24</td>
<td>.808</td>
</tr>
<tr>
<td>SE</td>
<td>72.87</td>
<td>76.67</td>
<td>-1.47</td>
<td>.143</td>
</tr>
</tbody>
</table>

Note. GCS = Glasgow coma scale; MaxLux = maximum light exposure; TST = total sleep time; SD = sleep duration; SE = sleep efficiency; WASO = wake time after sleep onset; DPCA = direct patient care activities. 

$N = 59$. $^an = 22$. $^bn = 37$. 

* $p < .05$, two tailed. ** $p < .01$, two tailed.
Chapter 5. Discussion

The purposes of this study were to: (a) describe the sleep patterns of adults with traumatic brain injury in the acute rehabilitation setting; and (b) examine the effects of environmental stressors including exposure to light and patient care activities on patterns of sleep. Pearlin’s stress process model was used to guide the study. Using a descriptive, correlational, explanatory design, 64 subjects were recruited from an acute rehabilitation unit, although 63 subjects were used in the final analysis. Actigraphy was used to collect sleep and light data and an investigator developed PCESL was used to collect patient care activity data. This study adds to the body of knowledge regarding how the hospital environment affects both quantity and quality of sleep as well as provides a basis for future studies for examining the necessity, frequency, and timing of direct care activities and associated light exposure that can subsequently interfere with the recovery process.

Summary of Findings

Demographic

A majority of subjects in the sample were male (76.2%), Caucasian (66.7%), single (55.6%), and employed (50.8%) at the time of injury. A majority of subjects sustained the TBI from a fall (50.8%) or motor vehicle crash (19.0%), and the average time since injury was 17.33 days. Hypertension (34.9%), and diabetes (20.3%) were the most common comorbidities, although 33.3% of subjects had no comorbidities documented in the electronic medical record. The average GCS score was 11.40, representing moderate brain injury. The mean number of pain medication episodes administered over the 48 hours the actiwatch was worn was 3.47 episodes. Constant supervision was not required for 39 subjects (62.7% of days), and therapy sessions began at 9:00 a.m. or earlier 52% of the days the actiwatch was worn.
Sleep

Sleep was measured both objectively using an actiwatch, and subjectively using the Verran Snyder-Halpern Sleep Scale. Sleep patterns for TBI subjects in this sample were characterized by decreased SE, and increased WASO compared to recommendations by the NSF (Ohayon et al., 2017). Total sleep time was within recommendations by the NSF, although daytime sleep time was high and subjects took frequent naps. In general, subjects perceived their sleep to be average.

Numerous studies have investigated sleep patterns in traumatic brain injured persons although many have been in the community setting (Imbach et al., 2015, Shekleton et al., 2010) and many have used subjective measures (Gardani et al., 2015; Kempf et al., 2010) or non-standardized tools to measure sleep (Makley et al., 2008). Few studies have investigated sleep patterns of persons with TBI in the acute rehabilitation setting and only two studies by Makley et al. (2009) and Towns et al. (2016) used actigraphy as an objective measurement of sleep. TBI subjects in this sample had sleep patterns similar to findings from other studies (Shekleton et al., 2010; Sommerauer et al., 2013; Nakase-Richardson et al., 2013).

Sleep Quantity

In the current study, the quantity of TST was high with a mean TST of 539.10 minutes or approximately nine hours. This is similar to findings by Chiu et al. (2013) who also found mean TST to be 703.3 minutes in a sample of TBI subjects in an acute care neurosurgical ward. Imbach et al. (2015) investigated TBI subjects 6 months post injury and found TST ($M = 8.3$ hours) to be longer compared to healthy age matched controls ($M = 7.1$ hours). These findings suggest that persons with TBI require an increase in sleep time after the brain injury that continues into the community setting.
In the current study, nighttime SD was 428.94 minutes or approximately seven hours. Since nighttime sleep was analyzed based on sleep time during the defined 8-hour period, these findings may have been different if the start of sleep was prior to 11:00 p.m. or extended beyond 7:00 a.m. Although the 8-hour time frame was chosen to identify the effects of environmental stressors during nighttime hours, and to what extent the stressors affected sleep, this pre-determined time frame may have affected the findings of actual nighttime sleep by counting late evening or early morning sleep as daytime sleep.

Nighttime sleep time or the number of minutes scored as sleep between the start and end of sleep was 346.48 minutes or approximately five hours and 45 minutes. These findings are similar to a study by Towns et al. (2016) who measured sleep among TBI subjects (mean time since injury 85 days) in an inpatient rehabilitation unit and reported that the mean nighttime sleep time for 8 hours was 380 minutes measured by actigraphy. Chiu et al. (2013) also investigated TBI subjects in an acute neurosurgical unit and found a mean nighttime sleep of 396.1 minutes. Although sleep time was similar in all three studies, subjects’ nighttime sleep time in the current study was 34-50 minutes shorter. The reasons for these findings are unclear. One explanation may be related to the method of measuring sleep. In the study by Chiu et al. (2013), bedtime and wake time were recorded by primary caregivers or subjects, while this study used pre-determined sleep start and end times (11:00 p.m. and 7:00 a.m.) since asking staff to record bedtimes and wake times could be unreliable and cause burden to staff. In addition, subjects may not have had the cognitive ability to accurately record or use the actiwatch to set sleep and wake times. Additionally, the previous studies by Towns et al. and Chiu et al. did not investigate factors affecting sleep patterns, which may have influenced nighttime sleep.
Regardless, findings in the literature are consistent in reporting that TBI subject in the hospital setting sleep less during nighttime hours with the potential to impact recovery.

Although TST time was high, approximately 36% of the time spent sleeping was during daytime hours. On average, subjects slept 194.94 minutes during the daytime hours (7:01 a.m. to 10:59 p.m.) or 3 hours and 15 minutes. On average subjects took 4.3 naps each day with naps lasting approximately 45 minutes. The NSF (Ohayon et al., 2017) suggests that across all age groups taking four or more naps in a 24-hour period indicates ‘not good sleep quality’. Based on these recommendations by the NSF, subjects did not have good quality sleep. Since the NSF provides recommendations for quantity and quality of sleep across age groups, but does not specify sleep needs for healthy persons versus those recovering from illness or injury, it is unclear whether these recommendations are appropriate for persons with TBI in the early stages of recovery. Interpretation of these findings leads to more questions regarding potential causes of the need for increased daytime sleep. It is unclear whether daytime naps were a result of poor sleep quality during nighttime hours, or if 3 hours of therapy each weekday contributed to the increased need for naps. In addition, physiological changes that occur as a result of TBI that can affect the sleep wake centers of the brain (Baumann et al., 2009) cannot be discounted as potential causes of increased daytime sleep.

Results indicating increased daytime sleep in the current study are consistent with studies done in the community setting. Baumann et al. (2007) reported that 28% of subjects 6 months post TBI reported excessive daytime sleepiness, while Kempf et al. (2010) reported 12% of subjects complained of excessive daytime sleepiness 3-years post TBI. Other studies have found that in addition to complaints of daytime sleepiness, subjects also report complaints of fatigue. These findings can have a significant impact on subjects who are in the rehabilitation phase of
recovery due to the intense physical and cognitive demands are placed on subjects as a result of therapy.

**Sleep Quality**

Subjects also had poor quality of sleep. Sleep efficiency percentage was low, \( M = 72.18\% \) and WASO was high \( M = 82.47 \) minutes. The numbers of wake bouts, and the average time for each wake bout was collected to better understand sleep patterns during nighttime hours. On average, subjects experienced 20.46 wake bouts during the night lasting 4.21 minutes. Persons with TBI who are disrupted frequently throughout the night, or spontaneously wake on their own may not be completing full sleep cycles which affects the amount of time spent during SWS and REM sleep, both important for memory consolidation, new learning, rest, and recovery (Peigneux & Smith, 2011; Scharf et al., 2008). A similar study by Bloomfield et al. (2010) also found that TBI subjects with “poor sleep” measured by a combination of sleep assessment tools (PSQI, Insomnia Severity Scores, and actigraphy), had significantly poorer sustained attention than those subjects with “good sleep”.

There are numerous studies that support these findings both in the hospital (Chen et al., 2015; Chiu et al., 2013), and community settings (Kaufman et al., 2001; Makley et al., 2009; Shekleton et al., 2010). Chen et al. (2015) found similar results among TBI subjects 1-month post TBI, with mean SE scores of 69.77%. Chiu et al. (2013) also found both SE scores to be low \( M = 68.7\% \), and WASO to be high \( M = 174 \) minutes in TBI subjects from three neurosurgical wards. Using polysomnography to measure sleep, Shekleton et al. (2010) found SE for TBI subjects at least 6-months post TBI to be lower than age and gender matched controls (82.15%, 89.71%, respectively). Although findings from the study were not significant, subjects with TBI showed a trend toward less REM sleep \( p = .75 \) suggesting that TBI subjects who have low SE
and high WASO representing fragmented sleep, are not getting enough restorative sleep time which could lead to less than optimal functional and cognitive outcomes during inpatient rehabilitation. Restorative sleep is necessary to allow persons with TBI to function their highest level to facilitate rehabilitation efforts.

**Perception of Sleep**

The mean total VSH score was 415.05 with a possible range of 0-800. Although sleep complaints may be underestimated based on the ability to accurately report sleep disturbances (Ohayon et al., 2017), subjects who completed the VSH Sleep Scale did report sleep complaints. According to the NSF, one limitation of self-report instruments in measuring sleep is the loss of consciousness that occurs during sleep, making it difficult for individuals to be accurate reporters of sleep patterns (Ohayon et al., 2017). Although subjects who did not have the capacity to consent were excluded from completing the VSH Sleep Scale, other cognitive deficits that do not affect capacity for decision-making such as memory, may have altered subjects’ ability to accurately report perceptions of sleep.

Sleep data based on actigraphy indicated a decrease in SE and increase in WASO; therefore, it was not surprising that the lowest perceived scores reported by subjects on the VSH scale by subjects were sleep fragmentation and sleep depth. The self-report perception of sleep fragmentation and sleep depth were not significantly related to objective measures of sleep. However, if subjects perceive sleep to be less than optimal, it is likely they are not functioning both physically or cognitively at their highest level.

Although sleep onset latency, (amount of time it takes to fall asleep) was not measured because of subjects’ potential cognitive limitations, TBI subjects scored highest on the subscale of ‘sleep delay’ ($M = 61.6$), indicating that patients perceived that falling asleep was less
disruptive than fragmentation, length, or depth of sleep. Findings may suggest that subjects were
tired and did not have difficulty initiating sleep, although they had trouble staying asleep at least
partially as a result of frequent awakenings from DPCAs and light exposure. Total VSH scores
had a significant negative relationship with maximum light exposure ($r = -0.38, p = 0.046$), and pain
($r = -0.39, p = 0.035$), also suggesting that these factors were associated with decreased perceptions
of sleep.

Study results related to sleep initiation are not consistent with the literature. Both
Fichtenberg et al. (2002), and Clinchot et al. (1998) have reported complaints of sleep initiation
by persons with TBI. Inconsistent findings regarding sleep initiation could be related to the time
since injury (TSI). For both of these studies TSI was between 3.8 months and 1-year in these two
community-dwelling samples. Another interesting finding from the current study was that the
length or amount of sleep was perceived as second least disruptive to TBI subjects indicating
subjects felt they had sufficient sleep. These subjective findings are consistent with objective
measures of 24-hours sleep, which were within recommendations according to the NSF. Studies
of persons with TBI immediately post injury (Fichtenberg et al., 2002), and in the community
setting (Clinchot et al., 1998; Kempf et al., 2010) have also reported findings similar to the
current study. Commonly reported sleep disturbances include maintaining sleep (Clinchot et al.,
1998; Fichtenberg et al., 2002), and overall poor sleep quality (Fichtenberg et al., 2002; Gardani
et al., 2015; Shekleton et al., 2010).

Summary

A summary of findings from this study suggest that although 24-hours total sleep fell
within recommendations based on the NSF (Hirshkowitz et al., 2015), the need for additional
sleep was reflected in the amount of sleep experienced by persons with TBI during daytime
hours. Data demonstrated that sleep is fragmented based on the amount of time subjects were awake during nighttime hours and low SE scores. Although correlations between VSH Sleep Scale scores and sleep variables (TST, SD, WASO and SE) were not significant, subjects did report lowest scores for sleep fragmentation and sleep depth that were possibly related to interruptions during nighttime hours. Findings are consistent with a study by Redeker et al. (2000), who found that 48% of post-operative cardiac subjects reported interventions performed by health care providers as the most disruptive to sleep.

Both objective and subjective measurements of sleep reported in the literature indicate that sleep disturbance is prevalent among persons with TBI. Screening and assessment of sleep disturbance is essential for all persons recovering from illness or injury, but may be even more important for persons in the rehabilitation phase of recovery. Rehabilitation encompasses intense therapy sessions that increase both physical and cognitive demands on TBI patients. Accurate identification of sleep disturbance allows healthcare providers to implement strategies to improve sleep, thereby promoting improved outcomes in the hospital and community setting.

**Patient Care Activities**

Both direct and indirect patient care activities occurred during nighttime hours with indirect patient care activities occurring nearly 3.5 times more often (n = 2298) than direct care activities (n = 684). The average number of indirect patient care activities was 18.24 activities with a majority being documentation (M = 11.52) and observation (M = 5.55). The number of documentation episodes was likely related to approximately 1/3 of subjects (37%) needing constant supervision, which requires documentation of patient behavior and activity to be completed in the subject’s room. Of those subjects not requiring constant supervision, hourly
rounding is a routine standard of care practice thus reflecting the increased number of observation episodes.

The average number of DPCAs performed was 5.43 activities per subject per night. Similar to a study by Le et al. (2012), patient care activity data was recorded by nursing staff rather than by direct observation suggesting that results could have been overestimated or underestimated which could have biased results. This possible limitation reported by Le et al. was not substantiated in this study. Reliability checks between the PCESL and electronic documentation in this study were found to be between 94% and 100% agreement.

The frequency of interruptions in this study was less than those observed in intensive care unit settings (Celik et al., 2005; Tamburri et al., 2004) where patients mean nighttime patient care activities were 51 and 43, respectively and almost 10 times as frequent as in the rehabilitation setting. This discrepancy is likely related to differences in the medical acuity of patients in the intensive care units and the need for additional patient care activities during nighttime hours. Despite this, DPCAs occurred throughout the night in the rehabilitation setting with 2.30 (SD = 1.74) DPCAs performed between 5:00 a.m. and 7:00 a.m., compared to 1.22, (SD = 1.65) between 11:00 p.m. and 1:00 a.m., and 2.19 (SD = 2.33) between 1:00 a.m. and 5:00 a.m. Celik et al. (2005) also conducted a retrospective study investigating the frequency and timing of patient care activities in the ICU setting. Unlike this study where DPCAs were more frequently performed between 5:00 a.m. and 7:00 a.m., in the ICU setting patient care activities were more frequently performed between 12:00 a.m. and 5:00 a.m. (Celik et al., 2005). Since REM sleep increases with each cycle of sleep throughout the night (Carskadon & Dement, 2011), the longest time spent in REM sleep would occur in the early morning hours when
frequent DPCAs are occurring in the rehabilitation setting further highlighting the need to manage the timing of patient care activities.

Although the average number of direct patient care activities in the rehabilitation setting was low compared to acute care settings, the number of wake bouts \((M = 20.46)\) and the average time of each wake bout \((M = 4.21 \text{ minutes})\) was high. This suggests that subjects were awake frequently during nighttime hours, which could contribute to interruptions in sleep cycles. Given that the mean nighttime SD was 428.94 minutes, and the average number of wake bouts was 20.46 per night, this suggests that subject’s sleep was disturbed approximately every 20 minutes with interruptions at least partially responsible for contributing to fragmentation of sleep.

Changes in sleep architecture can reduce the amount of time spent in stages of sleep that are believed to be responsible for memory consolidation, brain plasticity and new learning (Peigneux & Smith, 2011). Since on average, subject’s sleep is disrupted every 20 minutes, further questions arise as to how fragmented sleep in this study impacts time spent in restorative sleep, which could impact the ability for TBI subjects to function at optimal levels. Health care providers need to be aware of the frequency, timing, and necessity of interruptions during nighttime hours and how these interruptions serve as stressors that can affect sleep quantity and quality.

**Light**

Overall, light levels were low during nighttime hours with total light exposure \(M = 760.83 \text{ lux}\), mean light exposure 1.74 lux, and maximum light exposure, \(M = 38.57 \text{ lux}\). As a measure of comparison, light levels of 100 lux are comparable to light levels during an overcast day while 10 lux is comparable to a very dark day (The Engineering Toolbox, 2018) indicating that even when subjects were exposed to the maximum levels of light during the night \((M = 38.7\)
lux), the levels were relatively low. Additionally, recommended light levels for bedroom lighting range from 200 to 300 lux and 300 to 500 lux for classroom lighting (DiLaura, et al., 2011) suggesting that nursing staff may have been intentionally or unintentionally keeping light levels low in attempts to not disturb subjects.

Mean light exposure ($M = 1.74$ lux) was lower than reported by Bernhofer et al. (2014) who found mean light exposure to be 7.07 lux during nighttime hours on five acute medical units. Maximum light exposure (38.7 lux) was also lower than reported by Missildine et al. (2010) who found maximum light exposure to be 64 lux on medical units. There are several possible explanations for these findings. First, the decreased light levels in the rehabilitation setting may have been related to a decreased acuity of subjects resulting in less light needed to conduct patient care activities during nighttime hours. Next, since there are multiple different sources of light in the subject’s room, nursing staff may have used different light sources depending on the type of direct patient care activity provided. For example, a staff member turning or repositioning a subject may have turned on the bathroom light to minimize light exposure, while an overhead light may have been used for DPCAs such as medication administration, or venipuncture.

Regardless of the low light exposure found in the current study, low levels of light have been found to disturb sleep. In a study by Cho et al. (2013) bedside light during the entire nighttime period was used to investigate the effects of light exposure on sleep quality. Results indicated that subjects exposed to 40 lux throughout the night experienced a significantly higher percentage of Stage 1 sleep, and a decreased percentage of NREM Stage 3 and 4 SWS. These findings have implications for persons with TBI in the hospital setting since exposure to light during nighttime hours can influence not only sleep patterns, but also the timing and phases of
sleep cycles responsible for restorative sleep. Given that the mean maximum light exposure for subjects in the current study was 38.7 lux, compared to 40 lux in the study by Cho et al. (2013) more questions arise as to whether sleep architecture was affected in this sample of TBI subjects leading to a decrease in restorative sleep.

In summary, a variety of studies investigating the effects of interruptions on sleep have been completed in the ICU and acute hospital settings. No studies have explored the frequency and timing of both DPCAs, and light exposure in the rehabilitation setting and their effect on sleep patterns. In comparison with other studies in different settings, both DPCAs, and exposure to light during nighttime hours were less than reported in the literature. This is a reasonable and expected finding since the medical acuity of subjects is generally lower in the rehabilitation setting. Findings from this study suggest that a variety of DPCAs occurred during nighttime hours and that the necessity of these activities should be explored. Environmental stressors including DPCAs and light exposure are amenable to change and provide an opportunity for health care providers to control these stressors that may influence long-term outcomes.

**Covariates**

Age, severity of injury, and pain were used as covariates in this study since they have been found to affect sleep. An unexpected finding in the current study was that age, severity of injury, and pain did not contribute to explaining TST, SD, WASO, or SE.

The amount of SWS, or restorative sleep decreases with age, while the number of nighttime arousals increase with age (Carskadon & Dement, 2011). The NSF also provides recommendations for duration of sleep for individuals over 65 years, which are less than recommendations for individuals between 18 years and 65 years of age (Hirshkowitz et al., 2015). Findings in the literature are inconsistent in describing the relationship between age and
Sleep. Cantor et al. (2012) found no statistically significant differences in age between subjects with and without insomnia. In contrast, Ponsford et al. (2013) found age to be associated with TST and nighttime sleep. Although there was a wide range of ages represented in this sample, it is unclear why age was not found to contribute to explaining sleep patterns in this study.

Severity of brain injury has been reported to be associated with sleep. Although findings in the literature have been mixed, greater severity of injury has been found to be associated with increased daytime sleep time (Chiu et al., 2013), increased TST (Chiu et al., 2013; Ponsford et al., 2013), and increased daytime sleepiness (Baumann et al., 2007; Ponsford et al., 2013). While some studies have suggested severity of injury impacts sleep, Makley et al. (2008) found no significant differences in GCS scores between subjects with and without sleep wake cycle disorders. An explanation to why severity of injury may not have explained TST, SD, WASO or SE is that the first GCS score at time of admission to the hospital was used to represent severity of injury. Since subjects may decline medically in the first few days after injury, the first GCS score may not best reflect the true severity of injury. In future studies, using the lowest GCS score obtained within the first three days of admission is recommended.

Finally, pain has been associated with insomnia (Butterworth et al., 2013) and persons with TBI who have pain have been found to be twice as likely to have sleep complaints (Beetar et al., 1996). The relationship between pain and sleep was not consistent with the literature possibly due to a variation in the number of pain medication episodes between subjects well as how well pain was managed. While the mean pain medication episodes were 3.47, 19% of subject did not take any pain medication during the 48 hours the actiwatch was in place and 25.4% took pain medication on 5 or more episodes. The frequency of pain medication may not be related to sleep quantity or quality if pain control was adequate. In future studies a more
accurate measurement of pain would be patient’s self-report or use of a standardized pain assessment tool rather than the frequency of pain medication administration.

Pearson’s product moment correlation analysis indicated that older subjects had less pain ($r = -0.30, p = 0.015$), and higher VSH scores were significantly associated with less pain ($r = -0.39, p = 0.035$). The negative relationship between perceptions of sleep and episodes of pain medication is both supported by the literature (Beetar et al., 1996) and an expected finding, since theoretically persons who take less pain medication, possibly indicating less pain, would likely have better perceptions of sleep.

**Stressors and Sleep**

This study conceptualized that environmental stressors (DPCAs and maximum light exposure) would be negatively associated with TST and SE. Conversely, DPCAs and maximum light exposure were conceptualized as being positively associated with WASO.

In bivariate analyses, DPCAs had a significant negative relationship with SD ($r = -0.27, p = 0.035$) and SE ($r = -0.38, p = 0.002$) and a significant positive relationship with WASO ($r = 0.28, p = 0.028$), although no statistically significant relationships were found with TST. Maximum light exposure was positively related to WASO ($r = 0.36, p = 0.004$), but not significantly related to TST, SD, or SE. In multivariate regression analyses, DPCAs contributed to explaining SD and SE while maximum light exposure explained WASO. Of the proposed full models ($N = 63$), the only entire model that explains sleep is the model examining WASO.
Direct Patient Care Activities and Quantity of Sleep.

In the multivariate model, DPCAs were not an independent predictor of TST, nor did the entire model explain variance in TST. In contrast, DPCAs were a significant predictor of nighttime SD although when included with light, the entire model did not explain SD.

Patient care activities during the nighttime are often conducted either as a routine standard care practice, based on convenience related to workflow across all shifts, or as a result of a patient initiated need thus decreasing nighttime sleep. An explanation why DPCAs did not explain TST is that since sleep time was decreased during nighttime hours, subjects may have slept more during daytime hours in attempts to “catch up” on missed sleep during the night. An increase in daytime sleep would affect the TST by offsetting lost sleep during the night with increased sleep during daytime hours. Although data were not collected on the effects of DPCAs on daytime sleep, 36% of TST occurred during daytime hours.

Direct Patient Care Activities and Quality of Sleep

Regression analyses were conducted to determine variance in WASO and SE, explained by DPCAs and maximum light exposure. When DPCAs were entered in Step 2 as an independent predictor, DPCAs were a significant independent positive predictor of WASO, although became non-significant when light was added to the model in the final step.

Given that WASO is defined as the time scored as wake between the start and end of sleep, it was expected that an increase in direct patient activities would contribute to explaining an increase in WASO. A similar study by Gabor et al. (2003) found that in a sample of seven mechanically ventilated subjects in the ICU, patient care activities were responsible for 7.1% of awakenings measured by polysomnography.
Several possible causes for this finding were explored. Multicollinearity between DPCAs and maximum light exposure could be a potential cause but was not substantiated since the correlation between DPCAs and maximum light exposure was $r = .22$. Next, a trimmed model was constructed to exclude subjects with low lux levels to determine if measurement error contributed to findings. In the trimmed model, unexpectedly, DPCAs did not contribute to explaining WASO. Although the intent was to determine if measurement error altered results, findings did not support this assumption. Finally, specification error was considered as a possible reason for these results. Other environmental stressors such as noise that may have influenced the results were not explored. Further investigation is needed in future studies to examine other environmental stressors that may affect sleep.

Direct patient care activities were a significant independent negative predictor of SE; although when combined with light, the final model did not explain variance in SE. Since SE is a measure of sleep quality and accounts for the percentage of sleep in a given time period, it is expected that DPCAs would contribute to explaining decreased SE. Since the five most common patient care activities performed during nighttime hours (toileting, vital signs, assessment, medication administration, and repositioning), may involve waking patients to accomplish the task, it is not surprising that DPCA would at least partially explain an increase in WASO and decrease in SE.

Since the total variance in WASO, and SE accounted for by DPCAs and maximum light exposure combined was 10.6% and 8.6% respectively, this data suggests that there are additional factors that contribute to subjects having poor quality of sleep during nighttime hours. Damage to sleep centers in the brain resulting from the trauma (Vermalaelen, Grieffenstein, & deBoisblanc, 2015) as well as biochemical (Baumann et al., 2007), hormonal (Shekleton et al.,...
2010), medical, pharmacologic, (Vermalaelen et al., 2015) and other environmental stressors (Gabor et al., 2003) have been found to be factors contributing to sleep disturbance. Although many of these additional factors are not modifiable, findings from this study highlight the importance of managing those factors that can be controlled.

**Light and Quantity of Sleep**

Maximum light exposure did not contribute to explaining TST, or nighttime SD. Similarly, the entire model did not explain TST or nighttime SD. Although there were no significant findings between maximum light exposure, and TST or SD measured by actigraphy, the literature indicates that even low levels of light affect sleep architecture. Cho et al. (2013) used polysomnography to measure sleep architecture in a sample of 10 healthy adults when exposed to low levels of light during nighttime hours. Participants exposed to 40 lux light placed one meter away from their eyes during sleep time resulted in decreased Stage 3 and Stage 4 NREM SWS ($p < .0001$), increased Stage 1 sleep ($p = .044$), and increased arousals ($p = .003$) compared to participants with lights off during nighttime sleep. Since the current study did not measure sleep with polysomnography it is unknown whether sleep architecture was affected by light in this sample of TBI subjects.

One explanation why maximum light exposure did not contribute to explaining either SD is that SD is scored as the time between the start and end of sleep, without accounting for time subjects are awake during the SD time period. The difference between how these two sleep variables (SD and WASO) are measured may explain why light contributed to explaining WASO but not SD. In addition, since TST includes both nighttime and daytime sleep, differences in the variation in light exposure provides and explanation why TST was not influenced by maximum
light exposure. Additional investigation is needed into the relationship between maximum light exposure at night and TST and SD.

**Light and Quality of Sleep**

Maximum light exposure was a significant independent positive predictor of WASO. The entire model was also significant in explaining WASO. In contrast, maximum light exposure was not a significant independent predictor of SE, nor did the entire model explain SE.

Light serves as a key environmental signal that synchronizes the biological clock (Czeisler & Buxton, 2011) but can have detrimental effects on sleep patterns, sleep architecture, and circadian rhythms when there is exposure to light during nighttime hours (Cho et al., 2013). Light has been referred to as light pollution with negative effects on human physiology and circadian rhythms (Stevens et al., 2007). As a result, the impact of light exposure during nighttime hours cannot be underestimated in a setting where sleep is necessary to participate at an optimal level for recovery.

Maximum light exposure did not add to the model in explaining variance in SE. These findings are consistent with a study by Missildine et al. (2010) in which age, light, and noise were found to account for a nonsignificant 4.7% of the variance in SE. Sleep efficiency is defined as the number of one-minute epochs scored as sleep between the start and end of sleep divided by the rest interval (480 minutes) x 100. Sleep efficiency takes into account the entire 8-hour rest interval period, not only the time between the start and end of sleep. The way in which SE is measured may explain why light exposure explained WASO, but not SE, and may have resulted from a measurement issue involving predetermined rest intervals set by the Primary Investigator rather than subjects determining when they were intending on going to sleep. If subjects were awake at the time the rest interval started or had an increase sleep latency (time it
takes to fall asleep), light exposure during this time period before sleep was initiated would have affected SE results.

**Constant Supervision**

A post hoc analysis was conducted to explore differences between subjects with and without constant supervision to determine if subjects requiring constant supervision were different from the entire sample enrolled in the study. Findings suggested that subjects with constant supervision had more DPCAs ($Mdn = 6$ versus $4.5$, $p = .012$, respectively), and had increased severity of injury ($Mdn = 11.39$ versus $13$, $p = .009$, respectively) compared to subjects without constant supervision. These findings are expected since persons requiring constant supervision typically represent TBI subjects with more serious injuries affecting cognition and function who require additional DPCAs to maintain safety and provide necessary nursing and medical care as a result. Although differences were noted in the number of DPCAs and severity of injury, no differences were found among sleep variables.

**Methodological Considerations**

Actigraphy provided a valid, reliable, and cost-effective method of collecting both sleep and light data and was found to be feasible with TBI subjects in the rehabilitation setting. Of the 64 subjects enrolled, only one subject removed the actiwatch before completing study procedures and there was only one occasion when the actiwatch malfunctioned. These findings are similar to a study by Towns et al. (2016) where 86% of the sample ($n = 19$) was able to wear the actiwatch for three consecutive days. Similarly, Higgins et al. (2007) also reported that subjects did not feel wearing the actiwatch was burdensome and nurses did not report that the actiwatch interfered with care. No nurses in this study reported complaints from subjects wearing the actiwatch or with the actiwatch interfering with care.
Both the sensitivity (ability to detect sleep), and specificity (ability to detect wake) of the actiwatch have been compared to polysomnography. Marino et al (2013) reported high sensitivity (.97) while specificity was low (.33). Limitations of actigraphy reported in the literature include overestimating sleep quantity and SE if used on an extremity with paresis, or as a result of scoring quiet wakefulness as sleep. Contrarily, WASO can be overestimated if used on an extremity with spasticity. These issues were addressed during screening of subjects by assessing for paresis, paralysis, muscle tremors, or spasticity to minimize potential error in measurement. In the current study, five subjects had paresis of the upper extremity resulting in the actiwatch being placed on the opposite wrist. No subjects had contractures or spasticity.

Measuring light posed many challenges; therefore, two approaches were used to better understand light measurement. First, a methodological approach was used to minimize possible systematic error. The lens of the light sensor was cleaned between subjects to minimize debris interfering with full exposure to light. In addition, subjects were asked to keep the arm with the actiwatch above the blankets during nighttime hours if possible. Nursing staff documented visibility of the actiwatch only 63.9% of the time during hourly rounding suggesting systematic error with light exposure data. What is not known is whether lights were off or on when the actiwatch was covered by the blankets further making interpretation of light data difficult. Both Bernhofer et al. (2014) and Higgins et al. (2007) also reported limitations with the potential for the lens of the actiwatch being covered during light measurement. Results from the current study and previous studies suggest that using actigraphy to measure light poses challenges. Although other studies have positioned light monitors near the subject’s bed to minimize issues with blankets covering the light sensor, this methodology does not take into account time when subjects are not in their hospital bed. Additionally, another study used a light monitor attached to
the subject’s forehead, although this method is burdensome for subjects. Future research is needed to determine the most reliable and valid methods for collecting light data in the clinical setting.

Despite attempts to maintain control over these potential causes of error through design, findings from the study required further investigation. In order to further investigate the validity of light measurement, a variety of analytical methods were used to identify possible systematic error. Inter-device reliability was conducted and although findings did not indicate perfect agreement between the three actiwatches for total light exposure (range 3.53 lux to 3.72 lux), or maximum light exposure (range .02 lux to .07 lux), these differences were small and considered to be not clinically significant.

A second method of investigating the validity of the light data was done by comparing the agreement between the actiwatch placed on the headboard of the subject’s bed, and the subject’s wrist using the Bland Altman plots. Findings from one-sample t-tests were not significant indicating some level of agreement between devices although regression analysis results were significant suggesting bias between the two devices. Limitations included only 11 observations between the two measurement methods and several outliers identified in the scatterplots.

Findings from this study are in contrast with a study by Higgins et al. (2007) who also used Bland Altman plots to measure agreement between a wrist-worn Sleepwatch-L (Ambulatory Monitoring, Inc., Ardsley, NY) and an Extech light meter (Extech Instruments Corporation, Waltham, MA). Higgins et al. did find a level of agreement within 1.96 standard deviations between the Sleepwatch-L and the light meter. Differences between the two studies may have been related to a small sample size in this study as well as outliers. In future studies, to
better understand the validity of light data, it is recommended to measure whether lights are on or off simultaneously when measuring if the light sensor is visible or not visible. Another recommendation is to perform reliability testing with all subjects with one watch placed on the headboard and the other on the subject’s wrist. Also, it is recommended to increase the sample size for reliability testing.

**Trimmed Model for Regression Analysis**

A trimmed model of 47 subjects with maximum light exposure greater than 5 lux was constructed to further investigate unusual findings in the initial regression analyses possibly related to systematic error in measurement of light. In addition to removing subjects with low light exposure, covariates of age, severity of injury, and pain were not included in the trimmed model since they did not contribute to explaining TST, SD, WASO, or SE in the full model.

In the regression analysis for WASO using the full sample ($n = 63$), DPCA had a significant beta in Step 2, which became non-significant in the Step 3 (entire model) when maximum light was added. Also, the beta for maximum light exposure was positive in the regression analyses for both SD and SE using the full sample ($n = 63$), when theoretically it should have been negative. The trimmed model was constructed to evaluate whether the subjects with low maximum light exposure may have had influence on the model contributing to the unusual findings. Results of the regression analysis using the trimmed model ($n = 47$) had similar findings for TST and SD.

In contrast, compared to the full model for WASO where the beta was significant in Step 2, but became non-significant in Step 3 (the entire model), the beta for DPCAs was found to be non-significant in both the first or second step of the trimmed model. This was an unexpected
finding since the intent of the trimmed model was to exclude subjects with low lux exposure that may have been related to measurement error.

Another difference in the trimmed model was that the entire model became significant in explaining SE when it had been non-significant in the full model. Finally, in the trimmed model for SE, the beta for maximum light became negative compared to the full model when it was unexpectedly positive. Based on the unexpected findings in the trimmed model, further investigation is needed to explore ways to manage light measurement error.

**Theoretical Model**

Pearlin’s stress process model was used to guide this study in exploring how hospital environmental stressors impact sleep outcomes in persons with TBI in the rehabilitation phase of recovery. Findings partially support the relationships identified within the model. The entire model including environmental stressors (DPCAs and maximum light exposure), and covariate of age, severity of injury and pain explained variance in WASO, although did not explain TST, nighttime SD or SE.

The stress process model proposes that undesirable, uncontrolled, or unscheduled life events serve as primary stressors that lead to chronic strains, thereby serving as secondary stressors. Both the primary and secondary stressors converge leading to outcomes that are manifested by the stressors (Pearlin, 1989). In this study, the initial TBI event served as the primary stressor resulting in hospitalization. The hospitalization exposed the TBI person to environmental stressors including patient care activities and light exposure during nighttime hours, which served as secondary stressors. Both the stressful experience related to the TBI followed by exposure to environmental stressors combined to affect sleep patterns, which were identified as the outcome proposed in the model. The model also proposes that life events and
chronic strains do not occur in isolation and that additional factors can influence outcomes. The TBI person’s age, severity of injury, and pain were considered factors that may have contributed to sleep outcomes although findings did not support this assumption. Possible factors that may have affected results that were not explored were noise, physiological, and psychological factors experienced during hospitalization.

In summary, findings from this study at least partially support the stress process model. While the entire model including environmental stressors of DPCAs and light exposure did not explain variance in sleep quantity TST, SD, or SE, the model did explain variance in WASO.

**Limitations and Future Directions**

This study had several limitations. The setting was limited to one study site, which limits the generalizability of the study findings. Also, since this study used a convenience sample of TBI subjects in the rehabilitation setting was used, selection bias could affect external validity. Although there are many benefits to using actigraphy in the clinical setting, actigraphy has been found to have low specificity so that periods of quiet restfulness are potentially scored as sleep, thus overestimating TST. Although the VSH scale is a reliable self-report measurement instrument for measuring perceptions of sleep, the VSH has never been used to measure perceptions of sleep in persons with TBI and consequently may not allow for comparison of results with other studies in the same setting.

Several issues arose regarding measurement of light in this study. Since the nursing staff was not blinded to the study, changes in patterns of light exposure or patient care activities may have occurred introducing bias and influencing results. Additionally, the light monitor being positioned under blankets 63.9% of the time impacted the validity and reliability of light data. This study investigated the effects of patient care activities and light on sleep patterns although
other environmental factors such as noise that may have affected sleep were not measured. This study controlled for pain by using the number of episodes of pain medication during the time period that the actiwatch was worn, although other medications that could have affected sleep patterns were not investigated. This study monitored sleep patterns for two consecutive days although extended periods of time for monitoring sleep beyond 48 hours would have allowed for examination of sleep patterns over time.

During preliminary analysis to determine how to manage two days of patient care activity, light, and sleep data, no individual differences between day one and two were identified except for TST. A decision was made to sum and average day one and two data, in addition to analyze each day separately for TST. The model for day one and day two remained non-significant, consistent with the model including both days combined. One difference identified was that in the model for day two, DPCAs ($\beta = -.35, p = .018$), and age ($\beta = .30, p = .043$), made a significant contribution to explaining the model.

This study provides a basis for future research investigating environmental stressors and their influence on sleep in the rehabilitation setting. Future studies are needed to better understand how additional environmental stressors such as noise contribute to sleep disturbance. Noise may further contribute to explaining sleep patterns since persons with TBI are sensitive the increased environmental stimulation. More knowledge is also needed regarding ways improve the validity of light measurement. Finally, studies focusing on effective nursing interventions that can improve sleep are needed to optimize cognitive and functional outcomes from persons recovering from TBI.
Contribution to Scientific Knowledge

The domains of nursing practice focus on the nurse, patient, health, and environment. Nurses have a responsibility to promote the health and well-being of patients while optimizing their recovery by managing both the care being delivered and the environment in which this takes place. The current study has contributed to the scientific knowledge regarding sleep patterns of persons with TBI in the acute rehabilitation hospital setting. Results have demonstrated that persons with TBI sleep more during daytime hours, have decreased sleep time during nighttime hours, and have frequent nighttime awakenings leading to fragmented sleep patterns. The influence of hospital environmental stressors on sleep outcomes has been highlighted in this study. Although a focus of the nursing discipline and profession is on promoting and optimizing health, standard care practices do not reflect these core values. This study has identified that nurses conduct patient care activities during the night that can affect sleep quantity and sleep quality. Specifically, patient care activities have been shown to decrease SD at night as well as decrease the efficiency of sleep. Additionally, this study has provided information about light exposure during nighttime hours and its impact on overall sleep patterns. Light levels in the rehabilitation setting were found to be low although did have a positive relationship with the time subjects were awake during nighttime hours.

Pearlin’s stress process model was used to guide the study in identifying relationships between potentially stressful environmental factors and sleep outcomes. Findings from this study did support the model showing that the primary stressor of sustaining a traumatic brain injury, led to a secondary stressor including hospitalization and an unfamiliar environment. The conditions of the hospital environment including patient care activities and light exposure during the night served as secondary stressors resulting in alterations in sleep patterns. Literature has
consistently identified sleep disturbance across the continuum of care from the acute hospital setting, and in the community. This study provides further evidence that sleep disturbance occurs in the rehabilitation setting and that hospital environmental stressors play a role in affecting sleep.

Finally, this study has provided knowledge about use of actigraphy as an objective measurement for sleep in persons with TBI in the rehabilitation setting. The study has demonstrated that use of actigraphy for measurement of quantity and quality of sleep in subjects with TBI in rehabilitation is feasible. Additionally, the study has provided insight into use of the Verran and Snyder-Halpern Sleep Scale as a subjective measure of sleep in the TBI population and its relationship to sleep measured using actigraphy.

Conclusions

Findings from this study highlight the role that environmental stressors play on quantity and quality of sleep. This study has shown that patient care activities, along with increased light exposure, are occurring throughout nighttime hours. The study has also demonstrated that activities occurring during nighttime hours as well as increased lighting can impact both the quantity and quality of sleep for persons with TBI.

Since studies have shown that significant gains in recovery occur during the acute inpatient rehabilitation phase of recovery (Agrawal & Joshi, 2014) the importance of optimizing sleep cannot be underestimated. Therapy needed to improve both cognitive and physical function of persons with TBI takes enormous energy and effort on the part of the TBI survivor thus promoting sleep in the rehabilitation setting must be a priority.

Persons with TBI are a vulnerable population since they are predisposed to sleep disturbance. Injury to the brain can damage areas responsible for regulating sleep patterns,
therefore stressors in the hospital setting that disturb sleep can further compound sleep disturbance. This study has identified that patient care activities occur throughout the nighttime hours leading to fragmentation of sleep. Studies have shown that fragmented sleep patterns can lead to decreased SWS and decreased REM sleep which is the time when restoration, memory consolidation, brain plasticity, and new learning occurs. Since a focus of rehabilitation for persons with TBI is regaining both cognitive and physical function, the importance of promoting effective sleep patterns cannot be underestimated.

Information from this study provides evidence of the need for health care providers to examine activities that are necessary during nighttime hours as opposed to activities that are done for hospital convenience. Promoting sleep patterns that optimize recovery is an interdisciplinary issue that should be addressed by all healthcare providers responsible for the care of persons with TBI. Patient care activities that can reasonably be performed during non-sleeping hours such as routine daily vital signs or routine venipuncture should not be performed based on standard care practice or convenience for staff members. Additionally, waking patients early due to staffing concerns, early appointments, or in attempts to disperse tasks across all shifts should be eliminated unless needed for medical necessity.

Consistent evidence in the literature exists to support the concept that sleep disturbance is common both immediately after the TBI and continues into the community setting. There is an opportunity for health care providers to manage modifiable environmental stressors that could potentiate sleep disturbances after discharge from the hospital setting. This study offers information about the feasibility of actigraphy in measuring sleep patterns that can be used to evaluate targeted interventions to improve sleep. Findings from this study have also provided
information that will be useful when evaluating standard care practices and provide a basis for making policy changes to optimize sleep in the hospital setting.

**Contributions to the TBI Literature**

Much research has been done investigating sleep patterns in persons with TBI although little has been done with that population in the acute rehabilitation setting. This study provides initial research with this specific TBI population and provides a basis for future studies to examine the relationship between sleep patterns, and cognitive and functional outcomes. This study has shown that there is a negative relationship between environmental stressors of patient care activities and light, and sleep quantity and quality during nighttime hours. This study can serve as empirical support for future studies investigating differences between subjects not interrupted during nighttime hours versus those provided with standard of care. Additionally, research is needed to further understand the role that timing of pain medication has on sleep patterns as well as the impact of sleep promoting medications on sleep patterns in the rehabilitation setting. This study found that severity of injury and age did not explain quality or quantity of sleep. Further research is needed to determine if using different time parameters for measuring severity of injury would influence findings. Given previous research that suggests that sleep disturbances continue after hospitalization future studies are needed to identify how hospital environmental stressors affect sleep disturbance in the community setting.

**Conclusion**

Sleep disturbances were found to be common in the TBI population in the rehabilitation setting. Sleep disturbances were characterized by decreased nighttime sleep time, increased daytime sleep, increased TST, increased WASO and decreased SE. Although causes of sleep disturbance are multi-factorial and can include biological, hormonal, physical, and psychological...
factors, environmental stressors including patient care activities and light exposure have also been shown to contribute to disturbed sleep. Both direct and indirect patient care activities occurred throughout the night and while light exposure to during nighttime hours was low, it did occur during nighttime hours. Sleep affects the recovery process and can have an impact on cognition, mood, functional performance and fatigue, which can limit optimal rehabilitation efforts and recovery. Given the brevity of inpatient rehabilitation, it is essential for health care providers to examine environmental factors that serve as stressors and ultimately impact sleep patterns.
References


Change in trend over the past ten years. *Journal of Neurology, Neurosurgery & Psychiatry, 75*(1), 161-162.


doi:10.5665/sleep.3142


doi:10.1016/j.gerinurse.2010.02.013


doi:10.1016/j.sleep.2013.07.009


doi:10.1016/j.apmr.2009.05.011


doi:10.1080/15402002.2012.726203


doi:10.1177/1545968315619697


NOTIFICATION OF INITIAL APPROVAL

Date: February 4, 2016

From: Ann Avery, M.D.
To: Shelly Amato  CC:

Key Personnel:

Shelly Amato

Department Chair:

Molly McNett, PhD

Re: Study # IRB15-00861  Effects of Environmental Factors on Sleep Patterns in Traumatic Brain Injured Adults in the Rehabilitation Setting

Link: IRB15-00861

Renewal Period: 12 Months

EMR Note Required: Not Required

Risk: Not Greater Than Minimal Risk

I am pleased to inform you that the above referenced protocol was approved on 2/4/2016. Approval of the protocol and the consent form(s) is for the period of 2/4/2016 to 1/11/2018.

Expedited Regulatory Category:


**********
The request for a Partial Waiver is Approved under 45CFR164.512(i)(1)(ii) as the protocol detail adequately documents that: (a) the purpose of the Partial Waiver is recruitment; (b) the use of the Protected Health Information (PHI) involves no more than minimal risk to the privacy of patients; (c) the PHI will be used solely to facilitate the research protocol as an aid to recruitment or to expand the research study; (d) information about potential subjects will be destroyed if they decline participation; (e) the PHI will not be reused or disclosed; (f) the PHI will not leave the premises of The MetroHealth System.

**********

Regulation 45CFR46.109(e) requires that the Institutional Review Board review all studies "not less than once per year". The Institutional Review Board will attempt to notify investigators in advance of impending expiration, but it remains the responsibility of investigators to remain aware of the study expiration date and to submit a Continuing Review Application in a timely manner.

No deviations from the Approved Protocol may be initiated without MetroHealth Institutional Review Board review and approval, except when necessary to eliminate apparent immediate hazards to the participant. Any such change must be reported promptly (within 24 hours) to the IRB via the eIRB reportable event application.

All Reportable Events or Unanticipated Problems that occur with this study must be reported to the Institutional Review Board within ten working days (from the time they become known to the Principal Investigator). Internal Subjects deaths must be reported to the IRB immediately (within 24 hours) of the Investigator or study team becoming aware of it. For any questions on reporting Adverse Events or Unanticipated Problems, please call the IRB Office (216-778-7575) or consult the MHS eIRB SOPs.

If applicable, all approved Consent Forms may be found on the Documents Tab of your approved Study (use only those listed under Approved Consent Forms). These are the only forms you are permitted to use to consent subjects. Subjects must be given a signed and dated copy of the consent prior to their participation.

All research conducted in The MetroHealth System must be conducted according to applicable Federal, State, and Local regulations and MetroHealth Policies and IRBs. In addition, investigators are required to follow Good Clinical Practices (GCPs) as outlined in the ICH Guidelines. This is a requirement not only of the MetroHealth Institutional Review Board but also of The MetroHealth System. If you are not familiar with these guidelines, you can find a copy of them on the IRB Home Page under General Information for Investigators.
If your study is selected for audit by the IRB, the study files and your conduct of the study will be assessed against all MetroHealth IRB SOPs, Federal, State, and Local Regulations governing research, Institutional policies, and GCPs.

This study is next subject to continuing review on or before 1/11/2018, unless it is closed before that date. Please inform the IRB promptly when your study is completed. To close this study, you will need to complete and submit a Continuing Review which will serve as your final report to the IRB. On the Continuing Review form question 1.2, select Completed/Closed then complete the forms and submit them to the IRB. Please contact the IRB Office at 216-778-5459 if you have any questions or need assistance.

All pediatric research is reviewed under the applicable regulations at 45CFR46.404-408 and the parallel FDA regulations if applicable.

Comments:
Even though this study has been approved by the IRB there may be other approvals needed before you can start your research. You may not start your study until you have a signed contract with your sponsor (if applicable). The contract language on subject injury must be consistent with the consent form language. If it is not you will need to submit an Amendment to the IRB before starting your study.

Approved Documents:

<table>
<thead>
<tr>
<th>Name</th>
<th>Version</th>
<th>Modified</th>
</tr>
</thead>
<tbody>
<tr>
<td>4-21-2016 VSH Sleep scale.docx.pdf</td>
<td>0.03</td>
<td>1/12/2017 11:31 AM</td>
</tr>
<tr>
<td>Amended Data Collection Tool Clean Copy.pdf</td>
<td>0.01</td>
<td>4/26/2017 11:34 AM</td>
</tr>
<tr>
<td>Combined HIPPA and ICF.docx.pdf</td>
<td>0.03</td>
<td>1/12/2017 11:31 AM</td>
</tr>
<tr>
<td>Data Collection Tool.docx.pdf</td>
<td>0.02</td>
<td>1/12/2017 11:31 AM</td>
</tr>
<tr>
<td>Flyer.docx.pdf</td>
<td>0.03</td>
<td>1/12/2017 11:31 AM</td>
</tr>
<tr>
<td>PCESL .docx.pdf</td>
<td>0.03</td>
<td>1/12/2017 11:31 AM</td>
</tr>
</tbody>
</table>

Sincerely,
Ann Avery, M.D.

MetroHealth Institutional Review Board, Chairperson
Appendix B

Capacity to Consent Checklist       Date: ____________________

Patient Research ID Number: ________________________________

<table>
<thead>
<tr>
<th></th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Able to ___ verbalize ___ write purpose of study</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Able to ___ verbalize ___ write procedures involved in study</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Able to ___ verbalize ___ write risks and benefits of study</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Able to ___ verbalize ___ write ability to withdrawal from study at any time</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 2. Bland Altman Scatterplot for Total Light Exposure
Appendix D

Figure 3. Bland Altman Scatterplot for Average Light Exposure

\[ \text{---Diff + (1.96 x SD)} \]
\[ \text{---Mean Difference} \]
\[ \text{---Diff – (1.96 x SD)} \]
Appendix E

Figure 4. Bland Altman Scatterplot for Maximum Light Exposure

--Diff + (1.96 x SD)
--Mean Difference
--Diff – (1.96 x SD)
Appendix F

Patient Care Environmental Stressor Log (PCESL)

<table>
<thead>
<tr>
<th>Time Slot</th>
<th>11-00</th>
<th>11:15-11:30</th>
<th>11:30-11:45</th>
<th>11:45-12:15</th>
<th>12:15-12:30</th>
<th>12:30-12:45</th>
<th>12:45-1:00</th>
<th>1:00-1:15</th>
<th>1:15-1:30</th>
<th>1:30-1:45</th>
<th>1:45-2:00</th>
<th>2:00-2:15</th>
<th>2:15-2:30</th>
<th>2:30-2:45</th>
<th>2:45-3:00</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Score pt. as:</em></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep=S</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Awake=W</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Actiwatch</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Position:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visible=V</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not Visible=N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indirect</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Documentation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Managing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Equipment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Care/Interact.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>with Roommate</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Direct</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vital Signs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assessment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medications</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Restraint</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Restraint</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Application</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>or removal</td>
<td>Repositioning</td>
<td>Bathing</td>
<td>Dressing</td>
<td>Toileting</td>
<td>Transfers</td>
<td>Radiological Processes</td>
<td>Tracheostomy Care</td>
<td>Suctioning</td>
<td>Venipuncture</td>
<td>Respiratory Care</td>
<td>Enteral Feeding</td>
<td>Other care</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------</td>
<td>---------------</td>
<td>---------</td>
<td>----------</td>
<td>-----------</td>
<td>-----------</td>
<td>------------------------</td>
<td>-----------------</td>
<td>------------</td>
<td>-------------</td>
<td>-----------------</td>
<td>----------------</td>
<td>-----------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Occurrence of an overhead page or alarm between 11pm-7am  ___YES   ___NO
Appendix G

Training for Completing Patient Care Environmental Stresoor Log (PCELS)

1. The PCELS should be completed between the hours of 11pm-7am for the two nights the Actiwatch is worn.
2. Each hour, during hourly rounding, score the patient as either sleeping=S, or awake=W.
3. Each hour, during hourly rounding, score “V” if the actiwatch is visible (above the covers), or “N” if the actiwatch is not visible (under the covers).
4. When an activity is performed, place a check mark under the time column in which the activity occurred.
5. The PCELS will be collected in the morning by the Primary Investigator (Shelly Amato).

Indirect Activities- Includes any activities that are done with the roommate or with the subject that do not involve direct care. See examples below:

- **Observation**- Entering the patient room to do hourly rounding on either of the patients without providing hands-on care to patients
- **Documentation**- Documenting in the room in EPIC for either patient in the room
- **Managing Equipment**- Turning on/off IV pumps, tube feeding pumps etc. for either patient in the room
- **Care/Interactions with roommate**- Any hands-on care that is provided to the roommate of the patient who is a subject in the research study

Direct Activities- Includes any hands-on care that is provided to the subject

- **Vital Signs**- Vital signs taken on the patient
- **Assessment**- Any type of physical assessment involving touching the patient such as listening to lung sounds, checking for drainage from a catheter etc.
- **Medications**- Medication that is given either IV or po to a patient during the hours of 11pm-7am (including IV medications)
- **Dressing change**- Dressing changes involving changing the entire dressing or reinforcing dressings (although you are also assessing the wound, do not include “assessment” unless you are assessing another body system.
- **Restrain application or removal**- Application, removal or readjusting restraints
- **Repositioning**- Assistance that is provided with repositioning in bed
- **Bathing**- Bathing can include partial or full bed baths or assistance at the sink or shower. Do not include cleaning incontinence episodes
- **Dressing**- Changing gowns, applying ted hose, pants, shirts etc.
- **Toileting**- Getting patients up to the toilet, use of the bedpan or cleaning up incontinent episodes
- **Transfers**—Transferring patient in or out of bed.
- **Radiological procedures**—Radiological procedure that is done in the room.
- **Tracheostomy care**—Changing trach dressing, cleaning around trach or removing the inner cannula
- **Suctioning**—Suctioning tracheostomy or the mouth
- **Venipuncture**—Phlebotomy or IV insertion
- **Respiratory Care**—Care provided involving respiratory treatments or incentive spirometry
- **Enteral Feeding**—Instillation of tube feedings, flushing feeding tubes, setting up or taking down tube feeding bags
- **Other care**—Any activity that is not described above

If two or more activities occur, account for each one individually (check all of them)

If care is provided by a healthcare provider other than nursing staff, (Radiology, Physician), please document the activity for the healthcare provider.

If a patient is transferred off the unit, remove the actiwatch prior to the patient leaving and place the actiwatch in the medication room.

**DO NOT REMOVE THE ACTIWATCH WHEN SHOWERING.** The actiwatch can be worn in the shower; Pat dry the actiwatch if gets wet.

**Case Example**

You enter the room to answer a call light that was put on by the roommate at midnight. The roommate asks to go to the bathroom. After assisting her to the bathroom, you document the output in the EMR at the bedside. While in the room, you notice that the subject is sleeping and the watch is under the covers.

**Document:**

Under Indirect Activities-

- Care/Interaction with roommate
- Documentation

Score “S” as sleeping

Score “N” for actiwatch not visible
**Case Example**

It is 5 am and you need to draw blood work. After drawing the blood, the patient asks to use the bathroom and then decides to wash up at the sink and get dressed. The patient returns to bed at 6am.

**Document:**

Under Direct Activities:

- Venipuncture
- Toileting
- Bathing
- Dressing

Score “W” as Awake

Score “V” as Visible
Appendix H

Verran and Snyder-Halpern Sleep Scale (VSH)

Directions: Answer each question by placing a vertical mark across the answer line at a point which BEST REFLECTS YOUR OPINION.

Answer all of the following questions about your last night’s sleep. Consider the night’s sleep to begin from the time your first tried to go to sleep to the time you were finally “up” in the morning.

Didn’t wake at all ____________________________________________  Was awake off and on all night

Didn’t move ____________________________________________  Tossed all night

Had no sleep ____________________________________________  Had 10 hours’ sleep

Fell asleep immediately ____________________________________________  Didn’t sleep at all

Slept lightly ____________________________________________  Slept deeply

Awoke exhausted ____________________________________________  Awoke refreshed
Awoke abruptly  _________________________________________  Awoke spontaneously

Had a bad night’s sleep  _________________________________________  Had a good night’s sleep

- Visual analogue uses a 100mm response line.
- Subjects answer each question about their previous night’s sleep by placing a vertical mark on the 100mm line
- Vertical marks are measured in millimeters from the zero endpoint and given the measured value.
- Scores on the bolded questions should be reversed, then scores on all items are summed.
- The higher the total score, the better the quality of sleep.
Appendix I

Data Collection Tool

________________________________________________________________________

Environmental Predictors of Sleep Patterns in Traumatic Brain Injured Adults in the Rehabilitation Setting

________________________________________________________________________

Data collector initials: __________

Date: __________________________
I. Screening Data

Inclusion Criteria:

- Traumatic Brain Injury [ ] yes [ ] no
- 18 Years of age or older [ ] yes [ ] no
- English or Spanish Speaking [ ] yes [ ] no
- Able to participate in 3 hours of therapy each day [ ] yes [ ] no

Exclusion Criteria:

- Documented Diagnosed Sleep Disorder [ ] yes [ ] no
- Non-Traumatic Brain Injury Diagnosis [ ] yes [ ] no
- Documented diagnosed depression [ ] yes [ ] no
- Documented diagnosed anxiety [ ] yes [ ] no
- Isolation [ ] yes [ ] no

Screening for Placement of Actiwatch

- Presence of contracture [ ] yes [ ] Rt. UE [ ] Left UE [ ] no
- Presence of spasticity [ ] yes [ ] Rt. UE [ ] Left UE [ ] no
- Presence of paresis or paralysis [ ] yes [ ] Rt. UE [ ] Left UE [ ] no
- Actiwatch placed on: [ ] Rt. UE [ ] Left UE
II. Baseline Patient Data

Primary Admission Diagnosis (Check all that apply)

☐ TBI  ☐ CHI  ☐ Cerebral Contusion  ☐ SDH  ☐ EDH  ☐ ICH  
☐ DAI  ☐ Intraparenymal Hemorrhage  ☐ SAH  ☐ IVH  

Mechanism of Injury:

☐ MVC/MVA  ☐ Fall  ☐ MCC  ☐ Assault  ☐ Struck Pedestrian  
☐ GSW  ☐ Other:

Comorbidities:

☐ Diabetes  ☐ CHF  ☐ Cancer  ☐ HTN  ☐ CAD/MI  
☐ Other  ☐ COPD  ☐ Liver Failure  ☐ Asthma  ☐ Renal Failure  ☐ Other  
☐ Not noted in documentation/unable to obtain due to mental status  

Glasgow Coma Scale on Admission: _____________

Time Since Injury in Days: ____________

Age: (years) ___________  Gender: ☐ Male  ☐ Female

Race:

☐ White  ☐ Black  ☐ Hispanic  
☐ Native American  ☐ Asian  ☐ Other  ☐ Unknown

Marital Status at time of injury:

☐ Married  ☐ Single  ☐ Divorced  ☐ Separated  ☐ Widowed  ☐ Engaged

Employment Status at time of Injury:

☐ Employed  ☐ Unemployed  ☐ Other  ☐ Not documented
Time of First Therapy Appointment:
☐ Before 9am  ☐ 9am  ☐ 10am  ☐ 11am  ☐ after 11am

Roommate:
☐ Yes  ☐ No

Hospital Day that Actiwatch was placed: ________________

Number of episodes pain medications given in 48 hour period Actiwatch is in place: ______

Patient on constant supervision Day 1  ☐ Yes  ☐ No

Patient on constant supervision day 2  ☐ Yes  ☐ No

Date Actiwatch Applied: __________

Date Actiwatch Removed: __________
A research study is being done on the Brain Injury Rehabilitation Unit to learn about sleep patterns of traumatic brain injured persons and how the environment affects sleep.

The study involves wearing an actigraph watch (similar to a wrist watch) for 2 days that will measure sleep and wake patterns and completing a sleep questionnaire.

In addition to wearing the watch, information will be collected about patient care activities and exposure to light during nighttime hours.

If you have had a traumatic brain injury and are 18 years or older, you or your legal authorized representative will be contacted to see if you are willing to participate in this study.

For more information or questions about the study, feel free to contact:

Shelly Amato PhD(c) RN, Primary Investigator (216) 957-3635
### Appendix K

**Number of Injuries Sustained and Glasgow Coma Scale Score**

<table>
<thead>
<tr>
<th>Glasgow Coma Scale Score</th>
<th>M</th>
<th>(SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>One TBI Injury(^a)</td>
<td>11.60</td>
<td>2.96</td>
<td>5 - 15</td>
</tr>
<tr>
<td>Two TBI Injuries(^b)</td>
<td>11.34</td>
<td>3.57</td>
<td>3 - 15</td>
</tr>
<tr>
<td>Three TBI Injuries(^c)</td>
<td>11.11</td>
<td>3.90</td>
<td>3 - 15</td>
</tr>
</tbody>
</table>

*Note.* TBI injuries can include subarachnoid hemorrhage, subdural hematoma, cerebral contusion, intracranial hemorrhage, intra-ventricular hemorrhage, diffuse axonal injury, epidural hematoma, intraparenchymal hemorrhage.

\(N = 63\). \(^a\)\(n = 26\). \(^b\)\(n = 25\). \(^c\)\(n = 12\).
### Appendix L

Table 26

*Hierarchical Regression Analysis for Variables Predicting Total Sleep Time Day 1*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.83</td>
<td>1.07</td>
<td>.12</td>
</tr>
<tr>
<td>GCS</td>
<td>-7.88</td>
<td>6.33</td>
<td>-.18</td>
</tr>
<tr>
<td>Pain</td>
<td>-2.20</td>
<td>5.92</td>
<td>-.05</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Step 1</strong></td>
<td>Adjusted $R^2$</td>
<td>-.020</td>
<td></td>
</tr>
<tr>
<td><strong>Model 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.10</td>
<td>1.08</td>
<td>.14</td>
</tr>
<tr>
<td>GCS</td>
<td>-10.08</td>
<td>6.79</td>
<td>-.23</td>
</tr>
<tr>
<td>Pain</td>
<td>-1.04</td>
<td>6.06</td>
<td>-.02</td>
</tr>
<tr>
<td>DPCA</td>
<td>-4.77</td>
<td>5.23</td>
<td>-.13</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td>Adjusted $R^2$</td>
<td>-.023</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$R^2 \Delta$</td>
<td>.014</td>
<td></td>
</tr>
<tr>
<td><strong>Model 3</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.10</td>
<td>1.09</td>
<td>.14</td>
</tr>
<tr>
<td>GCS</td>
<td>-10.06</td>
<td>6.85</td>
<td>-.23</td>
</tr>
<tr>
<td>Pain</td>
<td>-.97</td>
<td>6.17</td>
<td>-.02</td>
</tr>
<tr>
<td>DPCA</td>
<td>-4.85</td>
<td>5.37</td>
<td>-.13</td>
</tr>
<tr>
<td>MaxLux</td>
<td>.04</td>
<td>.44</td>
<td>.11</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Step 3</strong></td>
<td>Adjusted $R^2$</td>
<td>-.041</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$R^2 \Delta$</td>
<td>.000</td>
<td></td>
</tr>
</tbody>
</table>

*Note.* $N = 63$. GCS = Glasgow coma scale; DPCA = direct patient care activities; Max Lux = maximum lux exposure.

* $p < .05$. ** $p < .01$. 
### Appendix M

Table 27

*Hierarchical Regression Analysis for Variables Predicting Total Sleep Time Day 2*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>1.76</td>
<td>1.31</td>
<td>.19</td>
</tr>
<tr>
<td>GCS</td>
<td>-6.51</td>
<td>7.75</td>
<td>-.12</td>
</tr>
<tr>
<td>Pain</td>
<td>-10.00</td>
<td>8.86</td>
<td>-.15</td>
</tr>
</tbody>
</table>

Step 1  
Adjusted $R^2$ .016

| **Model 2** |      |       |      |
| Age       | 2.73 | 1.34  | .30* |
| GCS       | -14.91| 8.39  | -.27 |
| Pain      | -8.54| 8.60  | -.13 |
| DPCA      | -16.85| 7.56  | -.31* |

Step 2  
Adjusted $R^2$ .078  
$R^2 \Delta$ .074*

| **Model 3** |      |       |      |
| Age       | 2.77 | 1.34  | .30* |
| GCS       | -15.73| 8.42  | -.29 |
| Pain      | -6.92| 8.73  | -.10 |
| DPCA      | -19.29| 7.90  | -.35* |
| MaxLux    | .39  | .37   | .14  |

Step 3  
Adjusted $R^2$ .080  
$R^2 \Delta$ .017

*Note.* $N = 63$. GCS = Glasgow coma scale; DPCA = direct patient care activities; Max Lux = maximum lux exposure.  
* $p < .05$. ** $p < .01$. 

178