TESTING THE EFFECT OF EARPLUG USE ON SLEEP IN CRITICALLY ILL PATIENTS

by

MICHELLE ANNE MARIE HOUSE
B.S.N., University of Victoria, 1996

A THESIS SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE IN NURSING

in

THE FACULTY OF GRADUATE STUDIES

School of Nursing

We accept this thesis as conforming to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

April 2003

© Michelle A. House, 2003
In presenting this thesis in partial fulfilment of the requirements for an advanced degree at the University of British Columbia, I agree that the Library shall make it freely available for reference and study. I further agree that permission for extensive copying of this thesis for scholarly purposes may be granted by the head of my department or by his or her representatives. It is understood that copying or publication of this thesis for financial gain shall not be allowed without my written permission.

Faculty of Graduate Studies

Department of \underline{School of Nursing}

The University of British Columbia

Vancouver, Canada

Date \underline{April 11, 2003}

http://www.library.ubc.ca/spcoll/thesauth.html
ABSTRACT

A preliminary study was designed to assess the feasibility of testing the effect of earplug use, as a nursing intervention, on the sleep of critically ill patients using polysomnography. This study was done as a necessary first step in determining the feasibility of a larger, adequately powered study testing the same intervention as a cost-effective, non-invasive means of promoting sleep in the critically ill population. A single subject repeated measures design was used, and subjects served as their own controls. Two participants completed the study protocol.

Analysis of the study data resulted in a number of recommendations for future studies. First, it was demonstrated that a larger study testing the effect of earplug use in critically ill adults is needed, and that it is feasible to conduct such a study if sufficient funding is obtained. It is also suggested that the use of a research facilitator be considered to resolve some of the barriers encountered in recruitment of subjects, and to also overcome some of the logistical issues with nursing and medical support for the project. Access to interpretive services needs to be available for such a study to be successfully implemented in the same setting. It is further recommended that noise levels are measured concurrently with the polysomnographic sleep measurement. Planning for a future study must include careful consideration of eligibility criteria. Specifically, it is recommended that an upper limit of morphine equivalents of 30 micrograms per kilogram per hour be set. Finally, qualitative data regarding the participants' subjective experience of sleep should be collected to further inform the study.
TABLE OF CONTENTS

Abstract ........................................................................................................................... ii

List of Tables ................................................................................................................ vii

List of Figures ............................................................................................................. viii

Acknowledgements ....................................................................................................... ix

Dedication ...................................................................................................................... xi

CHAPTER I INTRODUCTION

1.1 Sleep is Necessary for Critically Ill Patients ......................................................... 1

1.2 Scope of the Problem ............................................................................................ 1

1.3 Purpose of the Study ............................................................................................ 3
  1.3.1 Objectives ......................................................................................................... 3

1.4 Significance ........................................................................................................... 4

1.5 Study Overview ..................................................................................................... 4

CHAPTER II LITERATURE REVIEW

2.1 Sleep: What It Is ..................................................................................................... 6
  2.1.1 Sleep Physiology .............................................................................................. 8
  2.1.2 Sleep Architecture ......................................................................................... 10
  2.1.3 The Terminology of Sleep Research ............................................................... 14

2.2 Rationale For Sleep .............................................................................................. 15
  2.2.1 Adaptive Theory and Restorative Theory ...................................................... 15

2.3 Sleep Deprivation .................................................................................................. 18
  2.3.1 Recovery Sleep .............................................................................................. 21
  2.3.2 Effects of Sleep Deprivation ......................................................................... 21
  2.3.3 Physiological Effects ...................................................................................... 23
  2.3.4 Psychological Effects ...................................................................................... 32

2.4 Ill People and Sleep ............................................................................................... 35
  2.4.1 The Effect of Illness on Sleep ....................................................................... 35
  2.4.2 Critical Illness: Effect on Sleep ................................................................... 36
2.5 Burden of Sleep Deprivation in Critical Illness ......................................................... 38

2.6 Stressors that Affect Sleep in Critically Ill Patients .................................................. 40
  2.6.1 Personal Factors ..................................................................................................... 40
  2.6.2 Environmental Factors ....................................................................................... 43
  2.6.2 Pharmaceuticals ..................................................................................................... 49

2.7 Sleep Measurement ..................................................................................................... 54
  2.7.1 Polysomnography ............................................................................................... 54
  2.7.2 Actigraphy ............................................................................................................ 55
  2.7.3 Observation ........................................................................................................... 56
  2.7.4 Patients' Perceptions ......................................................................................... 57

2.8 Interventional Studies ................................................................................................. 58

2.9 Summary .................................................................................................................... 63

CHAPTER III

METHODS

3.1 Theoretical Background .............................................................................................. 65

3.2 Assumptions ............................................................................................................... 66

3.3 Research Questions .................................................................................................... 66

3.4 Research Design ......................................................................................................... 67

3.5 Instrumentation .......................................................................................................... 68

3.6 Sampling and Data Collection .................................................................................... 71
  3.6.1 Setting .................................................................................................................. 73

3.7 Operationalization of Variables .................................................................................. 74
  3.7.1 The Use of Earplugs .......................................................................................... 74
  3.7.2 Sleep Parameters ............................................................................................... 75

3.8 Analysis ....................................................................................................................... 77

3.9 Limitations .................................................................................................................. 77

3.10 Ethical Considerations ............................................................................................... 78

3.11 Summary .................................................................................................................... 79
List of Tables

Table 1. Sleep Quantity: Jane Doe.................................................................87
Table 2. Sleep Quality: Jane Doe.................................................................88
Table 3. Sleep Quantity Expressed in Minutes: Jane Doe............................89
Table 4. Sleep Quantity: John Doe..............................................................97
Table 5. Sleep Quality: John Doe...............................................................98
Table 6. Sleep Quality Expressed in Minutes: John Doe...............................99
List of Figures

Figure 1. The Cyclic Nature of Sleep .................................................................12
Figure 2. Schematic Diagram of the 10-20 Electrode Placement System ...........70
Figure 3. Room Layout of the Main Intensive Care Unit .............................74
ACKNOWLEDGEMENTS

Like many projects of this nature, the contributions of many made the final product possible. I am indebted to the participants and their families for their willingness to participate in this study at a very stressful and upsetting time in their lives. This project was made possible by their generosity of spirit.

My heartfelt thanks are extended to my thesis chairperson, Dr. Pamela A. Ratner, for all the time, energy, and support she freely gave in mentoring me in the research process. I also want to express my appreciation to the other members of my thesis committee, Dr. B. Ann Hilton, and Dr. Joy L. Johnson for their expertise and help with this project.

I must acknowledge with gratitude the technical assistance and advice provided by Ron Polischuk, RPSGT, and Gordon Handford, Program Head, Neurodiagnostics, BCIT. Dr. John Fenwick and Harriet Tholin deserve thanks for their support in promoting nursing research in the Vancouver General Hospital Main Intensive Care Unit. I must also gratefully acknowledge Dr. Jane DeLemos for her kind assistance in participant recruitment and expert advice on pharmacological matters. Maureen Byrne also deserves thanks for her help in obtaining various equipment, as well as for her kind words of support. I must also gratefully acknowledge Jocelyn Reimer-Kent, RN, MSN, CNS, for sharing her ideas and stimulating some of the questions this study was undertaken to answer.

Thanks are due as well to my thesis support group for their never ending encouragement in this undertaking. Pam Munro, Linda Bachmann, Jacquie Wrightson, Su-Jin Chang, Chris Emery, and Peggy Wyatt were instrumental to the completion of this project. Thanks are also due to my family, particularly my husband, Peter Kokan, and
my mother, Irene House, for their ongoing encouragement in my endeavours. From word processing assistance to testing equipment to childcare, my family has provided extraordinary support that has made this work possible.

I wish to acknowledge as well the generosity of Stellate Systems in providing polysomnography equipment for this project. I especially thank Dr. Rajeev Agarwal of Stellate Systems for his assistance. This research project was also supported in part by the Katherine McMillan Director’s Discretionary Fund Research Bursary (University of British Columbia, School of Nursing), and by the Registered Nurses’ Foundation Research Bursary.
Dedicated to my husband, Peter Kokan, for his never-ending patience and support, and most of all, for his faith in me.
CHAPTER 1: INTRODUCTION

Sleep is Necessary for Critically Ill Patients

Each tissue cell and thus, all body organs, require a supply of oxygen to execute their functions. In critical illness, patients experience potentially life-threatening alterations in cellular oxygenation, resulting in cells being partially or completely unable to fulfill their purpose. Left uncorrected, these alterations can have devastating effects on critically ill patients’ morbidity and mortality. According to restorative theory, sleep is required to promote physical healing within the body (Oswald, 1978; Zepelin, 2000). Critically ill patients, however, suffer extensive sleep deprivation (Freedman, Kotzer, & Schwab, 1999; Hilton, 1976; McFadden & Giblin, 1971; Russell, 1999; Simini, 1999; Woods, 1972). The experience of sleep deprivation can directly and indirectly affect almost every aspect of a patient’s cellular oxygenation. Sleep deprivation has been linked to impaired healing, decreased immunocompetence, and the development of intensive care unit (ICU) psychosis. Sleep deprivation may interfere with the process of weaning from mechanical ventilation, a process vital to recovery and discharge from the intensive care unit (Schwab, 1994). There are numerous obstacles to sleep in the critical care environment and within patients themselves. This knowledge challenges healthcare providers to seek effective means to ensure that critically ill patients attain enough sleep.

Scope of the Problem

Sleep is a complex, multidimensional process that has been characterized as an essential human need (Carskadon & Dement, 2000; Roy, 1976; Watson, 1985). Despite this characterization, the exact physiology and functions of sleep remain largely unknown (Bonnet, 2000). It is known that sleep deprivation is not an uncommon occurrence in the
general population (Coren, 1996), and it is now clear that sleep deprivation is common in hospitalized patients as well. Numerous studies that have investigated patients' experiences in the ICU indicate that sleep disruption and deprivation is a consistent occurrence, and has been for many years (Freedman et al., 1999; Hilton, 1976; McFadden & Giblin, 1971; Russell, 1999; Simini, 1999; Woods, 1972). Almost all studies have confirmed that regardless of medical diagnosis, the majority of patients in the ICU in whom sleep can be measured have extremely fragmented and disturbed sleep (Culpepper-Richards, 1994). Gabor, Cooper, and Hanly (2001) suggest that sleep disruption is an ubiquitous phenomenon common to all types of intensive care units that affects most, if not all, critically ill patients.

Sleep deprivation is an issue that has major implications for critically ill patients. These include both physiologic and psychologic effects that can increase morbidity and possibly mortality (Redeker, 2000). It is likely that sleep deprivation in critically ill patients contributes to a longer length of stay (Freedman et al., 1999; Gelling, 1999; Schwab, 1994).

The American Association of Critical Care Nurses and the Canadian Association of Critical Care Nurses have both identified sleep deprivation in patients as a research priority (P. Price, personal communication, January 29, 2002). The issues of sleep disruption and deprivation have more recently received serious consideration from the critical care medical community in Canada (P. Hanly, personal communication, November 25, 2001). Research efforts have determined that there are a multitude of factors that disrupt sleep in critically ill patients. One of the major factors contributing to sleep deprivation in this population is environmental noise (Freedman et al., 1999;
Despite this knowledge, there are very few studies that have tested the efficacy of various interventions designed to improve the sleep of critically ill patients, and even fewer that specifically address the effects of noise on sleep in critically ill patients. Given the potential implications of sleep deprivation in critically ill patients, as well as the complexity of critical care units and the patients themselves, a feasibility study was proposed to explore the possibility of studying or implementing an intervention to reduce noise.

**Purpose of the Study**

The primary purpose of the study was to examine the feasibility of studying the effect of earplug use on the sleep of critically ill patients. As a feasibility assessment, this study also sought to distinguish the value of a larger future study, with the broad aim of improving the sleep of critically ill patients. Secondarily, this study was designed to explore the impact of this intervention for a presumed sleep disrupter, noise, on the sleep of patients in the intensive care environment.

**Objectives**

This study was intended to determine the feasibility of earplug use on the sleep of critically ill patients, with the long term goal of helping to improve their sleep. As a feasibility study, the researcher sought to gain an understanding of the suitability of the proposed intervention (earplug use), and the possibilities and potential difficulties associated with the selected data collection method as the primary component of the study. The researcher also intended to identify problems associated with the administration of the study protocol in this population and setting. Furthermore, the
researcher hoped to gain an understanding of the projected costs involved with completing such a study with a larger sample.

Significance

Given the potential effects of sleep deprivation on morbidity in critically ill patients, it is reasonable to infer that a prolongation in stay in critical care may result. This may impact patient outcomes, and certainly will impact health care costs. It is thus clear that cost-efficient, practical interventions that promote sleep in critically ill patients are needed.

The results of this study provide the first steps toward studying the effect of earplug use on the sleep of critically ill patients, and identify methodological issues that need to be resolved to best do so. The results provide insight into the practicality of using a cost-efficient, nurse-managed intervention to promote sleep in critically ill patients. The results of this study, however, are otherwise limited. As a feasibility study, the sample was not large enough to provide sufficient power to detect a statistically significant effect. Furthermore, this study design could not provide an in-depth understanding of critically ill patients' subjective experience of the quantity and quality of their sleep.

Study Overview

Like many other projects of this nature, this study involved a complex series of steps in its evolvement and completion. To facilitate its presentation, a brief overview of its entirety is described.

A comprehensive literature review is presented in Chapter 2. This literature review describes the concept of sleep, including its physiology and unique architecture and its functions as they are currently understood. The review describes the effects of sleep
deprivation, and presents the factors that are understood to affect sleep in critically ill adults. The measurement of sleep is described, and studies that have attempted interventions to affect sleep are presented and discussed.

The methods used in this project are discussed in Chapter 3. A conceptual framework of stress-related sleep problems that guided the development of the research design and research questions is presented, as are the assumptions that informed the research approach. The research design itself is described, including the operationalization of variables, instrumentation, sampling and data collection. The analysis of the data is then presented. Limitations of this study are identified and ethical considerations are discussed.

The results of this study are presented in case study format in Chapter 4. Each case is described and discussed separately, then the findings of the data collection are presented as they relate to the research questions.

An overall assessment of the feasibility of a larger study of this type is discussed in detail in Chapter 5. Included in this discussion are directions and suggestions for future research, and implications for nursing practice.
CHAPTER II: LITERATURE REVIEW

A search for published literature related to sleep and critical illness was carried out using the Medline, Web of Science, Cumulative Index of Nursing and Allied Health Literature (CINAHL), and EMBASE data bases. The search included English documents published between 1960 and 2002. Canadian and American government internet sites were accessed for statistical information. Authors of various relevant publications were contacted for direction and advice in the literature search process. Manual searching of relevant publications was also used as a search strategy. The following key words and key word combinations were used in the literature searches: sleep, critical illness, critical care, intensive care, sleep deprivation, polysomnography, mechanical ventilation, intensive care unit syndrome, intensive care unit psychosis, delirium, pharmacology, noise, and environment.

This review of the literature describes the current understanding of the phenomenon of sleep, including its physiology and architecture, and its physiological and psychological functions. The effects of sleep deprivation in healthy people are described and contextualized to critically ill adults. Factors that affect sleep are identified, and are further discussed within the context of critical illness. The techniques available for the measurement of sleep are described and compared. Finally, studies that were conducted to test the effects of various interventions on sleep are described and discussed.

Sleep: What It Is

Sleep has fascinated humans since the beginning of recorded time. The early Greeks distinguished human beings from deities on the basis of the need for sleep. The reason for
this was based on the idea of the eternal vigilance of the gods, as compared to the faltering of human beings while in sleep (Brewer, 1985).

Historically, sleep was conceptualized as a passive state. Until relatively recently, sleep was almost universally regarded as an inactive state of the brain; an inevitable result of reduced sensory input with consequent diminishment of brain activity. Waking was considered a simple reversal of this process, mostly as a result of bombardment of the brain by environmental stimulation. No real distinction was made between sleep and other states of dormancy such as coma, stupor, intoxication, hibernation or anesthesia (Dement, 2000). This conceptualization is illustrated in the first sentence of The Philosophy of Sleep, a book published in 1834 by one of the members of the faculty of physicians and surgeons of Glasgow:

Sleep is the intermediate state between wakefulness and death; wakefulness being regarded as the active state of all the animal and intellectual functions, and death as that of their total suspension. (MacNish [1834] as cited in Dement, 2000, p. 1)

Since the 1930s, the study of sleep has advanced markedly, particularly with the advent of electroencephalography (EEG). With these advancements, the current understanding of sleep has changed dramatically from seeing it as a passive state to one that is active. Sleep is now viewed as a complex, multidimensional process as opposed to a state of being. Sleep is cyclical, reversible, and characterized by a decrease in the levels of “cortical vigilance” when compared with wakefulness (Koella, 1978). Nursing theorists have categorized sleep as a universal, essential human need (Roy, 1976; Watson, 1985). Sleep appears to be a complex combination of physiological and behavioural processes that are dynamic in nature. A simple behavioural definition of sleep is offered by Carskadon and Dement (2000):
Sleep is a reversible behavioral state of perceptual disengagement from and unresponsiveness to the environment... and is usually (but not necessarily) accompanied by postural recumbancy, quiescence, closed eyes, and all the other indicators one commonly associates with sleeping. (p.15)

**Sleep Physiology**

The exact physiology of sleep is not well understood. Various neurologic structures and substances are known to be involved, but the exact role each plays and the interactions between them, are not known. The reticular activating system (RAS) extends from the brainstem up to and including the thalami, and is involved in producing the complex changes of sleep and wakefulness. Increased activity in the RAS stimulates other parts of the brain, creating a state of wakefulness. The RAS acts as a relay and screening station for impulses en route to the cerebral cortex. It also sends impulses down the spinal cord to increase the tone in all the muscles of the body. Increased muscle tone, in turn, initiates sensory impulses that excite the RAS. The degree of activation of the RAS determines the degree of wakefulness. Activation may also be initiated and reinforced by visceral, somatic, and special sensory input (Hilton, 1976; Jones, 2000).

The raphe nuclei are a thin sheet of nuclei located in the lower half of the pons and the medulla. Nerve fibres from these nuclei spread through the RAS and upward into the thalamus, neocortex, hypothalamus, and most areas of the limbic system. They also extend downward into the spinal cord. When these neurons are stimulated, many of their fibre endings secrete the neurotransmitter serotonin, and sleep results (Guyton & Hall, 1996).
Sleep is also promoted by stimulation of the sensory region of the medulla and pons by visceral sensory signals from the vagus and glossopharyngeal nerves. However, this does not occur if the raphe nuclei have been destroyed. This has led to the speculation that these regions act by exciting the raphe nuclei and the serotonin system (Guyton & Hall, 1996).

Stimulation of several regions in the diencephalon also promote sleep. These areas include the rostral part of the hypothalamus, mainly in the suprachiasmal area, and some areas in the diffuse nuclei of the thalamus (Guyton & Hall, 1996).

Numerous neurotransmitters have been implicated in the regulation of vigilance and wakefulness. Those most examined are serotonin, norepinephrine, and acetylcholine, although research continues to identify biologic substances with potential sleep generation capabilities. Some of these recently identified substances include gamma-aminobutyric acid, dopamine, melatonin, and various hypnogenic peptides (Fontaine, 1996). Neurotransmitters involved in the sleep-wake process can be characterized as vigilance enhancers, which promote wakefulness, or vigilance suppressors, which elicit sleep (Koella, 1985). Serotonin appears to be important throughout the entire sleep-wake cycle, while acetylcholine is released during rapid eye movement sleep at the same level as during the awake state. The vigilant awake state is under the control of the norepinephrine adrenergic system, which discharges from the locus ceruleus. The interaction among and modulation of these neurotransmitters permit the progression of sleep and waking stages (Fontaine, 1996).
Sleep Architecture

Two separate states within sleep have been defined on the basis of a number of physiological parameters. These two states are nonrapid eye movement (NREM) and rapid eye movement (REM) sleep, and each are as distinct from the other as each is from wakefulness (Carskadon & Dement, 2000).

NREM sleep is divided into four stages on the basis of electroencephalogram (EEG) waveforms. These four stages roughly parallel a depth of sleep continuum, based on "arousal thresholds". Arousal thresholds refer to the minimum amount of stimulation required to shift sleep toward wakening. The arousal thresholds are lowest in stage 1 ("lightest" sleep) and highest in stage 4 ("deepest" sleep). NREM sleep is usually associated with fragmented mental activity. It has been described as a sleep state in which there is a relatively inactive yet actively regulating brain, in a movable body (Carskadon & Dement, 2000). Adults generally enter sleep through NREM stage 1 sleep. This is a transitional, lighter stage of sleep state from which one can be easily aroused by a light touch or by the soft calling of one's name. Stage 1 comprises about 2 to 5% of a night's sleep, and is demonstrated by an EEG pattern of low-voltage, mixed-frequency waveforms with sharp vertex waves. As a person makes the transition from being awake to asleep, brief memory impairment may result. People will sometimes experience muscle jerks, called hypnic myoclonia, and recall vivid images upon awakening from this stage (Urden, Stacy, & Lough, 2002).

Stage 2 NREM sleep accounts for about 45-55% of sleep during a night, with sleep deepening and a higher arousal threshold being required to awaken the person. The EEG pattern may show sleep spindles and K complexes. As this stage continues, the EEG will
start to show high-voltage, slow-wave activity. When slow waves account for 20% of the EEG per page, the criteria for stage 3 sleep are met. Stage 3 generally constitutes 3-8% of sleep. Stage 3 sleep usually lasts only a few minutes, and is transitional to stage 4 as more high-voltage, slow wave activity occurs. Stage 4 NREM sleep is identified when this EEG activity is more than 50% of the record. At this stage, which generally constitutes about 10-15% of sleep, a larger stimulus is required to produce an arousal. Sleep researchers often refer to the combined stages of 3 and 4 sleep as slow wave sleep (SWS), delta sleep, or deep sleep (Carskadon & Dement, 2000; Urden et al., 2002).

In contrast, REM sleep is defined by EEG activation, muscle atonia, and episodic bursts of rapid eye movements. It is generally not divided into stages. The mental activity of human REM sleep is associated with dreaming, based on vivid dream recall after arousal from this state of sleep, although dreaming is not the exclusive property of any one stage. There is inhibition of spinal motoneurons via brainstem mechanisms. This mediates suppression of postural motor tonus in REM sleep. A shorthand definition of REM sleep is one in which there is a highly activated brain in a paralyzed body (Carskadon & Dement, 2000). It is sometimes referred to as paradoxical sleep, where the paradox is that some areas of the brain remain very active whereas others are suppressed. EEG waveforms are relatively slow voltage with sawtooth waves present. Increased cortical activity is present, with the EEG pattern resembling those of the awake state. Synchronized bursts of rapid side to side eye movements with muscle atonia are seen. REM sleep generally accounts for about 20-25% of sleep, and occurs in four to six discrete episodes (Carskadon & Dement, 2000; Urden et al., 2002).
Sleep Cycles.

NREM and REM sleep stages alternate throughout the night in a predictable fashion. Most normal, healthy, individuals have a mean sleep latency, or time to fall asleep, of about 10 to 20 minutes, with sleep onset usually occurring in stage 1 sleep. Sleep then progresses through stages 2, 3, and 4, then returns to stage 2, at which point the person usually enters REM sleep. After a period of REM sleep, the person will normally move back into stage 2 sleep, and then will progress again through stages 2, 3, 4, and REM. Figure 1 depicts a typical sleep cycle.

![Diagram](image)

*Figure 1. The cyclic nature of sleep. Note. From Thelan’s Critical Care Nursing: Diagnosis and Management (4th ed., p.79) by Urden, L., Stacy, K., & Lough, M., 2002, St. Louis: Mosby. Copyright 2002 by Elsevier.*

This first cycle takes about 70 to 100 minutes, with subsequent cycles lasting 90 to 120 minutes. Four to five cycles are completed during normal adult sleep. NREM predominates during the first third of the night, and REM predominates in the last third.
In older adults, sleep pattern changes include fewer episodes of stage 3 and 4 NREM and REM sleep (Carskadon & Dement, 2000; Krachman, D’Alonzo, & Criner, 1995; Urden et al., 2002).

Most young adults sleep approximately 7.5 to 8 hours per night, yet the variability is very high from person to person, and even from night to night (Carskadon & Dement, 2000). Most adults are able to maintain daytime performance and alertness when their sleep is decreased to not less than five hours per night (Horne, 1988). However, besides quantity, continuity of sleep is also important in maintaining daytime alertness and task performance (Carskadon & Dement, 2000).

Circadian Rhythms.

Many body systems cycle within approximately a 24-hour period, thus they are called circadian rhythms (“circa” means “about”, “dia” means “day”). Some of these body systems include hormone release, blood cell production, body temperature, and the sleep-wake rhythm. A bundle of cells in the anterior hypothalamus, known as the suprachiasmatic nucleus, functions as the pacemaker for these rhythms. These rhythms will persist with near 24-hour time lengths in time-free environments (e.g., laboratory settings), but normally are synchronized to the 24-hour day by environmental cues and stimuli, referred to as zeitgebers, or “time givers” (Mistlberger & Rusak, 2000). Zeitgebers for the sleep-wake rhythm include posture, exercise, temperature, social stimuli, and light, with light being the most influential. The process of synchronization involves daily zeitgeber-induced shifts that compensate for the difference between the intrinsic period of the pacemaker and the period of the environmental cycle. For example, if people are left to sleep and awaken whenever they choose, in an environment
free of day-night cues (e.g., a laboratory), over a period of time they will extend their sleep-wake cycle to approximately 25 hours instead of 24. They will gradually go to sleep later and later, and awaken later and later (Mistlberger & Rusak, 2000; Urden et al., 2002).

Normally, a person’s rhythms interact and influence one another. A person, for example, is more likely to sleep during the part of the body temperature cycle when it is lowering, and conversely, will tend to awaken as body temperature rises in the early morning hours (Czeisler & Khalsa, 2000). Aging on the other hand, affects circadian rhythms in terms of length, amplitude, and response to zeitgebers, which may disrupt circadian temporal organization, causing elders to become sleepy during the early evening and awaken during the early morning (Mistlberger & Rusak, 2000).

The Terminology of Sleep Research

There are many terms that are specific to the study of sleep that the reader may encounter while reading this document. Definitions of sleep terminology used throughout this document, were adapted from Williams, Karacan, and Hursch (1974).

*Latency to sleep onset:* the difference between the time a patient is quiescent and could possibly sleep, and the onset of the first sleep stage.

*Sleep period time (SPT):* the time from onset of stage 1 sleep until the final morning awakening. Sleep period time also includes any time spent awake after the onset of stage 1 sleep until the final morning awakening. The periods of REM sleep, waking stage, and sleep stages 1 through 4 are calculated as percentages of SPT.

*Stage shifts:* the number of changes from any sleep stage to another sleep stage.
Total sleep time (TST): total time spent, over the course of the sleep period, in any of the sleep stages. The total sleep time does not include any time spent awake during the sleep period time.

Awakenings: the return from any sleep stage to the waking stage.

Sleep efficiency index (SEI): percentage of time in bed spent asleep in any stage. Because ICU patients are usually on a regimen of bedrest, this definition was modified for the purpose of this study, to define time in bed as beginning at 2200 hours and ending at 0600 hours.

Arousals: a shift toward a wakening from any sleep stage; a shift from any sleep stage to a “lighter” one.

Rationale for Sleep

The overall function of sleep in humans remains largely unknown. Historically, the function of sleep has been the subject of much debate and discourse among scientists. However, despite extensive research over time, a definitive answer to this question has not been found. Sleep is generally accepted as a basic human need (Roy, 1976; Watson, 1985), but the purpose of this need remains elusive.

Adaptive Theory and Restorative Theory

There are two main theories of the function of sleep, the adaptive theory and the restorative theory. The adaptive theory asserts that sleep is an automatic, inherited response to conditions in our environment, based on the criteria of an inherent biological system (Webb, 1975). These criteria include being unlearned and developmental in nature, and specific to each species. It also must be self-perpetuating, and adaptive in relation to the organism’s environment.
The adaptive theory suggests that humans sleep to avoid exhaustion. Basically, this “non-behaviour” prevents any waste of energy through unnecessary moving about, and keeps humans and other mammals out of harm’s way. It also occupies the otherwise nonproductive hours of darkness. The problem with this theory is that although it fits the behaviour of sleep, it fails to explain the existence of the different types of sleep that occur during a typical sleep cycle (Horne, 1988; McGonigal, 1986).

The restorative theory, in contrast, suggests that sleep is a time when resources that are depleted during waking hours are replenished (Oswald, 1978; Zepelin, 2000). This theory differentiates between REM and NREM sleep, and correlates changes in physical parameters during sleep with the function of the specific stage. This theoretical view appears to be more frequently used as an assumptive basis for research and understanding of sleep.

**NREM sleep functions.**

Within the restorative theory of sleep function, the promotion of physical healing and growth is associated with NREM sleep (Evans & French, 1995; Hartmann, 1973; Urden et al., 2002). The parasympathetic nervous system dominates NREM sleep (Urden et al., 2002). Heart rate, cardiac output, and body temperature all fall during Stage 4 sleep (McGonigal, 1986), reducing tissue oxygen demand and consequently, metabolic rate. Levels of human growth hormone are elevated during NREM sleep (Sassin et al., 1969; Takahashi, Kipnis, & Daughaday, 1968; Van Cauter, 2000). Human growth hormone promotes protein anabolism, or the building of tissues, by rapidly stimulating protein and RNA synthesis and amino acid uptake by the cells, as well as promoting the conversion of glycogen to glucose (Krachman et al., 1995). Protein anabolism results in cell repair,
growth, and replication. Approximately 70% of the total 24-hour secretion of human growth hormone occurs during Stage 3 and Stage 4 sleep (Lee & Stotts, 1990). Prolactin, testosterone, and luteinizing hormone plasma levels are also increased during NREM sleep. These and other hormones have a role in protein anabolism (McGonigal, 1986; Urden et al., 2002).

Conversely, corticosteroids are endogenous substances that promote protein catabolism, or tissue breakdown. Levels of the corticosteroids peak and trough over the course of a 24-hour cycle, with a great reduction occurring around 2300 hours to 0200 hours. This coincides with the period when most Stages 3 and 4 sleep are taken (Akerstedt & Levi, 1978).

Epinephrine increases heart rate and contractility, which increase myocardial oxygen demand. Increased myocardial oxygen demand subsequently increases overall body oxygen demand. The secretion of this catecholamine is reduced during NREM sleep (Akerstedt & Levi, 1978). Thyroid stimulating hormone is also decreased during NREM sleep (Van Cauter, 2000).

The combination of decreased tissue oxygen demand and decreased metabolic rate, along with the described hormone changes, all suggest that NREM sleep is a time when protein anabolism in body tissues is promoted. Not only is a higher amount of cellular division occurring during sleep, but also, it takes a cell less than half the time to divide during sleep as compared to during wakefulness (Krachman et al., 1995). Further support for this viewpoint is provided by the observation that the amount of Stage 4 sleep increases during periods of physical stress, such as increased exercise, fasting, or
increased thyroid hormone production (Hartmann, 1973; McGonigal, 1986; Oswald, 1978).

**REM sleep functions.**

Mental and emotional restoration are broadly associated with REM sleep (Hartmann, 1973). In this type of sleep, the sympathetic nervous system dominates (Urden et al., 2002). Hormonal secretion during REM sleep continues to facilitate protein anabolism, but cerebral blood flow, intracranial pressure, heart rate, cardiac output, systolic blood pressure, and total oxygen consumption, which consequently lead to a large increase in blood flow to the brain, are all increased relative to NREM sleep. In fact, heart rate, respiration, and blood pressure can be highly variable in REM sleep, and can exhibit transient irregularities (Evans & French, 1995; Gabor et al., 2001; Krieger, 2000). The view that REM sleep facilitates the growth, repair, and development of brain tissues is supported by the observation that REM sleep predominates during neonatal and infant sleep, when the brain is in its most rapid period of growth and development (Urden et al.).

REM sleep also appears to be necessary for learning (Dracup & Bryan-Brown, 2000). Brown (1999) discovered that decreased REM sleep leads to impaired memory for cognitive or procedural material. According to Hartmann (1973), increased mental or emotional activity results in an increased need for sleep.

**Sleep Deprivation**

Although there is published evidence to support the restorative theory of sleep, exacting theories concerning sleep functions are limited. Most of the current literature and theories of sleep function are based not on sleep per se, but on studies in which
subjects have not had enough sleep. These studies have sought to gain a deeper understanding of the functions of sleep by analyzing what happens physically and psychologically to humans when they experience sleep deprivation.

Sleep problems and sleep deprivation are very common. On average, about one third of the American population get inadequate sleep, and approximately 10% to 20% experience chronic insomnia (Bonnet & Arand, 1995; Dracup & Bryan-Brown, 2000; National Heart, Lung, and Blood Institute Working Group on Insomnia, 1999). In his popular book, The Sleep Thieves, Coren (1996) contends that most individuals living within Western cultural norms are sleep-deprived to a certain extent.

There are essentially three specific types of sleep deprivation. These include total sleep deprivation, partial sleep deprivation, and selective sleep deprivation. Total sleep deprivation occurs when no sleep is obtained during at least one 24-hour cycle. If a person who usually sleeps from 2200 to 0600 hours awakens one morning at 0600, then stays awake until 2200 of the following day, that person has had 40 hours without sleep. Precisely speaking, that person has experienced eight hours of sleep deprivation. However, in general practice, the hours of continuous wakefulness, which in this example are 40 hours, are used as the amount of total sleep deprivation (Naitoh, Kelly, & Englund, 1990). Partial sleep deprivation is particularly common; most people have experienced it at some time in their lives. Partial sleep deprivation means sleeping less than usual, whether it is sudden (e.g., sleeping only five hours instead of a customary eight), or gradual (e.g., reducing sleep by a half hour each week) (Naitoh et al., 1990). Selective sleep deprivation does not occur in real world situations. It is created as a result of manipulation of sleep in laboratory settings, where changes in sleep stages can be
observed as they occur. Selective sleep deprivation entails the prevention of the occurrence of a particular sleep stage, while having minimal impact on total sleep time and other sleep stages (Bonnet, 2000; Naitoh et al.). For example, in REM sleep deprivation, sleepers are prevented from obtaining REM sleep by being awakened whenever they begin to enter that stage of sleep. In real life, the sleep deprivation that we typically experience is likely to be a mixture of these three types (Naitoh et al.). For example, we may experience both partial sleep deprivation and REM sleep deprivation when we awake a couple hours earlier than usual.

Sleep fragmentation is another sleep problem that may contribute to sleep deprivation. It refers to the interruption of sleep at various times during the night that prevents the individual from completing an entire sleep cycle or the normal sequence of sleep stages (Evans & French, 1995). Sleep fragmentation may incur arousals notable only by EEG, or may incur actual awakenings (Bonnet, 2000). Because sleep is a time-based cumulative process, frequent arousals or awakenings can slow or stop the process in an orderly fashion (Bonnet, 2000). With each awakening, some sleep is lost, and since it may take one half hour or more to sink back into Stage 3 or 4 sleep, it is more difficult to accumulate slow-wave sleep (Coren, 1996). Thus it may be assumed that sleep achieved in fragments is less restorative than non-fragmented sleep. In addition, it is theorized that sleep continuity is as important as total sleep time. Research in this area suggests that decrements in daytime function increase as the length of periods of consolidated sleep decrease (Bonnet, 1989; Freedman, Gazendam, Levan, Pack, & Schwab, 2000).

Sleep fragmentation is not uncommon during various life events (e.g., infant care), but also can be the result of various medical conditions, such as chronic pain. Most
of these situations are a combination of both chronic partial sleep deprivation and chronic sleep fragmentation, and the combination of these factors will have a greater impact than either factor in isolation (Bonnet, 2000).

**Recovery Sleep**

In almost all circumstances, only sleep is required to reverse the effects of sleep deprivation (Bonnet, 2000). After total sleep deprivation, there is a large increase in slow wave sleep over baseline amounts, usually at the expense of stages 1, 2, and REM sleep. This usually occurs during the first night of recovery sleep. By the second night of recovery sleep, slow wave sleep approaches baseline values, and REM sleep rebounds (Bonnet). Total sleep time is increased during recovery, but not to the extent of the amount of sleep lost (Horne, 1988). For example, if left undisturbed, young adults will sleep typically only 12 to 15 hours, even after 264 hours of sleep loss (Bonnet). In selective sleep deprivation, recovery sleep results in a rebound in the type of sleep that was lost (Naitoh et al., 1990).

**Effects of Sleep Deprivation**

The researched effects of sleep deprivation are somewhat contradictory. It is commonly believed that chronic sleep deprivation may shorten life span or increase morbidity (Naitoh et al., 1990). However, most people in Western countries have experienced sleep deprivation at some point. Many ordinary life events, such as working to meet a deadline, earning a living as a shiftworker, or managing a family, can lead to temporary, total, or partial sleep deprivation. A report prepared for the United States National Commission on Sleep Disorders states that in 1988 alone, the total cost of sleep related accidents, including motor vehicle, public transportation, and work-place
accidents, as well as accidents in public places (such as falls), and accidents around the home due to sleepiness, was $56.02 billion (Leger, 1994). In that same year, a total of 24,318 deaths resulted from accidents due to sleepiness (Coren, 1996; Leger, 1994). These numbers, astounding as they are, do not take into account injuries that did not result in death due to sleep-related accidents, nor do they provide an estimate of lost productivity in terms of lost workdays, or long term impacts, such as pain or other complications (Coren, 1996).

Several animal studies on sleep deprivation, most involving rats, suggest that sleep deprivation might actually result in death. Rats subjected to total sleep deprivation or to selective REM sleep deprivation died in three and five weeks, respectively (Bonnet, 2000; Naitoh et al., 1990). With these findings, it has been speculated that on the basis of energy requirements, humans would survive the loss of sleep “about 3.7 times longer than rats, or about 77 days of total sleep deprivation versus 21 days in rats” (Rechtschaffen, Gilliland, Bergmann, & Winter, 1983, p. 180). Horne (1988) however, raises doubts about generalizing the findings in rats to humans because of the differences in thermoregulatory mechanisms between the two species. He suggests that death in severely sleep deprived rats is due to thermoregulatory failure as opposed to sleep loss. He goes further to argue that thermoregulatory failure would not occur in humans because the larger human body has more adequate thermoregulatory mechanisms.

It has been noted that the majority of research on sleep deprivation is based on highly controlled laboratory studies using short-term data collection. Sleep loss in real life circumstances is usually far different from those conditions and thus challenges the credibility of these findings. Furthermore, most human studies involving the effects of
sleep deprivation have been performed in healthy people, who underwent a single stressor – sleep loss – for the purpose of the study. Many of the studies actually involved healthy, physically fit young people (Naitoh et al., 1990). These findings therefore, cannot be generalized to middle aged or older populations, and especially not to people with less than ideal physical or mental health, such as critically ill patients.

Despite the controversy over whether sleep deprivation can directly cause death or disability in humans, there are documented effects of sleep loss in humans. These effects can be broadly categorized as physiological or psychological. In some research, the physiological effects have been found to very “roughly” correlate with NREM sleep loss, and the psychological effects to very “roughly” correlate with REM sleep loss (Bonnet, 2000; Evans & French, 1995). However, most of these data involve total sleep loss (total loss of sleep over time regardless of sleep stage), so these generalizations about REM and NREM loss are not solid. Despite the questionable credibility of these groupings according to REM and NREM sleep, research does show that physiological and psychological effects occur in sleep deprivation. Furthermore, even though most sleep deprivation research has utilized animals or healthy volunteer subjects, it may be logical to deduce that these same effects would occur in critically ill people, and perhaps to an even greater degree (Gabor et al., 2001; Redeker, 2000; Schwab, 1994). What follows is a review of this literature and its relevance to sleep deprivation in the critically ill patient.

**Physiological Effects**

The physiological effects of sleep deprivation are myriad and involve a number of systems within the body. For ease of discussion, these are presented in terms of the body responses involved.
Adrenomedullary activity and sleep loss.

Two catecholamines are produced by the adrenomedullary system, epinephrine and norepinephrine. Both these substances act on all organs of the body causing "sympathetic activation", which refers to a group of effects that produce the emergency "fight or flight" physical stress response. These effects include increased heart rate, increased strength of cardiac contraction, arteriolar constriction in the skin, and both vasodilation and vasoconstriction of voluntary muscle arterioles. Blood pressure is raised, glucose and fat stores are mobilized for energy, and smooth muscles relax (Urden et al., 2002). According to an extensive review of research related to the effects of sleep deprivation (Naitoh et al., 1990), short term (less than three days) total sleep loss results in no significant increases in adrenomedullary activity, as long as the sleep deprivation takes place under physically and mentally nondemanding conditions. When conditions are more physically or mentally demanding, such as high workloads or mental stress, or entail uncomfortable environmental conditions, the adrenomedullary activity may become significantly elevated (Naitoh et al., 1990).

Critical illness is considered a physically and mentally "demanding" condition (Urden et al., 2002). As such, it can be expected that sleep deprivation in these patients would then activate the adrenomedullary system. Sympathetic activation and its resultant effects combine to produce a markedly increased overall oxygen demand within the body. In the critically ill patient, this can tip the tenuous balance between oxygen supply and demand, so that supply cannot meet demand, to the detriment of the end organs and tissues.
Metabolism and sleep loss.

Normally, body metabolism slows during sleep. Thus, during sleep deprivation, this energy conservation does not occur, resulting in greater metabolic expenditure (Bonnet, 2000). This is supported by findings that oxygen consumption and carbon dioxide production are both increased during sleep loss (Bonnet, Berry, & Arand, 1991). In studies of sleep deprivation with healthy young men, it was found that a negative nitrogen balance can occur as a result of sleep deprivation, indicating a change in protein metabolism (Gabor et al., 2001; Scrimshaw, Habicht, Pellet, Piche, & Cholakos, 1966). This may provide evidence of sleep deprivation being a catabolic state, where protein is used to supply increased energy needs (Naitoh et al., 1990). As sleep loss continues, skeletal muscles and other body tissues switch their main energy substrate from carbohydrates to lipids to meet energy needs, resulting in increased plasma free fatty acids, increased plasma glucose, and sluggish plasma glucose use (Naitoh et al.).

According to Naitoh et al., sleep deprivation is also reported to increase plasma levels of thyroid hormone, which increases the overall metabolic rate.

Greater metabolic expenditure results in increased oxygen demand, as well as greater oxygen consumption. In a critically ill patient who may not be able to increase his or her oxygen supply to meet this demand, the potential for impaired oxygen supply to the tissues and end organs is significant.

Adrenocortical activity and sleep loss.

The adrenocortical system produces glucocorticoids, mineralocorticoids, and androgenic steroids. In sleep deprivation, the glucocorticoid cortisol may be the most severely affected (Irwin et al., 1996). Cortisol functions to increase hepatic glycogenesis,
suppress protein synthesis, and provide an anti-insulin action at the peripheral tissues. Production of cortisol increases in response to stress, for example, illness or anxiety. These actions together provide the energy needed for the body to respond in prolonged stressful circumstances (Naitoh et al., 1990; Urden et al., 2002). In times of stress, cortisol also causes the mobilization of amino acids and fats from cells, enabling the damaged tissues to utilize these newly available substrates for repair. Cortisol causes the stabilization of lysosomal membranes in damaged cells and reduces inflammation (Evans & French, 1995; Guyton & Hall, 1996). Prolonged secretion of cortisol, however, reduces lymphocyte and granulocyte functions that result in an interference with the body’s ability to heal and prevent infection. Prolonged cortisol secretion also depresses T cell function, which leads to increased susceptibility to infection (Guyton & Hall, 1996).

During sleep, cortisol secretion is usually diminished (Van Cauter, 2000). This has led to the speculation that in sleep deprivation, cortisol secretion would lose its circadian periodicity (Irwin et al., 1996), and remain at least somewhat elevated, especially if the sleep loss is combined with other stressors such as illness (Van Cauter). Naitoh et al. (1990), however, found in their review of sleep deprivation studies that total sleep loss of up to 72 hours has little effect on cortisol. Again, it must be observed that these studies were primarily performed on young, healthy volunteers experiencing no other acute stressors other than sleep loss. It follows then that the exact impact of sleep deprivation on cortisol remains unclear, particularly within the critically ill patient.

**Autonomic activity and sleep loss.**

As stated previously, autonomic activity generally slows during NREM sleep, especially during slow wave sleep, then becomes highly variable during REM sleep.
This cyclical rhythmicity is disrupted during total sleep deprivation (Bonnet, 2000). Heart rate tends to increase in its variability, such as increasing fluctuations with respirations. Respiratory rate also increases in variability with sleep loss (Naitoh et al., 1990). These effects can result in episodes of hypoxemia, or insufficient oxygen supply to the tissues (Gabor et al., 2001). Total sleep deprivation can result in ST depression, which is an electrocardiographic change that indicates myocardial ischemia. This appears to reverse after several days of sleep and rest (Naitoh et al., 1990). Thermoregulation is disrupted; the mean core body temperature lowers during total sleep deprivation, and the circadian variation reduces (Landis, Savage, Lentz, & Brengelmann, 1997; Naitoh et al., 1990).

The negative implications of variable autonomic activity in critically ill patients are many. The irregularities, especially in heart rate, respiratory rate, and blood pressure, are potentially dangerous to a critically ill patient, especially if they are associated with an episode of hypoxemia. It has been suggested that the variability in autonomic activity associated with sleep loss may play a role in the etiology of postoperative myocardial ischemia and infarction (Gabor et al., 2001; Kavey & Altshuler, 1979; Knill, Moote, Skinner, & Rose, 1990). It has been suggested that critically ill patients are particularly prone to these effects during recovery sleep, when REM rebound, or a recovery in REM sleep, occurs (Gabor et al., 2001).

**Epileptiform discharges and sleep loss.**

Total sleep deprivation of 48 hours or less increases cerebral irritability, which may induce epileptiform discharges, or seizure-like brain electrical activity, in people with suspected epilepsy. Prolonged total sleep deprivation combined with other stressors may
also induce epileptiform discharges in healthy individuals (Bonnet, 2000; Naitoh et al., 1990). It has been suggested that critically ill people probably have the same possibility of experiencing epileptiform discharges as a result of sleep loss as healthy volunteers (Schwab, 1994).

**Physical working capacity and sleep loss.**

In their thorough review of the literature related to the health effects of sleep deprivation, Naitoh et al. (1990) concluded that although physical work capacity appears not to show deterioration in sleep loss of three days or less, physical movement is greatly reduced. Subjects' willingness to remain physically active is also severely negatively affected. In addition, the studies cited in this review revealed that physical work capacity of test subjects decreases by approximately 5% in terms of maximal oxygen consumption, and by approximately 20% in terms of time to exhaustion. As with other studies discussed thus far, the subjects were physically fit young men.

There are, however, significant implications from these findings for critically ill individuals. When the physical stress of critical illness is coupled with sleep deprivation, the patient may experience severe alterations in his or her already diminished capacity for physical work. One area in which this becomes particularly relevant, for example, is during the patient's weaning from mechanical ventilation. The patient must be able to undertake some of the physical work of breathing, but in the presence of sleep deprivation, may not have the physical capacity to do so. This finding has been supported by the research of Jenny and Logan (1994) who, in their qualitative study of weaning from mechanical ventilation, found sleep a significant factor in patients' ability to successfully wean from mechanical ventilation.
Hematological function and sleep loss.

According to Naitoh et al.'s (1990) review of the effects of sleep deprivation, the body's iron equilibrium is disturbed during sleep loss. This disturbance may cause anemia, especially if the sleep loss is combined with anticoagulant therapy. Because hemoglobin carries oxygen to all the body's cells, anemia can seriously alter the body's cellular function. Critically ill patients frequently suffer from anemia due to trauma or malnutrition and may receive anticoagulants to ward off potential clot formation (Urden et al., 2002). If sleep deprivation further contributes to anemia, the critically ill patient is dually challenged to receive adequate life supporting oxygen to his or her cells.

Immunological changes and sleep loss.

The effects of sleep loss on immune function are not entirely clear. In animals, studies have shown that sleep deprivation can suppress antibody responses (Benca & Quintans, 1997; Bonnet, 2000; Schwab, 1994). However, the results have not been so clear in humans. There are numerous technical challenges to studying the impact of sleep loss on immune function in humans because of the complexity of the cell mediated and humoral immune responses, and the complexities involved with testing parameters. According to Benca and Quintans, no studies have taken a fully comprehensive approach toward assessing sleep and immune function. However, an increasing body of literature has suggested that sleep is important for the proper functioning of host defense systems. Sleep deprivation may result in impaired lymphocyte and granulocyte function (Bonnet, 2000; Palmblad, Petrini, Wasserman, & Akerstedt, 1979; Schwab, 1994), reduced number and activity of natural killer cells and lymphokine-activated killer cells (Irwin et
al., 1994; 1996), and decreased T-cell release and/or utilization of interleukin-2 (Irwin et al., 1996).

There is also some evidence that sleep deprivation may actually enhance some aspects of host defenses. Benca and Quintans (1997) as well as Schwab (1994) report two studies that showed modest activation of the immune system after moderate sleep loss, although the exact amount of sleep loss was not described. The general consensus at present is that sleep and sleep loss affect host defenses, but whether their impact is direct or indirect is not known (Benca & Quintans). It is generally accepted in health practice that sleep loss results in reduced host immunity (Cooper et al., 2000; Rogers, Szuba, Staab, Evans, & Dinges, 2001; Wallace, Robins, Alvord, & Walker, 1999).

Reduced host immunity has significant implications for the critically ill patient. Critically ill patients are at great risk for iatrogenic infection, due to institutionalization, invasive devices, disruption of skin integrity such as traumatic or surgical wounds, and physical stress (Urden et al., 2002). A reduction (or further reduction) in their immunocompetence due to sleep deprivation places them at even greater risk for overwhelming, potentially life-threatening infection.

Respiratory function and sleep loss.

Studies have shown that sleep deprivation can affect the respiratory system. Sleep deprivation can impair both respiratory muscle function and endurance (Chen & Tang, 1989; Phillips, Cooper, & Burke, 1987). Specifically, Chen and Tang noted that both inspiratory muscle endurance and maximal voluntary ventilation were significantly decreased in 30 healthy men after only 30 hours of sleep loss. This effect is more pronounced in people with chronic obstructive pulmonary disease (Phillips et al., 1987),
leading to decreased ventilatory ability and loss of pulmonary reserve (Wallace et al., 1999). Furthermore, sleep deprivation can decrease both the hypercapnic ventilatory response and hypoxic ventilatory response (Cooper & Phillips, 1982; Schiffmann, Trontell, Mazar, & Edelman, 1983; White, Douglas, Pickett, Zwillich, & Weil, 1983). These data suggest that respiratory chemosensitivity may be blunted in people with sleep deprivation, resulting in a decreased ventilatory response to high blood carbon dioxide levels or low blood oxygen levels (Meyer et al., 1994; Schwab, 1994; Wallace et al., 1999). This may result in acute respiratory failure and potentially death.

The effects of sleep deprivation on respiratory function have particular significance for the critically ill patient. The above noted effects all have the potential to interfere with the critically ill patient’s oxygen supply and demand balance, specifically by impairing oxygen supply and increasing oxygen demand. They affect the critically ill patient’s ability to engage in the work of breathing necessary to obtain oxygen for the body’s cells. The loss of muscle endurance in an already compromised individual may also interfere with the process of liberation from mechanical ventilation (Gabor et al., 2001; Redeker, 2000; Schwab, 1994), a necessary process in recovery from many types of critical illness.

**Human growth hormone and sleep loss.**

There is a known relationship between human growth hormone and slow wave sleep. Maximal growth hormone release occurs within minutes of the onset of slow wave sleep (Van Cauter, 2000), however the effect of sleep loss on its secretion is not as clear. Selective partial sleep deprivation does not necessarily suppress or delay the increased release of growth hormone during slow wave sleep onset, suggesting that there may be a
circadian rhythm supporting the secretion of this hormone that exists independent of
sleep (Van Cauter). However, it is known that sleep fragmentation decreases nocturnal
growth hormone secretion. This may in part be because sleep fragmentation can prevent
the occurrence of slow wave sleep (Van Cauter).

Since human growth hormone supports protein anabolism for cell repair, growth, and
replication, decreased secretion has serious implications for the critically ill patient. Cell
repair and growth are necessary for healing. Furthermore, human growth hormone
promotes the conversion of glycogen to glucose. This in turn provides energy substrate
for body functions, including healing. A decrease in human growth hormone, then, could
lead to impaired healing, especially in a compromised critically ill patient.

**Psychological Effects**

Psychologically and mentally, sleep deprivation has many effects. It is well
documented that sleep loss results in mood changes, including increased sleepiness,
fatigue, irritability, anxiety, difficulty in concentrating, lapses in attention, and
disorientation (Bonnet, 2000; Coren, 1996; Naitoh et al., 1990). According to Bonnet,
ipairment of recall for elements placed in short term memory is a classic finding in
sleep deprivation studies. According to this same researcher, sleep loss is more likely to
affect newly learned skills more than well-known activities, and performance on simple
tasks declines less than performance on more complex tasks during sleep loss.

Illusions and hallucinations, primarily visual, are not uncommon, and tend to occur
more frequently if highly visual tasks are required during sleep loss (Bonnet, 2000;
Coren, 1996; Naitoh et al., 1990). Normal individuals undergoing sleep loss may express
paranoid thoughts, and a small percentage (approximately 2%) may experience temporary states resembling acute paranoid schizophrenia (Bonnet).

There are many implications for critically ill patients in terms of the psychological effects of sleep deprivation. Difficulty concentrating, lapses in attention, and impaired short term memory all can impact any teaching done with patients, such as preoperative teaching, postoperative teaching, or related to weaning from mechanical ventilation. In regard to other psychological effects of sleep deprivation, a brief discussion of ICU syndrome is warranted.

**ICU Syndrome.**

The ICU syndrome, also known as ICU psychosis, is a syndrome defined as a reversible confusional state secondary to hospitalization in the intensive care unit (ICU) (Schwab, 1994; Urden et al., 2002) that often develops between the third and seventh day after ICU admission. ICU syndrome usually resolves within 48 hours of discharge from the unit. Patients are usually lucid for one to two days prior to the development of symptoms (Gelling, 1999; Schwab, 1994; Urden et al., 2002). The exact prevalence of ICU syndrome is unknown, but has been estimated to occur in 7% to 72% of patients admitted to the ICU (Gelling, 1999; Hale, Koss, Kerstein, Camp, & Barash, 1977; Schwab, 1994). Although there is a large range of estimated prevalence rates, if only the lowest figure of 7% is representative, it can be said that a large number of patients will experience some form of this disturbance. The clinical manifestations of the syndrome include a wide degree of psychological reactions, including hallucinations, disorientation, anxiety, depression, fear, paranoia, irritability, restlessness, and combativeness (Gelling, 1999; Helton, Gordon, & Nunnery, 1980; Urden et al., 2002; Wilson, 1987). These
symptoms frequently predispose patients to self-harm, especially as many ICU patients have several invasive, critical pieces of equipment attached to them that may easily be dislodged. Physiological changes include symptoms similar to those induced by stress: peripheral vasoconstriction, increased blood pressure, epinephrine release, muscle tension (Gelling). All of these physiological changes serve to increase overall oxygen demand, while at the same time, reduce oxygen supply. According to Schwab and Gelling, it is likely that ICU syndrome increases critically ill patients' length of stay in the ICU because it hinders patients’ recovery by adding more stress to their already impaired psychological and physical status.

The development of ICU syndrome depends on a number of factors, including severity of illness, surgical procedures, medications, metabolic aberrations, hypoxia, sepsis, sensory overload and sensory deprivation. Many authors have suggested that sleep deprivation may be the primary contributing factor (Gelling, 1999; Granberg, Engberg, & Lundberg, 1996; Helton et al., 1980; Hilton, 1976; McGonigal, 1986; Schwab, 1994; Walker, 1972; Wilson, 1988). Interestingly, many of the symptoms noted in ICU syndrome mimic those of sleep deprivation. Furthermore, studies have shown that when sleep deprived healthy people are allowed one night of recovery sleep, their signs of psychosis resolve (Helton et al., 1980; Schwab, 1994). Analogously, the symptoms of ICU syndrome disappear when patients are transferred out of the intensive care unit (Gelling; Helton et al.; Schwab) and, presumably, have less interrupted sleep.
Ill People and Sleep

The Effect of Illness on Sleep

It is generally accepted that when a person develops a sickness, or infection, he or she will have greater sleep needs. In fact, it is not unusual for sleep length to double when a person is ill. This increase in sleep length has been linked to the effects of interleukins (Coren, 1996). Krueger (as cited in Coren, 1996) found that the presence of interleukins in cerebral tissue induced sleepiness, and increased the total amount of slow wave sleep.

With regards to specific illnesses, infectious diseases result in increased interleukins as one part of the complex immune response, resulting in increased sleepiness. Bacterial and fungal infections have increased NREM sleep and decreased REM sleep in animal studies. Gram negative organisms tend to cause these changes in sleep structure earlier than gram positive organisms (Schwab, 1994). These changes have been noted in patients with acquired immune deficiency syndrome (Wooten, 1994).

Chronic diseases such as chronic obstructive pulmonary disorder (COPD) and congestive heart failure (CHF) have been associated with poor sleep quality (Brezinova, Catterall, Douglas, Calverley, & Flenley, 1982; Dark et al., 1987; Findley et al., 1985; Fleetham et al., 1982; Hanly et al., 1989; Krachman et al., 1995; Takasaki et al., 1989). Total sleep time is decreased, with increased sleep latency and more interrupted sleep, with an increased number of arousals noted. Sleep architecture is also altered. A high proportion of Stage 1 sleep and proportionately less Stage 3 and Stage 4 sleep occurs (Krachman et al.). In chronic renal failure, these same changes are noted, along with a reduction in REM sleep. After dialysis, these patients tend to show an improvement in sleep, with fewer arousals, and more slow wave sleep (Wooten, 1994).
Sleep disruption is also recognized as a complication of acute illness (Cooper et al., 2000). It is characterized by reduced nocturnal sleep efficiency, increased sleepiness, and altered sleep architecture, again with increased arousals and Stage 1 sleep, and decreased slow wave and REM sleep (Cooper et al.; Wooten, 1994). These changes have been noted in burn patients, trauma patients, cardiac patients, cardiac surgery patients, general surgery patients and patients with hyperthyroidism (Aurell & Elmqvist, 1985; Broughton & Baron, 1978; Fontaine, 1989; Gottschlich et al., 1994; Hilton, 1976; Kavey & Altshuler, 1979; Knill et al., 1990; Orr & Stahl, 1977; Rosenberg, Wildschiodtz, Pedersen, van Jessen & Kehlet, 1994; Woods, 1972; Wooten).

Critical Illness: Effect on Sleep

Sleep disruption in the intensive care unit is a phenomenon that has been observed for at least three decades. A seminal work by Hilton (1976) described the quantity and quality of sleep in 10 patients with respiratory failure using 48 hours of continuous polysomnography (PSG). Polysomnography is a measure of electrical activity during sleep that is considered the gold standard of sleep measurement. She noted a significant decrease in total sleep time, with only 50% of sleep occurring during the night. There was an increase in Stage 1 sleep, numerous arousals, and a marked decrease in REM sleep and Stage 4 sleep. In some patients, almost no sleep was obtained, and no patient experienced a complete sleep cycle.

In a study on the effect of environmental noise on the sleep of critically ill patients by Freedman et al. (2000), it was noted that five patients developed sepsis during the course of the study. These patients all demonstrated similar EEG patterns of a baseline of low-voltage mixed-frequency waves, with intermittent theta and delta waveforms. This EEG
pattern was present whether the patients’ eyes were open or closed, leaving the researchers unable to discriminate the patients’ states of consciousness into definitive sleep or wake states. No clearly definable sleep was found in these patients during the course of their study. Interestingly, in four of these five patients, the EEG changes were noted up to eight hours prior to the onset of any clinical signs or symptoms of sepsis. These authors concluded that sepsis itself may somehow affect sleep in critically ill patients. Similar to Hilton’s study, these authors noted that in nonseptic patients, sleep architecture was abnormal, with a predominance of Stage 1 sleep, and decreased or absent Stages 2,3,4 and REM sleep. Patients tended to sleep for frequent, short periods that were not consolidated. These periods of sleep were abnormally distributed over a 24-hour day.

Cooper et al. (2000) studied 20 critically ill, mechanically ventilated patients using 24-hour PSG. These patients varied in their type and severity of illness. No patients demonstrated normal sleep. The authors categorized their results into three groups: disrupted sleep, atypical sleep, and coma. The disrupted sleep group had PSG features similar to Hilton’s (1976) and Freedman et al.’s (2000), but the atypical sleep group had EEG features somewhere between sleep and coma. These were characterized by almost absent Stage 2 and REM sleep, and frequent arousals. Pathological wakefulness was also noted in this group. Pathological wakefulness is a state where the behaviours typical of wakefulness, such as sustained EMG activity, are noted during times when EEG features of slow wave sleep are apparent. The coma group had EEG features characteristic of coma. These groups of patients were found to differ in their Acute Physiology and Chronic Health Evaluation (APACHE II) and Glasgow Coma Scale (GCS) scores on
their day of ICU admission, as well as in the amount of sedative medications they received. The GCS is a measure of level of consciousness, and the APACHE score is a measure of severity of morbidity and a predictor of outcomes. The atypical sleep group and coma group both had higher APACHE scores, lower GCS scores, and higher doses of sedatives. Based on their findings, these authors concluded that sleep cannot be identified in all critically ill patients, and have proposed criteria for the selection of critically ill patients at risk of disrupted sleep. These include: APACHE score less than 13, GCS greater than 10, and sedative medication (morphine and lorazepam) equivalents of less than 10mcg/kg/hour.

Other studies have reported that sleep architecture normalizes as time from admission increases (Broughton & Baron, 1978; Kavey & Altshuler, 1979; Knill et al., 1990; Richards & Bairnsfather, 1988). This has led Gabor et al. (2001) to surmise that the illness itself is the primary cause of disrupted sleep.

**Burden of Sleep Deprivation in Critical Illness**

The burden of sleep deprivation in healthy people has been well documented in terms of related accidents, morbidity and mortality, loss of productive days of work, and environmental consequences. However, the burden of sleep deprivation in patients, and especially critically ill patients, is not well described. The assumptions that sleep has a restorative function and that improving the sleep of critically ill patients will promote improved outcomes have yet to be proven. In addition, although there is a great deal of research that supports the potential importance of sleep to immune function, wound healing, respiratory functioning, and mental health, little research has been done to measure these effects in critically ill patients. There also has been little or no research
exploring other outcomes such as quality of life, patient satisfaction, morbidity, mortality, costs, or length of hospital stay.

One investigator found that patients with myocardial infarction and obstructive sleep apnea had no differences in morbidity, mortality, or length of stay in the ICU, compared with those without obstructive sleep apnea (Marin, Carrizo, & Kogan, 1998). However, Redeker, Mason, Wykpisz, Glica, and Miner (1994) found that circadian patterns of activity and rest were associated with better functional status and shorter length of hospital stay after coronary artery bypass surgery in women. One other study found a significant positive correlation between sleep disruption caused by environmental light and noise with duration of ICU stay (Freedman et al., 1999). Rogers et al. (2001) purport that all evidence to date suggests that sleep and sleep loss contribute to disease manifestations.

Despite the lack of objective evidence that sleep deprivation adversely affects outcomes in critically ill patients, other corroborative evidence supports the assumption that it does. If, for example, a patient has a prolonged course in weaning from mechanical ventilation as a result of sleep deprivation, his or her length of stay in the critical care unit likely will be prolonged. Should a patient experience ICU syndrome, his or her length of stay in the critical care unit may also be prolonged (Gelling, 1999; Schwab, 1994), especially if a patient harms him or herself during a period of delirium. Impaired healing and impaired host defenses will have an obvious impact on morbidity and mortality in critically ill patients. The monetary costs associated with ICU care are staggering. In one tertiary level Canadian hospital, it is estimated that the average cost of 24 hours in ICU care is $1,800, exclusive of costs associated with pharmaceuticals,
equipment and supplies, respiratory therapy, and physician costs. If these costs are included, the estimated 24-hour cost can easily exceed $5,000 (H. Tholin, personal communication, January 28, 2002). Therefore, a prolongation in stay of only 24 hours is extremely costly to the already over-burdened healthcare system. Thus, it is timely that further study on sleep in critically ill patients is undertaken, especially interventional studies that may identify strategies to improve the sleep of critically ill patients.

**Stressors that Affect Sleep in Critically Ill Patients**

There are numerous factors that affect sleep in critically ill patients. These can be broadly categorized into three general categories: personal factors, environmental factors, and pharmaceutical influences.

**Personal factors**

Sleep is a highly individualized phenomenon. Accordingly, there are numerous personal factors that can influence sleep in critically ill patients. This discussion will focus on age, gender, state of health/illness, pain and discomfort, anxiety/stress, and primary sleep disorders as the principal personal factors impacting sleep.

**Age.**

Overall, age is associated with many changes in sleep architecture. It is important to note that there can be sleep changes in elderly people as a result of a higher incidence of various diseases and pathologies, as opposed to changes due solely to aging (Bliwise, 2000). However, advanced age is associated with increased arousals and awakenings, prolonged time awake after onset of sleep, decreased sleep efficiency, and decreased slow wave sleep. Changes in circadian patterns, specifically earlier morning awakenings and more daytime napping, are also common (Bliwise; Redeker, 2000).
Gender.

Gender may be a factor that influences sleep during critical illness, although the available data are conflicting. Research studies focusing on women’s sleep disturbances, for example, have reported that women’s sleep may be more, less, and no different than men’s during acute care hospitalization (Redeker, 2000). Further study on the influence of gender on sleep during critical illness is needed.

State of health/illness.

As previously discussed, various diseases and pathologies can contribute to sleep disturbances (Bonnet, 2000). This may be due to arousals or awakenings occurring as a result of symptoms associated with a particular disorder, for example, nocturia as a result of urinary tract infection. However, as Gabor et al. (2001) have suggested, there is some beginning evidence that illness in general is somehow a primary cause of sleep disturbance. Krachman et al. (1995) concur with this view, suggesting that the severity and nature of illness along with any surgical procedure may contribute to sleep disturbances.

Pain and discomfort.

Pain and discomfort are common sleep disrupters in critically ill patients (Hilton, 1976; Redeker, 2000). This is an interesting idea given that little is known about the temporal relationships between pain and sleep. It is known that pain may interfere with sleep, but sleep deprivation may also increase pain perception (Redeker, 2000). Yet, surgical patients tend to rate pain and discomfort as the two worst sleep disturbers during and after their stay in critical care units (Closs, 1992; Redeker, 2000; Simpson et al., 1996a; Southwell & Wistow, 1995). Discomfort in critically ill patients can also result
from mechanical ventilation, which may result in increased arousals or awakenings (Gabor et al., 2001).

**Anxiety/stress.**

Psychological stress and anxiety may also influence sleep during acute or critical illness, although few studies have directly addressed this particular factor. Hilton (1976) noted anxiety to be a common sleep disrupter in her study involving respiratory patients, and more recent studies have been in accordance with her results (Simpson, Lee, & Cameron, 1996a; Southwell & Wistow, 1995).

**Primary sleep disorders.**

Another personal factor that may impact sleep in critically ill patients is whether they suffer from a primary sleep disorder. Primary sleep disorder is a broad classification that refers to abnormal sleep conditions (Aldrich, 2000). There are four major categories of primary sleep disorders: the dyssomnias, which are disorders that produce either insomnia or excessive sleepiness; the parasomnias, which are sleep disorders that include a group of behavioural or movement disorders that occur during sleep (e.g., sleepwalking and periodic limb movement disorder); disorders associated with medical or psychiatric disorders such as mental and neurological disorders (e.g., panic disorder and sleeping sickness); and proposed sleep disorders, which are disorders that have insufficient information about them to accept them as definitive disorders. These include sleep-disordered breathing commonly known as obstructive sleep apnea (OSA) (Krachman et al., 1995; Thorpy, 2001). Primary sleep disorders may result in chronically decreased continuity of sleep, and increased sleep deprivation (Redeker, 2000). In addition, acutely ill patients with sleep-disordered breathing may develop respiratory failure, or have

Environmental Factors

Environmental factors that impact the sleep of critically ill patients include noise, light, and interruptions for patient-care activities. In fact, several studies have concluded that patients and nurses identify these environmental factors as the most common factors that disrupt patients’ sleep (Freedman et al., 1999; Hilton, 1976; Meyer et al., 1994; Redding, Hargest, & Minsky, 1977; Redeker, 2000; Schwab, 1994; Simpson, Lee, & Cameron, 1996b). Before discussing the literature concerning these factors in detail, some discussion of the physical layout of a typical ICU is warranted because this layout frequently serves to make these factors more pronounced.

The typical ICU layout is designed to allow easy monitoring of, and access to, critically ill patients. It is not unusual for an ICU to be designed as one large room with a central nurses’ station, and beds separated only by curtains. There is generally an abundance of technological equipment attached to each patient, including mechanical ventilators, cardiac and hemodynamic monitors, and intravenous infusion pumps, to name a few. All these pieces of equipment typically have auditory alarms that periodically ring when attention is required. Patients’ charts and care plans are usually kept on the nurse’s desk area near the end or side of the bed. In fact, all discussion and action related to the patient occur directly at his or her bedside. This type of layout can only contribute to and accentuate other environmental factors that impact the sleep of critically ill patients.
Early studies identified excessive light as a significant source of sleep disturbance (Keep, 1977; McGonigal, 1986; Woods, 1972). Simpson et al. (1996a), in a more recent study involving cardiac surgery patients, noted that light was considered a source of sleep disturbance by patients. The normal light-dark cycle that is known to support the natural sleep-wake cycle can be virtually lost if bright lights are used on a continuous basis (Krachman et al., 1995). In current practice, however, one must question the relevance of these findings to critically ill patients’ experience of sleep deprivation because it appears that the use of bright lights at night is not as common as it might once have been (Redeker, 2000). In addition, Meyer et al. (1994) found that light levels in one ICU had a circadian rhythm, with higher levels during the day, and lower levels at night, although the levels always remained higher than those recorded in private rooms. Further research is needed to determine the significance of light as a determining variable in critically ill patients’ experience of sleep deprivation.

Interruptions for patient-care activities.

The care of a critically ill patient involves an entire team of people, each of whom needs time with the patient. Unfortunately, this involvement may be uncoordinated, resulting in frequent interruptions and disturbances for interventions and diagnostic testing (Hilton, 1976). The very nature of critical illness is such that it requires intensive nursing care; activities related to this care include frequent monitoring, treatments, medication administration, turning and positioning, and intervening to promote oxygenation. These activities, necessary for the patient’s physical health, frequently occur throughout the night as well as the day, often hourly. Thus, they often do not allow
for blocks of uninterrupted time to complete 90-minute sleep cycles (Hilton, 1976; Richards, 1994; Richards & Bairnsfather, 1988; Schwab, 1994; Walker, 1972). Freedman et al. (1999) found that disruption caused by human intervention and diagnostic testing was perceived by patients to be as important a factor as any other environmental factor, including noise. These authors noted that their results were consistent with those of Woods (1972), who found that postcardiotomy patients had their sleep interrupted by human intervention an average of 59.5 times per night. Meyer et al. (1994) found that interruptions caused by human intervention occurred at least hourly throughout the day and night. Very few studies have explored the feasibility and effectiveness of changing the timing of patient care to accommodate sleep. Providing a six-hour uninterrupted sleep period resulted in improvement of self-reported sleep in a study of post cardiac surgery patients (Sakallaris & Orell, 1997). In one other study, changing the timing of nitroglycerin paste dosages from every four to every six hours improved quality of sleep without increasing any symptoms of angina (Ryan, Gallagher, & Wandel, 1992).

Sleep is disrupted by multiple healthcare practitioners not solely out of necessity. Pulling (1991) found that critical care nurses are not knowledgeable about sleep in general, although they tend to rate sleep as a high priority for patients. It appears that critical care nurses are aware of the importance of sleep, but they do not necessarily differentiate between essential and nonessential tasks to permit uninterrupted time for sleep, nor do they attempt to control the environment to promote sleep (Morgan & White, 1983; Pulling).
Noise.

Noise has been defined as a "desirous or injurious" sound that travels through the air as waves (Snyder-Halpern, 1985). Sound is measured in decibels (dB), which are a unit of sound that approximates the human perception of loudness (Snyder-Halpern). The dB range begins at 0, which is the threshold for hearing, and generally reaches up to 120, which is the threshold for pain (Bentley, Murphy, & Dudley, 1977). At 90 dB, hearing damage can result within eight hours of exposure. The dB range of some typical sounds is 30 dB for a whisper, 50 for normal conversation, 60 dB for a radio set at full volume, 80 for a noisy factory, and 100 dB for a subway (Snyder-Halpern). The decibel scale is logarithmic, thus a sound increase of 10 dB (e.g., from 50 to 60 dB) is perceived by the human ear as twice as loud (Meyer et al., 1994).

Bursts of sound are known to result in arousal from sleep (Bonnet, 1989; Meyer et al., 1994). The threshold necessary for arousal depends on the sleep stage the subject is in, the age of the patient, and possibly habituation to the environment (Bonnet, 1989; Meyer et al., 1994; Williams, Hammack, Daly, Dement, & Lubin, 1964). According to Meyer et al. (1994), sound peaks over 70 dB result in arousals in all stages of sleep, and the older the individual, the lower the threshold necessary for arousal. Aaron et al. (1996) found that sound peaks of 80 dB were very strongly correlated with arousals from sleep in a critical care unit, and concluded that sound levels may be an important cause of sleep disruption in the ICU. Topf and Davis (1993) found that subjects exposed to recorded critical care unit noise in a laboratory environment suppressed REM sleep. Exposure to recorded critical care unit noise resulted in subjects self-reporting poorer subjective sleep than subjects in a quieter environment (Topf, Bookman, & Arand, 1996). Some authors
suggest that although bursts of noise may cause arousals from sleep, excessive ambient noise may also prevent patients from falling asleep in the first place (Schwab, 1994; Snyder-Halpern, 1985; Topf & Davis). Schwab suggests that noise levels of less than 35 to 40 dB are necessary for a patient to fall asleep. The United States Environmental Protection Agency (1974) has recommended that noise levels in the hospital not exceed 45 dB during the day, and 35 dB at night. To compare, typical values for a living room during the day are 40 to 50 dB, and for a quiet bedroom at night are 20 to 30 dB (Krachman et al., 1995).

Researchers have repeatedly described intensive care units as having excessively high noise levels. Woods and Falk (1974) found that mean sound levels in a recovery room were above 60 dB 48% of the time, and 73% of peak sound levels were over 60 dB. Redding et al. (1977) measured sound levels in four intensive care units. They noted that background levels of noise averaged 71-77 dB during the day. Various alarms associated with equipment attached to patients ranged from 71 to 92 dB. Similarly, Bentley et al. (1977) found that daytime ICU noise levels averaged 72 dB, and at night averaged 62 dB. Furthermore, they found that loud noises above 70 dB occurred about every nine minutes at night in the ICU. Both these studies identified staff talking and equipment as the two main contributors to noise. In a study in one large hospital ICU, Hilton (1985) found that sound levels were between 50 and 60 dB 77% of the time, while 23% of the time, sound levels were between 60 and 70 dB. Decibel levels generated from equipment reached 90 dB on occasion. Another study found similar results in a cardiac surgery intensive care unit, with the minimum sound level recorded in a 24-hour period 61.3 dB, and the highest 100.9 dB (McLaughlin, McLaughlin, Elliott, & Campalani, 1996). Meyer et al. (1994)
found peak sound levels of greater than 84 dB in an intensive care unit during the day, while at night in that same unit, the peak sound levels only decreased to 80 dB. Simpson et al. (1996a) interviewed patients after discharge from an ICU to discover their perceptions of environmental factors that disturbed their sleep after cardiac surgery; specific noises accounted for 21 of 35 different factors noted.

Some differences in noise levels appear to be related to the design of the intensive care unit, with higher noise levels recorded in areas with more patients (Meyer et al., 1994; Southwell & Wistow, 1995) and more nurses (Woods & Falk, 1974). Many researchers have found that most sources of noise in the intensive care unit setting are preventable. Excessive noise, primarily as the result of staff conversing and equipment use, is also caused by other patients, visitors, telephones, pagers, and background noises such as air conditioners and construction work (Bentley et al., 1977; Hilton, 1985; Meyer et al., 1994; Redding et al., 1977; Simpson et al., 1996a; Woods & Falk, 1974). One study found that talking amongst staff and sound from televisions accounted for 49% of the sound peaks greater than 80 dB in an ICU (Kahn et al., 1998). Furthermore, when these authors implemented a behaviour modification program targeting those sources of noise, their intervention significantly reduced sound peaks greater than 80 dB. Other researchers also tested behaviour modification programs targeting excessive noise in hospitals, although not specifically in intensive care units (Nazzaro, 1972; Webber, 1984). Unfortunately, there have been no studies published that address the long-term success of this type of program.

In contrast, there are some authors that suggest that noise is not the most sleep-disturbing factor in the intensive care unit. Freedman et al. (1999) found that although
noise is perceived to be disruptive to patients’ sleep, human interventions and diagnostic testing were found to be equally disruptive. Another well-designed study found that noise was responsible for only 11.5% of arousals, and for 17% of awakenings, from sleep in an intensive care unit (Freedman et al., 2000). These authors concluded that noise is not responsible for the majority of sleep disruption, as earlier researchers suggested. These authors further suggest that patients may adapt to the disruptive effects of noise on sleep. However, Kam, Kam and Thompson (1994) assert that although individuals may adapt to continuous noise over time, they will still be disturbed by intermittent noises such as ringing telephones or alarms. Further research is necessary to corroborate these findings.

**Pharmaceuticals**

Many medications given to critically ill patients can contribute to disturbed sleep. Some medications are intentionally given to promote sleep, while others are given for reasons other than sleep. There are a number of difficulties in summarizing the relevant literature and in comparing the effects of particular drugs because of methodological differences and limited research. Most studies are conducted on young, healthy male individuals, thus generalization is not advisable (Schweitzer, 2000), particularly to the critically ill population. Pharmacodynamics and pharmacokinetics are difficult to predict in critically ill patients (Gabor et al., 2001), and furthermore, critically ill patients are often on a variety of drugs in combination, which may affect one another. The following is a description of common drugs used in critical care, and the effects they have on sleep.
Opiates.

Opiates are commonly given to ICU patients for sedation, pain control, and anginal management. One of the most common opiates used is morphine. In studies examining the effects of morphine, it has been suggested that morphine can decrease REM activity, increase the number of arousals during the night, and increase Stage 1 sleep (Krachman et al., 1995). Withdrawal from opiates such as morphine further contributes to sleep alterations because it can result in REM-rebound (Gabor et al., 2001). REM rebound occurs when there has been REM sleep deprivation, and is essentially an exaggerated expression of REM sleep. Thus, all the physiological effects normally present in REM sleep occur in a heightened manner (Bonnet, 2000; Gabor et al.).

Hypnotics.

The most commonly used hypnotics in the critical care area are benzodiazepines. Benzodiazepines include such drugs as quazepam, lorazepam, temazepam, flurazepam, midazolam, and triazolam. These hypnotic agents are often used for sedation and anxiety management in critically ill patients. They differ in their time of onset and length of action (Urden et al., 2002). Benzodiazepines can significantly decrease slow wave sleep (SWS); with repeated use, SWS can be abolished (Krachman et al., 1995). Total sleep time is increased, arousals after sleep onset are diminished, but Stage 1 sleep is increased, and REM sleep is mildly suppressed. These drugs can also result in residual daytime sedation (Schweitzer, 2000; Urden et al., 2002).

Stimulants.

There are two broad categories of stimulants that are commonly used in the critical care area. These are sympathomimetics and nonsympathomimetics.
Nonsympathomimetics include xanthine derivatives such as caffeine, scopolamine, strychnine, pentyletriazole, and modafinil. These drugs are used for a variety of reasons in critically ill patients. Effects of these drugs on sleep include increased sleep latency, decreased total sleep time, decreased REM and SWS, and increased arousals after sleep onset (Urden et al., 2002).

Catecholamines and alpha- and beta-receptor agonists belong in the category of sympathomimetics. These include drugs such as epinephrine, norepinephrine, phenylephrine, dopamine, phenylpropanolamine, and isoproterenol. Many critically ill patients receive these substances, often by continuous intravenous administration, to support cardiac output and blood pressure, and to improve overall oxygen supply via oxygen delivery. Many of these drugs are important neurotransmitters in the central nervous system. For example, norepinephrine is associated with the cortical activating system, and therefore wakefulness, while dopamine has a role in maintaining responsiveness to the surrounding environment (Krachman et al., 1995). These drugs can result in increased wakefulness, and a faster REM sleep onset, as well as a decreased sensation of fatigue, although further studies are needed to determine the exact effects (Urden et al., 2002).

**Antihypertensives.**

There are numerous antihypertensives, and they are commonly used in a variety of patients in critical care. The two most common categories of this type of drug is the beta antagonists (beta blockers), and alpha 2 agonists. In terms of beta antagonists, probably the two most prominent are propranolol and metoprolol. These chemicals block beta receptors, resulting in such effects as decreased heart rate and lowered blood pressure.
These drugs impact sleep by increasing total wake time and sleep latency, while decreasing REM sleep. They may also result in insomnia and nightmares (Schweitzer, 2000; Urden et al., 2002).

Atenolol and clonidine are two alpha 2 agonists that are not uncommon in critical care. These drugs can result in decreased REM sleep, and decreased total sleep time in hypertensive patients, but increased total sleep time in normotensive patients. Nightmares and daytime sedation can also result. Methyldopa is an alpha 2 agonist that impacts sleep slightly differently. This drug results in increased REM sleep and increased total sleep time, and can also cause sedation, insomnia, and nightmares (Schweitzer, 2000; Urden et al., 2002).

**Tricyclic Antidepressants.**

Tricyclic antidepressants are sometimes used in the critical care setting for patients who require such therapy. There are a number of drugs in this category that can impact sleep. Amitriptyline, doxepin, imipramine, clomipramine, desipramine, and nortriptyline can all increase total sleep time or increase wakefulness, ultimately resulting in excessive daytime drowsiness (Schweitzer, 2000; Urden et al., 2002).

**Theophylline.**

Theophylline may affect sleep architecture, depending on the patient population in which it is used. It is used primarily in patients with lung disease, although not commonly today. In patients with chronic obstructive pulmonary disease or asthma, this drug has not been shown to affect the amount or architecture of sleep. Yet, in patients with severe obstructive sleep apnea, theophylline can significantly decrease total sleep
time and increase the number of sleep stage changes during the night (Krachman et al., 1995; Schweitzer, 2000).

**Corticosteroids.**

Corticosteroids are used in the critical care setting for several reasons. For example, they are used to decrease the inflammatory response in a number of conditions such as traumatic brain injury, transplantation, and autoimmune disease. Although widely believed to disturb sleep, results of objective studies of the effect of corticosteroids on sleep are inconsistent. The most consistent effect of corticosteroids on polysomnographic data in normal subjects is a marked decrease in REM sleep, although there is some evidence for increased wake time during the night with cortisol, dexamethasone, and prednisone. Hydrocortisone appears to increase SWS, although dexamethasone does not (Schweitzer, 2000).

**Phenothiazines.**

Phenothiazines are used for sedation in critical care. Two of the most commonly used are chlorpromazine and methotrimeprazine. Methotrimeprazine is used more commonly, because it is not as heavily sedating as chlorpromazine and does not have the same degree of adverse hemodynamic effects (Brown & Scott, 1998). There is little published research on the effects of methotrimeprazine on sleep. However, there is evidence that propropamazine, a drug that is very similar to methotrimeprazine (G. Brown, personal communication, March 15, 2002) has little effect on sleep in healthy subjects. The only noted effect is a slight decrease in total REM sleep (Almqvist et al., 1987).
Sleep Measurement

There are both subjective and objective measures of sleep. The currently available techniques include polysomnography (PSG), actigraphy, observation, and patient perception. In the critical care areas, both the environment and the nature of the critically ill patient present some unique challenges for sleep measurement.

Polysomnography

Polysomnography (PSG) is the "gold standard" of sleep measurement against which all other techniques are evaluated. It is the measurement of electrical activity during sleep, where recordings of the electroencephalogram (EEG), the electromyelogram (EMG), and the electro-oculogram (EOG) are made simultaneously. It is the only measurement that provides information on sleep stages, based on wave amplitudes and frequency, eye movement, and muscle tone. Eye movement is recorded to capture the cardinal sign of REM sleep: bursts of rapid eye movements. In addition, the onset of sleep in most humans is accompanied by slow, rolling eye movements, that also occur during transitions to Stage 1 sleep during the night. The EMG from muscles beneath the chin is also used as a criterion for staging REM sleep (Carskadon & Rechtschaffen, 2000; Richards, 1987; Richards, O'Sullivan, & Phillips, 2000).

There are both advantages and disadvantages to PSG. The equipment for PSG is mobile, although it is generally only found in sleep laboratories. It is expensive, however, and requires technical training to master electrode application. Some patients report that they feel constrained by the presence of the PSG leads and as a result they spend more time supine than they otherwise would (Metersky & Castriotta, 1996).
Consequently, it is possible that the process of PSG monitoring, in itself, may affect sleep time and quality.

The collection of one night of sleep data requires the continuous presence of a person trained in the technique of PSG, and the manual scoring of sleep parameters requires about two to four hours per eight hours of continuous data (Richards et al., 2000). Other potential disadvantages include the amount of space the equipment may occupy at the bedside, as well as the possible necessity of shielding precautions for 60 cycle interference (Richards, 1987). There is also no information provided about the patient’s perspective of his or her sleep. However, the advent of computerized polysomnography may alleviate some of the current difficulties associated with PSG. Computerized PSG entails paperless recording, thus reducing the size of the equipment. The data are scored in a computer-assisted or automatic manner, reducing time and costs associated with conventional PSG. Furthermore, the computerized systems do not need to be continuously attended. It is important to note, however, that data scored automatically still need to be checked for accuracy (Hirshkowitz & Moore, 2000).

Actigraphy

An actigraph is a wrist watch sized accelerometer applied to the wrist or ankle that measures motion. The technology is based on the idea that during sleep, there is little movement, whereas during wakefulness, there is increased movement. The data are examined for activity versus inactivity, and analyzed for wake versus sleep (Ancoli-Israel, 2000; Richards, 1987; Richards et al., 2000). According to Ancoli-Israel, researchers agree that actigraphy correlates well with PSG data in normal sleep, with reliability coefficients ranging from 0.89 to 0.98. However, this same author purports
that correlations are lower in disrupted sleep. Thus, actigraphy may not be the best method to measure sleep in a population that is suspected of sleep deprivation or sleep disorders.

Although less costly than PSG, actigraphy remains an expensive monitoring tool. It provides no information on sleep stages (Richards et al., 2000), and its use may be of limited value in the critically ill population because most patients are on continuous bedrest, and may be sedated, thus decreasing their overall activity level.

**Observation**

Observation of patients' sleep by nurses or research assistants has been used as a technique for measuring sleep in critically ill patients. The advantages of observation include a lack of interference with patients' sleep and minimal costs because no equipment or data scoring are required. Edwards, Schuring, and Foote (1993) compared staff nurses' observations of patients' sleep-wake states in a medical intensive care unit with simultaneously conducted PSG. They found that nurses' assessments correlated with PSG 81% of the time. However, other authors have noted that nurses' assessments of patient sleep do not consistently correlate with PSG measures (Aurell & Elmqvist, 1985) or patients' perceptions (Dotson, Kibbee, & Eland, 1986).

Obvious disadvantages to this method include the questionable validity of the observations, as well as the lack of information regarding sleep stages, especially in critically ill patients. For example, a critically ill patient with eyes closed, body quiet, muscles relaxed, and even snoring, may be asleep, in a coma, or in a late shock state.
Patients' Perceptions

There have been several patient questionnaires developed with the intent to measure sleep. The Sleep Pattern Questionnaire is an 11-item sleep and dream log that measures quantity and quality of sleep behaviour (Baekeland & Hoy, 1971). The validity of the instrument has been addressed by correlation of its results with PSG data in normal, healthy sleepers. This questionnaire could potentially be used in the critical care environment, however, rephrasing of some questions would be required. Many of the questions could not be answered by a patient with any oral communication barrier, such as endotracheal intubation. In that case, testing of the revised instrument’s reliability and validity would be necessary before it could be used with the ICU patient population (Richards, 1987; Richards et al., 2000; Richardson, 1997).

The Leeds Sleep Evaluation Questionnaire was designed to quantify subjective reports of sleep for studies involving various psychoactive drugs. It has not been correlated with PSG data (Richards, 1987; Richardson, 1997) and has not been used in the critically ill population.

The Verran/Snyder-Halpern Sleep Scale is a 10-item questionnaire that has been tested in a community-dwelling sample. It has been tested in three studies with critically ill patient samples (Fontaine, 1989; Knapp-Spooner & Yarcheski, 1992; Simpson et al., 1996a) to determine sleep quantity and quality. At this time, this scale, although reliable, does not demonstrate validity when compared with PSG (Richards et al., 2000).

The Richards-Campbell Sleep Questionnaire (RCSQ) was originally developed as a brief, cost-effective measure of perception of sleep for use with critically ill patients. Preliminary testing in older critically ill, but “stable” males has demonstrated internal
consistency reliability of 0.90. The total score accounted for 33% of the variance in sleep efficiency (Richards et al., 2000). Although there is evidence to support the reliability and validity of the RCSQ, further testing in a more diverse critically ill population is needed.

It is apparent, therefore, that there exists a variety of questionnaires aimed at measuring sleep from the perspective of patients' perceptions. While many of these questionnaires have evidence to support their validity and reliability, they tend to lack measurement details concerning sleep stages, and they require alert, oriented, and cooperative patients. Unfortunately, in the critical care areas, this ideal is rare.

**Interventional Studies**

Most of the studies in acutely ill patients are descriptive and already have been discussed. There are few studies that have tested the effect of an intervention on sleep, and even fewer that have tested interventions on sleep in the critically ill population. In an extensive review of the literature, only eight studies were found that tested the effect of interventions on sleep. Six of these eight used a critically ill sample, and only four studies involved PSG.

Sakallaris and Orell (1997) tested the effect of a six-hour block of uninterrupted time on the sleep of postcardiac surgery patients' perceptions of sleep and pain. One hundred and six patients were studied for three consecutive nights using the RCSQ, although the report is not clear about how or when the intervention was applied. In their abstract, the authors stated that this intervention showed a significant relationship between interruptions and sleep scores (p<.0012), but they did not describe this relationship further. They did, conclude, however, that their study provided support for utilizing this
type of intervention clinically. It should be noted that there are several limitations to this particular study. First, there is not enough information for the reader to draw conclusions about the quality of the research. There is no description of the sample, no description of the way the intervention was applied, and no comment as to the research design. Second, although a six hour block of uninterrupted time was appropriate for this sample, this may not be a practical or possible intervention in many critically ill patients because of their need for frequent interventions.

Ryan et al. (1992) studied the effect of changing the administration time of nitropaste on sleep and nocturnal angina in a sample of 33 patients hospitalized with coronary artery disease. These authors changed the time of administration of this medication from every four hours overnight to six. Instead of dosing patients at midnight and four in the morning, they applied the medication at midnight and six in the morning. Using an undescribed self-report measure of sleep, they compared one night using the regular routine (four in the morning dosing) with one night using the prolonged administration time (six in the morning). These authors found that sleep with the six hour schedule was more than one hour longer than with the four hour schedule (p < .025), and that sleep quality was significantly better (p < .005, no effect size provided), with no difference in nocturnal angina between the two nights. There are important limitations to this study. First, the tool they used to measure sleep was not described, nor is there any description of what is meant by "sleep quality" or how it was measured. Because of this lack of description, it is difficult to assess the quality or usefulness of this study. Second, limiting the assessment to two nights provided little control over the many unassessed factors that could disturb sleep or produce angina.
Zimmerman, Nieveen, Barnason, & Schmaderer (1996) tested the effects of music, music video, and scheduled rest periods on sleep on the second and third postoperative day following coronary artery bypass grafting (CABG). Ninety-six patients who were alert and oriented were randomly assigned to one of the three interventions. The RCSQ was used to assess the patients' sleep. The music video group had statistically significantly higher sleep scores than other groups. There were no differences between the groups for pain, although pain diminished significantly over the study period. The authors concluded that their study lends support to the use of music therapy to enhance sleep. This study also has several limitations. First, the interventions were administered either during the afternoon or early evening, while the sleep scores were obtained the next afternoon. There was no comment as to whether the timing of the interventions impacted sleep scores, making it difficult to infer a causal relationship between the interventions and sleep. Furthermore, all the patients were moved out of the critical care area before the interventions were administered. Finally, generalization to other critically ill patients is limited, because the sample in this study was homogeneous in terms of diagnosis, type of surgery, age, gender, and race, although this added to the internal validity of the study.

A study conducted by Olson, Borel, Laskowitz, Moore, & McConnell (2001) investigated the effects of a quiet time protocol on the sleep of patients in a neurocritical care unit. The authors used nursing observation to assess sleep frequency in patients with Glasgow Coma Scale scores > 10 during quiet time conditions and during usual care conditions. A pretest-posttest design was used and 843 patients were enrolled. The quiet time protocol included dimmed lights, closed blinds, televisions turned off, and visits
from family, critical care unit staff, and consultants minimized during the hours of 2:00 PM and 4:00 PM, and 2:00 AM and 4:00 AM. Measures of sound and light were made at the same time. These authors found that implementation of the quiet time policy decreased mean sound and light levels during these times, and the overall probability of patients being asleep during these times was 1.5 times greater than during usual conditions (p = .001). Both light and sound levels were independent predictors of sleep. The authors concluded that patients have a greater tendency to sleep when sound and light levels are decreased in a neurocritical care unit. Again, this study has limitations. The trained observers were nurses who also worked in the unit, thus observations may have been biased. In addition, observation is not the most accurate method to assess sleep (Aurell & Elmqvist, 1985). It is difficult to infer causality with this method. Furthermore, the authors stated that the staff found this protocol very difficult to implement and adhere to, so its practicality is questionable.

Four interventional studies using PSG were found. The earliest is a study by Aurell and Elmqvist (1985) where continuous PSG was conducted on nine post-surgical patients, along with concomitant nursing observations of sleep. These authors optimized conditions for sleep as the intervention. Optimization included constant pain relief, and a concerted staff effort to reduce environmental disturbances. There was no further description of how this optimal state was actually achieved, nor any description of any protocol. These authors found that all patients were severely sleep deprived, and that sleep time as estimated by the nursing staff was often grossly misjudged, and consistently overestimated in comparison with PSG data. The authors speculated that there may be a fundamental disorder of the sleep-wake regulating mechanism that caused the noted sleep
disturbances. Unfortunately, these authors did not discuss their statistical methods. As an interventional study, there are major limitations that curb its usefulness. The first is the lack of a control group. The second is the lack of description of the intervention condition. This study is more useful as a description of sleep after major surgery.

Richards (1998) used one night of PSG to investigate the effects of two interventions on sleep: back massage and guided relaxation and imagery. In a posttest only design, 69 male subjects with cardiovascular illness who were hemodynamically stable were randomly assigned to a group that received back massage, a group that received a guided relaxation session, or to a control group that received regular nursing care. The sleep efficiency index (the variable of interest) was 14.7% higher in the massage group compared to the controls, but the relaxation group did not have a statistically significant difference in their sleep. Additional information gleaned by Richards was the most frequent event directly preceding awakenings or arousals. The most frequent event was provision of nursing care, and the second most frequent was noise. This study, although well done, would have been stronger had the author used a pretest-posttest design instead of posttest only. Further, since up to 50% of total sleep in critically ill patients is known to occur during the day, 24-hour PSG may have elucidated different results. Generalization to other critically ill populations is also limited because of the homogeneity of the subjects, although this characteristic added to the internal validity of the study.

Wallace et al. (1999) used PSG to measure the effect of soft foam earplugs in reducing simulated intensive care unit noise in healthy volunteers. A repeated measures design with randomized order of earplug use during exposure to the noise condition was used.
The findings of this well-controlled study included: sleep is disrupted by exposure to ICU noise, and the use of earplugs results in more REM sleep. The authors concluded that this study provided a reasonable basis for testing the use of earplugs in reducing noise during sleep in critically ill patients, although generalization to this population cannot be done, due to the use of healthy subjects in a laboratory setting.

Wallace, Robins, and Walker (1998) used a randomized, unblended, crossover design to measure the effect of soft foam earplugs in reducing noise during night time hours on the sleep of critically ill patients with PSG. This pilot study included eight patients. Usual nursing care was given. Results indicated moderate effect sizes for sleep efficiency (0.55) and large effect sizes for REM sleep (0.73). In this abstract, there is no discussion of further details that would allow for an assessment of the study conditions or analyses.

Summary

In summary, sleep is a complex multidimensional phenomenon that eludes exact definition and measurement. Researchers continue to pursue an understanding of the physiological processes it entails. Many have sought to define sleep architecture and there exists some acceptance of two discrete phases (NREM and REM) of sleep; each with its own intermittent stages and functions. Much of this theory has been supported with the onset of EEG therapy and its inherent ability to monitor brain activity during apparent sleep-wake cycles.

In keeping with the restorative theory, sleep is a vital human function that supports healing and restoration of the body. Lack of sleep or sleep deprivation has been specifically linked to such physiological problems as impaired wound healing, decreased
immunocompetence, decreased physical capacity for work and impaired mental
functioning. In critically ill patients, little is known about the effects of sleep deprivation.
One can only assume that the general effects of sleep deprivation are greatly magnified in
patients who already suffer severe alterations in their physiological stability.

The research regarding the effects of sleep deprivation are at best contradictory. In
addition, studies designed to measure these effects have tended to focus on healthy young
males as their subjects, making generalizability difficult. The research regarding
interventions to diminish sleep deprivation is even scarcer, particularly with critically ill
patients. The research that has been done has limitations in terms of internal validity and
generalizability. Only four studies have directly investigated the effect of a noise
reduction intervention on sleep, and of those, only three used PSG. Further research is
needed to test the effectiveness and feasibility of interventions on sleep in critically ill
patients, especially those that reduce environmental noise, using objective sleep measures
such as PSG. Given the potential adverse effects of sleep deprivation on critically ill
patients, as well as the wide-spread incidence of sleep deprivation in this population,
further study is even more crucial.
CHAPTER 3: METHODS

Theoretical Background

Further study is needed to develop effective and practical nursing interventions to promote sleep in critically ill patients. Because the etiology of sleep disruption in critically ill patients is multifactorial, interventions must be developed that address its various causes. Environmental noise such as staff voices and equipment is one of the leading causes of sleep disruption and deprivation in critical care (Aaron et al., 1996; Hilton, 1976; Schwab, 1994; Simpson et al., 1996a; Snyder-Halpern, 1985; Topf & Davis, 1993). It follows then, that studies exploring the efficacy of interventions to reduce environmental noise are imperative. The use of earplugs is a noninvasive, low-cost intervention to reduce noise that is practical in critically ill patients, and can be initiated by a nurse. To date, only one American study has investigated the use of earplugs in critically ill patients. The results are limited but show promise for this intervention (Wallace et al., 1998). No Canadian studies have been identified that offer insight into the feasibility and effectiveness of implementing such an intervention.

The theoretical framework guiding this study was based on Davis, Pack, and Logan’s (1997) conceptual framework for stress-related sleep problems. Generally, the theory asserts that critically ill patients are inundated with numerous stressors that can cumulatively result in sleep disturbances. These stressors include: (a) environmental stressors, (b) mechanical stressors, (c) psychological stressors, and (d) physiological stressors. Sleep disturbance in turn results in further stress and ineffective coping. The cumulative effect results in sleep deprivation that in turn, leads to other serious clinical
problems. This study did not examine or test all components of the conceptual framework, but was designed with these assertions serving as central assumptions.

**Assumptions**

In addition to the central assumptions derived from the theoretical framework, the following serve as assumptions underlying the basis of the study. These have been derived from an extensive review of the literature related to sleep and sleep deprivation:

1. Sleep is beneficial.
2. Sleep is necessary.
3. Uninterrupted sleep is better than interrupted or fragmented sleep, and
4. Sleep promotes healing.

**Research Questions**

This study was designed to assess the feasibility of conducting a trial of the efficacy of earplug use in promoting sleep in critically ill patients. The goals were to assess the practicality of using a particular brand of earplugs, and of PSG monitoring in a busy ICU, among other considerations. Several research questions guided this study. The primary ones were:

1. What number of patients from the Vancouver General Hospital Main Intensive Care Unit would meet the specified inclusion criteria in a 12-week period?
2. What would be the participation rate of those who met the criteria?
3. What barriers would be encountered in identifying and recruiting patients who met the inclusion criteria?
4. Of the participants that could be recruited, how many would complete the study? If they did not complete the study, what were the reasons for their drop out?
5. What would be the feasibility and practicality of the Stellate Emerald system of monitoring polysomnography in the Intensive Care Unit?

6. What would be the feasibility and practicality of using Hear Tech World's Finest Brand wax earplugs?

7. What would be the contextual considerations that affected this study?

8. What would be the key budgetary considerations?

There was also a secondary research question that the study attempted to address. The capacity to answer it fully was contingent on the feasibility of the design (i.e., how successful we would be at enrolling patients and monitoring them). This question was:

1. What would be the efficacy of Hear Tech World's Finest Brand wax earplug use on the sleep of critically ill patients?

**Research Design**

This study was originally designed to support a quasi-experimental, single-subject, time-series experiment (i.e., ABA design). In this particular design, repeated pre-treatment and post-treatment measures of the dependent variable are taken, while repeated measures of the dependent variable are taken during the treatment condition. The pre-treatment measures are the basis of comparison for the treatment condition measures, to determine whether an effect has taken place. The post-treatment measures are collected to determine if the dependent variable reverts back to its pre-treatment level. Reversal of the dependent variable back to its pre-treatment level is considered to be a crucial element for demonstrating that the experimental treatment versus some other variable, caused the change in the dependent variable. In this design, each participant serves as his or her own control (Christensen, 1991).
Instrumentation

During this study there were three periods in which data were collected. These periods included three consecutive nights over the time of 2200 to 0600 hours. On the first night, the subjects were prepared for sleep in the evening as per usual nursing care. Usual nursing care was that which is normally provided for patients, including any scheduled treatments or tests. The researcher attached the polysomnography (PSG) monitoring leads and tested the equipment at 2100 hours. Monitoring began at 2200 hours and ended at 0600 hours the following morning. No other attempt to control the environment or experimental treatment was instituted. This period of continuous monitoring served as the pre-treatment measure, or condition “A” of the selected design.

The second night, the subjects again were prepared for sleep in the evening as per usual nursing care. The researcher attached the PSG monitoring leads and tested the equipment at 2100 hours, as well as initiated the treatment condition. The treatment condition entailed the insertion of earplugs into the participants’ ears by the researcher. Monitoring again occurred between 2200 hours and 0600 hours of the next morning. This period of monitoring served as the treatment measure, or condition “B” of the selected research design.

The third night again began with the researcher applying the PSG leads and testing the equipment at 2100 hours following usual nursing care. Monitoring continued from 2200 hours to 0600 hours the following morning with no attempt to control the environment. This period of monitoring served as the post-treatment measure, or return to condition “A” of the chosen research design.
The polysomnographic monitoring equipment was attached according to international standard sleep monitoring criteria, known as the 10-20 system, as described by Carskadon and Rechtschaffen (2000). In this system, electroencephalography (EEG) monitoring is used to distinguish the four stages of NREM sleep, while electro-oculography (EOG) monitoring is used to identify REM sleep, as well as the onset of Stage 1 sleep. Electromyography (EMG) is used in this system to stage REM sleep and to assess for the presence of sleep disorders. Four EEG leads are generally used in this system. One of these leads was placed in the left central position (called C3), and was referenced to another electrode placed on the contralateral mastoid (called A2), while another was placed in the right central position (called C4), and was also referenced to another indifferent lead placed on the contralateral mastoid (called A1). Another lead was placed in the left occipital position (called O1) and referenced to A2, and a lead was placed in the right occipital position (called O2) and referenced to A1. The proper positions were ascertained by measuring intervals of 10% and 20% of the total distance between landmarks. The four landmarks included the nasion, inion (external occipital protuberance) and left and right preauricular points. Measurements were made at 10% and 20% of the distances from inion to nasion, from left and right preauricular points, and around the circumference of the head. Please see Figure 2 for a schematic representation of the measurements for the 10-20 electrode placement system.
After the measurements were made, the hair was separated and the scalp cleaned in preparation for electrode application. Cleansing was accomplished by brisk rubbing with gauze. The electrodes were attached with Ellefix, a conducting medium that does not evaporate for 24 to 36 hours (Carskadon & Rechtschaffen). Lead placements for the EOG were standard. Standard EOG lead placements include the right outer canthus (ROC) and left outer canthus (LOC), slightly offset horizontally. One of these electrodes was referred to the auricular reference on the opposite side, while the other was referred to the same reference. These leads were also attached after skin preparation using the same technique. Paper tape was used to help secure these leads, since there was little, or no hair in this area. The EMG lead placements were also standard. These included two electrodes placed beneath the chin, overlapping the mentalis and submentalis muscles.
These electrodes were affixed following skin cleansing using the same technique. Again, paper tape was applied to help ensure attachment. The researcher received approximately 20 hours of instruction and training in PSG techniques and monitoring from a registered polysomnography technician prior to undertaking data collection.

The polysomnographic equipment used in this study was the Emerald portable EEG system by Stellate Systems (Montreal, Quebec). This system consisted of a laptop computer with monitoring software that the PSG electrodes attached to via a jackbox and amplifier. The signals were stored electronically on a computer hard drive with Harmonie Diagnostics software. The entire system easily fit onto a small bedside table, and its cables were long enough to allow the researcher to monitor the recording approximately four metres from the subjects' bedsides. The electrodes used were standard gold cup EEG electrodes. The PSG studies were continuously attended by the researcher. Recordings were reviewed on an hourly basis by the researcher, and if skin impedances exceeded 10 kilo Ohms, the electrodes were reapplied.

Other data were collected from the patients' medical charts and included the following: diagnosis and medical history, medication regimes, vital signs, respiratory parameters including oxygen saturations, and hemodynamic information. Demographic data regarding age and gender were also collected.

**Sampling and Data Collection**

The sample consisted of critically ill adults hospitalized at the Vancouver General Hospital, Main Intensive Care Unit. This convenience sample was selected on the basis of specific inclusion criteria, within the allotted time frame of 12 weeks. The inclusion criteria were based on Cooper et al.'s (2000) description of critically ill patients who are
appropriate candidates for the study of sleep. These inclusion criteria included: Glasgow Coma Scale (GCS) > 10 and sedatives of lorazepam equivalents < 10 mcg/kg/hour in the preceding 24 hours. These authors also suggested an upper limit of 10 mcg/kg/hour of morphine equivalents in the preceding 24 hours. However, this upper limit is extremely low for a standard adult dose of morphine equivalents (J. DeLemos, personal communication, August 12, 2002), and as such, would have essentially precluded any patients who received any amount of narcotic from participating in the study. A discussion with one of the authors of that particular study revealed that the subjects who were deemed appropriate for sleep studies received up to 30 mcg/kg/hour of morphine equivalents, and the published criterion of 10 mcg/kg/hour was arbitrarily chosen to be certain to eliminate the effect of narcotics on sleep (P. Hanly, personal communication, August 16, 2002). Following this discussion, it was decided in concert with the clinical critical care pharmacist that the upper limit of narcotic for this study would be 30 mcg/kg/hour (J. DeLemos, personal communication, August 17, 2002). Other inclusion criteria were: 18 years of age or older, no history of deafness, no history of primary brain dysfunction, no known history of sleep disorders, on mechanical ventilation, spent at least one night in the Intensive Care Unit, English language proficiency, and consent to participate provided by the patient directly or a family member. Subjects were recruited by referrals from personnel in this unit, primarily from the intensivist in charge of the unit, although recruitment notices were posted in family waiting room areas and throughout the ICU. The purpose and intent of the study was relayed to potential subjects and their families through a personal visit by the researcher.
Setting

As previously noted, the study took place in the Main Intensive Care Unit at Vancouver General Hospital. This unit is a 20 bed open style unit. Most beds are placed in a central corridor, separated only by curtains. At one end of this corridor are two large rooms, called pods, that contain three beds each, again, separated only by curtains. There are four private rooms at one end of the central corridor that usually function as isolation rooms, and another two private rooms just beyond the two pods. There are two nurses' stations in the middle of the corridor where central computers, telephones, addressographs, and pneumatic tube systems are placed. The medication cupboard is behind the nurses' stations, and the main supply area is an open room just behind the medication area. At one end of the unit, there is another telephone and computer desk along with a supply and medication area. The opposite end of the unit holds another nurses' station complete with central computers, telephones, addressograph machines, pneumatic tube system, medication area and supply area. There is an annexed area that holds another four ICU beds separately, located in the post-anesthetic recovery room. These ICU beds have a layout almost identical to the main unit, with a nurses' station adjacent to the beds with central computers, telephones, addressographs and pneumatic tube systems, as well as a medication cupboard and a supply area. All supply areas contain ice machines. Common patient bedside equipment includes cardiac and hemodynamic monitors, mechanical ventilators, and intravenous pumps, and may include further specialized equipment, depending on the status of the patient. Each bedside is normally attended by a nurse who has a bedside table for charting and at least one chair. Visiting hours are unrestricted. Please see Figure 3.
Operationalization of Variables

According to the conceptual framework for stress-related sleep problems, environmental stressors are major factors contributing to sleep loss in ICUs. They are also the most amenable to change (Davis et al., 1997). Noise is a predominant environmental stressor that can produce sleep loss. Due to the focus of this study, the noise levels of the ICU environment were not measured. It is important to note, however, that given the particular ICU environment in which the study took place, there is no reason to suggest that there was less noise than that described in the published literature (Hilton, 1985; Meyer et al., 1994; Simpson et al., 1996a; Southwell & Wistow, 1995).

Two variables were specified for this study: (a) the use of earplugs and (b) sleep parameters that indicate the objective quantity and quality of sleep. These have been listed as independent and dependent variables, respectively.

The Use of Earplugs

The literature is scarce regarding interventions to reduce noise and promote sleep. In two separate publications, Wallace et al. (1998, 1999) reported promising results.
concerning the effectiveness of earplugs in reducing intensive care unit noise, although
the generalizability of their findings was limited. The use of earplugs in this study is
similar to previous research in terms of the type of earplugs used.

The use of earplugs was operationalized as the placement of Hear Tech World's Finest
Brand wax earplugs (Beneficial Products, Inc., Ashland, Oregon) by the researcher in the
external ear canals of the study participants. Earplugs attenuate noise by providing a
barrier to block sound waves, thus reducing the noise entering the ears (Wallace et al.,
1999). In keeping with the conceptual framework, these earplugs decreased patients'
perceptions of the environmental stressor, noise. The earplugs used in this study were
hypoallergenic wax plugs with a noise reduction rating (NRR) of 34 decibels. This NRR
is the highest rating earplugs alone can achieve (T. Bergman, personal communication,
April 15, 2002). With noise reduction of 34 decibels, a vacuum cleaner operating at 70
decibels will be reduced to a sound similar to a whisper in volume. These particular
earplugs were chosen for their high NRR and hypoallergenicity. As well, these earplugs
are easily molded to ensure an effective seal that does not easily dislodge. The researcher
kneeded each earplug vigorously for 15 seconds prior to insertion to soften it, then placed
the earplug against the ear opening, and flattened it to seal the external ear canal. The
earplugs remained in place from 2200 hours until 0600 hours the following morning of
the second study night.

Sleep Parameters

The dependent variables in this study were the various sleep parameters that
indicate quantity and quality of sleep. These sleep parameters include: the sleep
efficiency index (SEI) and slow wave (SWS) sleep. These sleep parameters were
measured polysomnographically, hence both quantity and quality of sleep were described in an objective manner. Please see Chapter II for a comprehensive discussion of polysomnography. For the purposes of this study, the sleep period was defined from 2200 hours until 0600 hours, although it was recognized that critically ill patients frequently obtain much of their total 24 hour sleep during the day. Because of personnel limitations in attending PSG monitoring on a 24-hour basis, and because the intervention (earplugs) was only applied at night, it was decided to measure sleep during this time alone.

The objective quantity of sleep was operationalized as the sleep efficiency index and the number of awakenings, as measured by PSG. The sleep efficiency index (SEI) is defined as the percentage of time in bed spent asleep in any sleep stage (Williams et al., 1974). Since critically ill patients are often on a bedrest regimen, this definition was modified to define time in bed as beginning at 2200 hours and ending at 0600 hours. The number of awakenings was defined as the return from any sleep stage to the waking stage (Williams et al.).

The objective quality of the patients' sleep was operationalized as the percentage of slow wave sleep (Stage 3 and 4 sleep) obtained, the percentage of REM sleep obtained, and the number of arousals, as measured by PSG in the specified eight hour period. Slow wave sleep was defined as the combined total amount of Stage 3 and Stage 4 sleep throughout the sleep period, as measured by PSG. REM sleep was defined as the total amount of sleep meeting the criteria for rapid eye movement sleep throughout the sleep period (Williams et al., 1974), as measured by PSG. The number of arousals was defined
as the total number of shifts toward awakening from any sleep stage, or shift from any sleep stage to a "lighter" one, during the sleep period as measured by PSG.

Analysis

In the first stage of data analysis, the polysomnographic sleep data were manually scored according to the standards advised by Carskadon and Rechtschaffen (2000) by a registered polysomnography technician. Once the sleep data were scored, the various sleep parameters were plotted for visual inspection. The peaks in sleep parameter scores on the second night were compared with the first night baseline scores. Peaks that reversed back to the baseline on the third night indicated that the intervention had a positive effect.

During the entire course of the data collection period, logistical problems were recorded and detailed. These included barriers to identifying and recruiting participants, problems experienced in setting up and using the Stellate Emerald polysomnography system, and difficulties in applying and using the Hear Tech World's Finest Brand wax earplugs. Furthermore, any other issue that impacted the execution of the study, such as contextual issues, was detailed and described.

Since only two participants completed the study protocol, no statistical analysis was performed. Each case was compared for similarities and differences in every aspect of the study protocol, including recruitment, application and monitoring of the PSG system, application of the earplugs, and all logistical and contextual issues.

Limitations

This feasibility study has several limitations that must be noted. These include:
1. It provided an estimate of the number of potential participants in only a selected tertiary level intensive care unit in a 12-week period.

2. The study used a sample of convenience and as a consequence, the results may be biased.

3. Only one person, the researcher, was trained to set up and monitor polysomnography, limiting the number of subjects who could participate in the study at any one time.

4. The sample was not large enough to provide sufficient power to detect any statistically significant effects.

**Ethical Considerations**

Ethical approval to conduct this study was sought and received from the Clinical Research Ethics Board of the University of British Columbia, and from the Vancouver General Hospital Ethics Review Board. Permission to carry out the study was sought and obtained from the Acting Medical Director and the Nurse Manager of the Vancouver General Hospital Main Intensive Care Unit. Every effort was made to protect the participants’ privacy. The participants or their family members were assured that their identity would be protected. At no point were specific names or other identifying information noted, or associated with any specific data set. All study information containing patient information was kept locked, and continues so. Participants or their family members were informed that their participation was voluntary, and that they had the right to withdraw their participation at any time during the study. Written consent was obtained from the participants. If the participant’s condition did not permit written consent, written consent was sought from the participant’s family as substitute decision.
makers. Three copies were made of the signed consent forms. One copy was given to the participant/substitute decision-maker, one was provided to the agency to place on the subject’s clinical record, and the original was retained by the researcher.

**Summary**

The design of this study was informed and guided by Davis, Pack and Logan’s (1997) conceptual framework for stress-related sleep problems. Specifically, this framework asserts that environmental stressors such as noise deleteriously affect critically ill patients’ sleep, and subsequently result in adverse consequences. Further assumptions regarding sleep were developed through the literature review process. Together, the conceptual framework and assumptions regarding sleep provided direction in developing the research questions that guided this study.

The study took place in the Main Intensive Care Unit of Vancouver General Hospital. Critically ill adults who met the inclusion criteria were the population of interest, from which the convenience sample was drawn. Polysomnography was performed on the participants for three consecutive nights. On the second of these three nights, the participants wore earplugs. Earplug use was the independent variable in the study, while the sleep parameters of the participants were the dependent variable.

The polysomnographic records were analyzed and interpreted by a registered polysomnography technician. The scores for each sleep parameter were plotted for visual inspection and analysis. In addition, all logistical problems that were encountered during the course of the data collection period, as well as any other issues that affected the execution of the study design were recorded and detailed.
Since this study was intended to assess the feasibility of conducting a larger, adequately powered study using this approach, there were several limitations that were inherent within its design. The sample size was not large enough to detect any statistically significant effects, and the number of participants who could participate in the study was limited by the timeframe and by the amount of equipment available.
CHAPTER 4: RESULTS

In this chapter, the findings of this feasibility study are presented. Since the primary purpose of this study was to examine the feasibility of studying the effect of earplug use on the sleep of critically ill patients, a detailed description of each case is warranted. As only two participants completed the study, the findings are presented in case study format. Following this discussion, the findings that are specific to each research question are addressed.

Case One: Jane Doe

Background

The first participant in this study is referred to as Jane Doe. She was a 36-year-old woman who was admitted to the ICU with a diagnosis of sepsis with acute renal failure. Jane Doe had a previous medical history of gastric ulcers. She was initially admitted to a community hospital with biliary colic, but quickly deteriorated. She required endotracheal intubation and mechanical ventilation, as well as continuous veno-venous hemodialysis for acute renal failure, after developing necrotising pancreatitis. She required several surgeries over the course of four weeks, including a subtotal pancreatectomy, drainage of pancreatic abscesses, a partial colectomy, a small bowel resection, and debridement of her abdomen. Her abdomen had been left open after her last surgery with multiple drains inserted, and she had frequent very complex dressing changes performed by a surgical resident.

Inclusion Criteria

Jane Doe had no history of deafness, primary brain dysfunction, or sleep disorders, and could speak and understand English. Her GCS was 12. Her regularly scheduled
medications included fluconazole, sliding scale insulin, and salbutamol. Other medications included norepinephrine as necessary to maintain her mean arterial blood pressure above 60 millimetres of mercury (mmHg), lorazepam to manage anxiety and discomfort as needed, methotrimeprazine in the evening for sleep, morphine as needed for pain, and three medications to manage pain, anxiety, and to relax muscles during a large abdominal dressing change. These included rocuronium, ketamine, and fentanyl. The morphine equivalents Jane Doe received in the 24 hours prior to participating in the study were 22.5 mcg/kg/hour. She received no lorazepam equivalents in the preceding 24 hours.

Recruitment

Jane Doe was identified as a potential participant two weeks into the data collection period by the clinical pharmacist. As Jane Doe was cognitively impaired, and thus unable to sign her own consent, the clinical pharmacist approached the patient’s substitute decision-maker, her husband, about this study. Once Jane Doe’s husband indicated willingness for his wife to participate in the study, the researcher met with him to discuss the study’s purposes and procedures, and answer any questions. The consent form was then signed and witnessed. Jane Doe’s husband was reminded of the right to withdraw consent at any time during the study.

Night One.

The researcher arrived at 2100 hours and explained the procedure to the patient. The patient was located in Bed 11 in the main corridor of the ICU. Set up of equipment and software required approximately 15 minutes. The equipment was set up on a bedside table approximately three metres from the patient’s bedside, past where the attending
nurse normally sat. Application of the electrodes required almost 45 minutes. The participant’s neck was very stiff, which necessitated the attending nurse’s assistance with positioning for electrode application. The application of the occipital electrodes was particularly difficult for this reason. The equipment was then tested, which showed high impedances (>10 kilo Ohms) for the central leads (C3 and C4), and for the EMG leads located under the chin. The skin in these areas was cleansed again and the electrodes replaced. Despite multiple reapplications, the impedances for the EMG leads remained borderline (8 kilo Ohms) for the duration of the night. The participant was febrile (39 degrees Celsius by axilla) and very diaphoretic, which was likely the reason for the poor contact of the electrodes.

The participant was mechanically ventilated in a pressure support mode of 18 centimeters (cm) of water pressure, with an oxygen concentration of 35%, and continuous positive airway pressure of 12. This remained unchanged overnight. Her respiratory rate ranged from 20 to 25 breaths per minute, and her oxygen saturation level ranged from 97% to 100%. Her heart rate ranged from 109 to 126 beats per minute overnight in a sinus tachycardia. Her mean arterial blood pressure was initially around 70 mmHg, but drifted to around 55 mmHg at 2320 hours. Norepinephrine infusion was started as a result. The mean blood pressure subsequently rose to 70 to 75 mmHg, and remained in that range for the duration of the night.

This first night was relatively uneventful. The patient was settled with usual nursing care at 2235 hours. Overnight, the participant was disturbed for turning, positioning, and skin care and assessment at 0015 hours and 0340 hours. The participant was also disturbed at 0550 hours for a chest x-ray. These disturbances were annotated as
“technician” related arousals on the sleep record. Twice in the night the participant had
prolonged bouts of coughing, requiring suctioning. These were also annotated as
“technician” related arousals. At 0240 hours, after the first bout of coughing, the
participant was given methotrimeprazine 12.5 milligrams (mg) for sleep. The PSG
recording was stopped at 0600 hours the following morning, and the leads were
disconnected from the participant.

Night Two.

The second night was similar to the first in terms of the initial set up. The researcher
arrived at 2100, explained the procedure to the participant, and set up the equipment in
the same manner as the previous night. The electrodes were again placed with difficulty
due to the participant’s very stiff neck and extreme diaphoresis. The attending nurse was
required to help with positioning the participant’s head for electrode application. The
entire procedure took about 35 minutes. The equipment was tested, and electrical
impedances were noted to be somewhat high for the EMG leads (7 kilo Ohms) located
under the chin, despite re-cleansing of the skin and reattachment of the leads. The likely
reason for this was the participant’s continued diaphoresis, which was particularly
noticeable in this area. After the equipment was set up and tested, the earplugs were
inserted into the participant’s ears by the researcher.

The participant’s mechanical ventilation parameters were unchanged from the
previous night and remained the same throughout this night as well. Her temperature was
somewhat higher at 39.6 degrees Celsius by axilla, as was her heart rate, which ranged
from 116 to 130 beats per minute. Her mean arterial blood pressure was supported with
norepinephrine infusion. Her GCS score remained 12. Notes on her chart indicated that
the attending intensivist felt she was deteriorating, and a full septic workup (involving samples for culture and sensitivity from blood, urine, sputum, and any wounds and drains) was planned for the morning. The participant's total morphine equivalents for 24 hours at this time were 25 mcg/kg/hour, and lorazepam equivalents were 0.42 mcg/kg/hour. No methotrimeprazine was administered.

The patient was settled for the night at 2215 hours. At 2300, the patient opened her eyes, and the attending nurse administered lorazepam 1 mg to help her settle to sleep. At 2320 hours, the attending nurse began organizing equipment, including all the drains that were attached to the participant. This process lasted approximately 30 minutes. This episode was annotated as a "technician" related arousal. The patient was disturbed at 0145, 0220, 0230, 0410, and 0430 for assessments, turns, and suctioning. These disruptions were noted as "technician" related arousals, as was a disruption at 0530 for a chest xray and bloodwork. There was a great deal more noise in the ICU this night. Some of this was due to an admission arriving at 0200 hours, as well as due to family visits to the patient in the adjacent bed from 0310 hours to 0430 hours. At 0600 hours, the polysomnography recording was stopped, the earplugs were removed, and the leads disconnected from the participant.

Night Three.

The third night began again with the researcher arriving at 2100 hours to set up the equipment and attach the PSG leads. Once again, the electrode placement was difficult due to the patient's stiff neck, and required the attending nurse's help for positioning. The participant remained very diaphoretic, and once again, upon testing the equipment, it was noted that electrical impedances for the EMG leads were high (>10 kilo Ohms).
However, once the skin in that area was re-cleansed and the leads replaced, the electrical impedances were less than 5 kilo Ohms.

The participant’s condition seemed to have deteriorated over the course of the day. Her temperature had increased to 39.8 degrees Celsius by axilla, and her mean blood pressure required continual support with norepinephrine to maintain a lower value of 65 to 70 mmHg. Her mechanical ventilation parameters were the same, except the oxygen concentration was less at 30%. Subsequently, her oxygen saturation levels were somewhat lower, ranging from 93 to 97%. Notably, her GCS had deteriorated to 10 to 11.

The total morphine equivalents for 24 hours were 26.25 mcg/kg/hour, while lorazepam equivalents were 0.83 mcg/kg/hour. No methotrimeprazine was given.

At 2215, the participant was settled for the night. At 2250, lorazepam 2 mg was given to help the patient sleep. At 0005 hours, the patient was disturbed for an assessment, and again at 0200 for turning and skincare. These were annotated as “technician” related arousals. At 0210, while turned on her side for skin care, the participant suffered an asystolic cardiac arrest. A full resuscitation ensued, with the participant being administered 100% oxygen and receiving chest compressions for a total period of about 60 seconds. At that time, the participant regained a regular sinus cardiac rhythm of 110 beats per minute and a mean blood pressure of 68 mmHg on her own. The entire incident took approximately 70 minutes to resolve and settle. During this time, all lights were on, many people were attending the bedside, and there was constant activity. At 0540 hours the participant was positioned for a chest x-ray; this again was annotated as a technician related arousal. At 0600 hours, the PSG recording was stopped and the leads removed.
Polysomnography Results

The PSG recordings were analyzed by a registered polysomnography technician using Stellate Harmonie software, then were manually scored by this same technician to ensure accuracy. For the purposes of this study, sleep quantity was defined by the number of awakenings the participant experienced over the monitoring period, and the sleep efficiency index (SEI). The SEI is percentage of time in bed spent asleep. Since ICU patients spend most of their time in bed, this definition was further modified to refer to the time from 2200 hours until 0600 hours the following morning. In a healthy young adult, the expected SEI is 96%. The expected number of awakenings for a healthy young adult is less than two (Dement, 2000; R. Polischuk, personal communication, January 6, 2003). Jane Doe’s sleep quantity results are presented in Table 1. For the purposes of this study, sleep quality was defined as the total amount of slow wave sleep obtained, and the total amount of REM sleep obtained. All sleep stages are expressed as a percentage of total sleep. The number of arousals was also included in this definition. As defined earlier, arousals are a shift from any stage of sleep towards a “lighter” stage. Jane Doe’s sleep quality results are presented in Table 2 and Table 3.

Table 1.

<table>
<thead>
<tr>
<th></th>
<th>Night One</th>
<th>Night Two</th>
<th>Night Three</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Efficiency Index</td>
<td>85%</td>
<td>59%</td>
<td>84%</td>
</tr>
<tr>
<td>Awakenings</td>
<td>33</td>
<td>88</td>
<td>7</td>
</tr>
<tr>
<td>Total Sleep Time</td>
<td>399 minutes</td>
<td>279 minutes</td>
<td>397 minutes</td>
</tr>
</tbody>
</table>
Table 2.
Sleep Quality: Jane Doe

<table>
<thead>
<tr>
<th></th>
<th>Night One</th>
<th>Night Two</th>
<th>Night Three</th>
<th>Normal&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1 sleep</td>
<td>6.8%</td>
<td>31.7%</td>
<td>2.8%</td>
<td>2-5%</td>
</tr>
<tr>
<td>Stage 2 sleep</td>
<td>81%</td>
<td>65.9%</td>
<td>94%</td>
<td>45-55%</td>
</tr>
<tr>
<td>Slow wave sleep</td>
<td>6.1%</td>
<td>0%</td>
<td>0%</td>
<td>13-23%</td>
</tr>
<tr>
<td>Stage 3 sleep</td>
<td>6.1%</td>
<td>0%</td>
<td>0%</td>
<td>3-8%</td>
</tr>
<tr>
<td>Stage 4 sleep</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>10-15%</td>
</tr>
<tr>
<td>REM sleep</td>
<td>6.1%</td>
<td>2.3%</td>
<td>3.3%</td>
<td>20-25%</td>
</tr>
<tr>
<td>Arousals</td>
<td>111</td>
<td>123</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>106</td>
<td>111</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>Technician</td>
<td>5</td>
<td>12</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Completed sleep cycles</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Normal values are adapted from Carskadon and Dement (2000) and reflect expected values for a healthy young adult.
Table 3.

Sleep Quality Expressed in Minutes: Jane Doe

<table>
<thead>
<tr>
<th></th>
<th>Night One</th>
<th>Night Two</th>
<th>Night Three</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1 sleep</td>
<td>27</td>
<td>88.5</td>
<td>11</td>
</tr>
<tr>
<td>Stage 2 sleep</td>
<td>323</td>
<td>184</td>
<td>373</td>
</tr>
<tr>
<td>Slow wave sleep</td>
<td>24.5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Stage 3 sleep</td>
<td>24.5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Stage 4 sleep</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>REM sleep</td>
<td>24.5</td>
<td>6.5</td>
<td>13</td>
</tr>
</tbody>
</table>

In terms of sleep quantity, Jane Doe received close to a normal amount of sleep on the first and third nights. Her SEI on these nights was 85 and 84%, respectively. This means that, for example, on the first night, 85% of the time between 2200 hours and 0600 hours, the participant was asleep. On the first night, the number of awakenings (33) was very high. Some of these awakenings occurred when the participant was disturbed for nursing or medical care. On the second night, the SEI was much lower at 59%, with an even greater number of awakenings. This is particularly interesting because this was the night that the intervention, earplugs, was applied. The expectation was that the quantity of sleep would have been higher on this night. However, there are various reasons as to why the participant's sleep was less on this night. Her physical condition had deteriorated, and it is possible that her illness itself further contributed to her disrupted sleep. The greatest number of interruptions occurred this night as well (annotated as
"technician-related arousals". For example, at one point the attending nurse re-organized all the patient equipment that was attached to the participant over the period of a half hour. On the third night, the SEI improved, and there were fewer awakenings. There are many possible explanations for this. First, the participant had received greater amounts of both narcotic and sedative medications than the previous nights. Second, the total number of interruptions for patient care activities was less than either the two previous nights, despite the participant’s cardiac arrest (technician related arousals were 3), especially in the early part of the night. Finally, it is well documented that following a period of sleep deprivation of any type, there will be a period of recovery sleep (Dement, 2000). It may be that the increased SEI on the third night reflected recovery sleep.

Jane Doe’s sleep quality was very poor. On the first night, the majority of her sleep was spent in Stage 2 (81%). She experienced very little SWS in total; time spent in Stage 3 was 6.1% of her total sleep time, while she experienced no Stage 4 sleep. REM sleep was markedly reduced at only 6.1% of her total sleep time. These findings are consistent with previous researchers’ findings describing disturbed sleep in critically ill patients whereby there is a predominance of Stage 1 and Stage 2 sleep, and decreased or absent SWS and REM (Cooper et al., 2000; Freedman et al., 2000; Hilton, 1976). There were frequent arousals as well. Jane Doe did not experience any complete sleep cycles. On the second night, her sleep quality was even worse. Almost one third of her total sleep time was spent in Stage 1, while almost two thirds were spent in Stage 2. She experienced no SWS, and very little REM (2.3% total sleep time). This indicates very abnormal sleep architecture, since REM sleep is normally entered after Stage 3 and 4. No entire sleep cycles were completed. There were many arousals this night (123).
Some of these were clearly related to interruptions for patient care activities (12
technician-related arousals), but many were unexplained. On the third night, the vast
majority of sleep time was spent in Stage 2 (94%), again with no SWS, and very little
REM (3.3%). Again, no entire sleep cycles were completed. The preponderance of
lighter sleep (Stage 1 and 2) may be explained in part by the administration of narcotics
and benzodiazepines. Both types of drugs decrease REM sleep, while promoting the
lighter sleep of Stage 1 and 2. The administration of morphine can markedly increase
arousals during the night, while benzodiazepines tend to decrease arousals (Krachman et
al., 1995; Schweitzer, 2000; Urden et al., 2002). Benzodiazepines can even abolish SWS
with repeated use (Schweitzer). It is interesting to note that Jane Doe’s SWS was
nonexistent on the second and third nights, when she had received increasing doses of the
benzodiazepine midazolam.

It is inappropriate to attempt to draw any conclusions about the effect of the earplug
intervention on the participant’s sleep. Jane Doe’s deteriorating and unstable physical
condition, in conjunction with the completely different sedation regimes administered, as
well as different numbers of interruptions for patient care activities render the control
conditions not comparable. Jane Doe was unable to give any subjective information as to
her experience of sleep with or without earplugs.

Case Two: John Doe

Background

John Doe was the second participant in the study. He was a 57-year-old man with a
past medical history of hypertension. John Doe had been in a light airplane crash that
resulted in multiple trauma. He sustained a severe facial fracture (LeFort III), bilateral
femur fractures, and bilateral hip dislocations. He had been admitted into the ICU postoperatively for ventilatory management for severe atelectasis and left lower lung lobe consolidation.

**Inclusion Criteria**

John Doe had no history of deafness or primary brain dysfunction, and no known sleep disorders. He could speak and understand English, and was able to communicate by writing. His GCS was 15. His regularly scheduled medications included a multivitamin, folate, cefazolin, clindamycin, and enoxaparin. John Doe also received zopiclone in the evening if needed for sleep, and morphine as needed for pain control. His total morphine equivalents in the 24 hours preceding the study were 15.3 mcg/kg/hour, and he had received no lorazepam equivalents.

**Recruitment**

John Doe was identified as a potential participant by the intensivist in charge of the ICU. The intensivist discussed the possibility of John Doe participating in the study, who then agreed to discuss the study with the researcher. The researcher met with the potential participant and described the study’s purposes and procedures, and answered any questions. The consent form was then signed. John Doe indicated he was interested in participating in this study since he felt he “had not slept a wink” since being admitted to the ICU (J. Doe, personal communication, October 11, 2002). John Doe was reminded of his right to withdraw consent to participate at any time during the study.

**Night One.**

The researcher arrived at 2100 hours and explained the procedure to the patient. The patient was located in Bed 26 in the annexed corridor of the ICU. Set up of equipment
and software required approximately 15 minutes. The equipment was set up on a bedside table approximately four metres from the patient’s bedside, past where the attending nurse normally sat. Application of the electrodes required about 30 minutes. Some assistance from the attending nurse was required to place the electrodes because the participant had decreased mobility of the head and neck due to his facial fractures and the position of the endotracheal tube. The equipment and software were tested. All electrical impedances were well within an acceptable range of less than 2 kilo Ohms.

The participant was mechanically ventilated on pressure support of 5 centimetres of water pressure, with an oxygen concentration of 50% and continuous positive airway pressure of 8 cm of water pressure. His respiratory rate was 20 to 29 breaths per minute, and his oxygen saturation levels ranged from 97% to 100%. John Doe was afebrile, with a heart rate ranging from 80 to 100 beats per minute in a regular sinus rhythm. His mean arterial blood pressure ranged from 100 to 123 mmHg.

At 2235 hours, the participant was turned and positioned, and evening care was given. Zopiclone 15 mg was administered by the attending nurse to help the participant sleep, and morphine was administered for comfort. The participant was disturbed at 2350 hours for an assessment; this was annotated as a “technician” related arousal. At 0010 hours, the participant was awake, sitting up and appearing somewhat confused as to where he was. He was reoriented by the nurse and given midazolam 2 mg to settle him. At 0025 hours, the participant was awake and restless. Morphine 2 mg was administered by the attending nurse to treat any pain. By 0030, the participant was awake and agitated; midazolam 2 mg was repeated by the attending nurse. This medication was repeated at 0045 when the participant began trying to extubate himself. By 0110, the participant was
extremely confused and disoriented, and was attempting to get out of bed. Midazolam 4 mg was administered by the attending nurse. At 0130, the participant’s son and friends arrived to visit; the participant remained restless and confused, although less agitated while his son was present. At 0155, the participant again began pulling at his endotracheal tube, and midazolam 4 mg was administered. At 0220, the participant was still awake, but not as agitated. The visitors left. By 0300, the participant again was agitated and pulling at the endotracheal tube. Midazolam 2 mg and morphine 2 mg were given. At this time, the pressure support level on the ventilator was increased to 8 centimetres of water pressure, while the oxygen concentration was decreased to 45%. The oxygen saturations remained unchanged, while his heart rate ranged from 95 to 110 beats per minute. John Doe’s mean blood pressure remained 110 to 125 mmHg during this time. Two mg of morphine were repeated at 0320, but the participant remained agitated. This period of agitation and wakefulness continued until 0415, when the participant appeared settled with eyes closed. However, by 0430 the participant was again agitated and disoriented, and tried to remove his endotracheal tube. Wrist restraints were then applied. The participant at this point was lying quietly with eyes closed, but every few minutes he would try to reach for the endotracheal tube. At 0530, morning assessments and care were given until 0600. At this time, the researcher stopped the PSG recording and removed the leads. It was later agreed that John Doe had likely experienced an episode of ICU psychosis during this night.

Night Two.

The researcher again arrived at 2100 to set up the equipment. The participant at this time was completely lucid with no memory of the previous night beyond the application
of the electrodes and the evening nursing care. The researcher explained the procedure to
the participant, and set up the equipment in the same manner as the previous night. The
electrodes were again placed with the assistance of the attending nurse. The entire
procedure took about 30 minutes. The equipment was tested, and electrical impedances
were noted to be less than 2 kilo Ohms. After the equipment was set up and tested, the
earplugs were inserted into the participant’s ears by the researcher. The participant
indicated in writing that the earplugs were comfortable.

The participant’s mechanical ventilation parameters were unchanged from the
previous night. He remained afebrile, but his heart rate was noticeably lower, ranging
from 70 to 80 beats per minute in a regular sinus rhythm. His mean arterial blood
pressure was also noticeably lower, ranging from 79 to 105 mmHg. His GCS score
remained 15. The lowered heart rate and blood pressure may have reflected the addition
of metoprolol to the participant’s scheduled medications. The participant’s total
morphine equivalents for 24 hours at this time were 3.7 mcg/kg/hour, and lorazepam
equivalents were 1.48 mcg/kg/hour. The conversion for midazolam into lorazepam
equivalents has been determined to be variable, and ranges from one mg of lorazepam
being considered equivalent to five, to nine, and to 14 mg of midazolam (J. DeLemos,
personal communication, August 12, 2002). It was decided to use the lowest conversion
factor (one mg lorazepam equivalent to 5 mg of midazolam) to stay as close as possible
to the guideline of less than 10 mcg/kg/hour of lorazepam equivalents recommended by
Cooper et al. (2000).

The participant was settled for sleep at 2230 hours, with zopiclone 7.5 mg
administered by the attending nurse. The night was relatively uneventful. The
participant was disturbed at 0005 and 0400 for assessments, turns and positioning. These were annotated as “technician” related arousals. At 0310, the participant was awakened by a bout of coughing that required suctioning. At 0600 hours, the PSG recording was stopped, the earplugs were removed, and the leads disconnected. The participant wrote that he felt he had slept very well, and requested to keep the earplugs for future use.

Night Three.

The researcher arrived on the third night at 2100 hours and explained the procedure again to the participant. Set up of equipment and software again required approximately 15 minutes, and application of the electrodes required about 30 minutes. Some assistance from the attending nurse was once more required to place the electrodes due to the participant’s decreased mobility of the head and neck. The equipment and software were tested, and all electrical impedances were less than 2 kilo Ohms.

The participant remained mechanically ventilated on pressure support of 5 centimetres of water pressure, with an oxygen concentration of 45% and continuous positive airway pressure of 5 cm of water pressure. His respiratory rate was 20 to 25 breaths per minute, and his oxygen saturation levels ranged from 97% to 99%. He continued to be afebrile, and his heart rate ranged from 67 to 90 beats per minute in a regular sinus rhythm. His mean arterial blood pressure ranged from 83 to 102 mmHg. The participant had no morphine equivalents in the preceding 24 hours, nor did he have any lorazepam equivalents.

The researcher again arrived at 2100 to set up the equipment. The participant remained completely lucid. The researcher set up the equipment in the same manner as the previous nights, and again placed the electrodes with the assistance of the attending
nurse. The entire procedure took about 30 minutes. The equipment was tested, and all electrical impedances were noted to be less than 2 kilo Ohms.

The participant settled for sleep later than the previous nights, because he had visitors in until 2330 hours. At 2340, he was settled for sleep with zopiclone 7.5 mg given by the attending nurse. The participant was not disturbed for assessments and turns unless he was already awake. The participant spontaneously awoke at 0130 and 0230 from coughing bouts, and again at 0530 to request a bedpan. At 0610, the PSG recording was stopped and the leads disconnected from the participant. John Doe wrote that he felt he did not sleep well. He also wrote that he found the ICU very noisy over night, and wished to use the earplugs again to help him sleep.

Polysomnography Results

As in the case of Jane Doe, the PSG recordings were analyzed by a registered polysomnography technician using Stellate Harmonie software, then were manually scored by this same technician to ensure accuracy. Once again, the results are discussed together. Table 4 presents John Doe’s sleep quantity, while Table 5 and Table 6 present John Doe’s sleep quality.

Table 4.
Sleep Quantity: John Doe

<table>
<thead>
<tr>
<th></th>
<th>Night One</th>
<th>Night Two</th>
<th>Night Three</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Efficiency Index</td>
<td>38%</td>
<td>38%</td>
<td>28%</td>
</tr>
<tr>
<td>Awakenings</td>
<td>17</td>
<td>31</td>
<td>34</td>
</tr>
<tr>
<td>Total Sleep Time</td>
<td>175 minutes</td>
<td>178.5 minutes</td>
<td>135.5 minutes</td>
</tr>
</tbody>
</table>
### Table 5.

**Sleep Quality: John Doe**

<table>
<thead>
<tr>
<th></th>
<th>Night One</th>
<th>Night Two</th>
<th>Night Three</th>
<th>Normal a</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stage 1 sleep</strong></td>
<td>15.1%</td>
<td>20.4%</td>
<td>25.5%</td>
<td>2-5%</td>
</tr>
<tr>
<td><strong>Stage 2 sleep</strong></td>
<td>66.0%</td>
<td>41.5%</td>
<td>40.2%</td>
<td>45-55%</td>
</tr>
<tr>
<td><strong>Slow wave sleep</strong></td>
<td>18.9%</td>
<td>23.2%</td>
<td>28.0%</td>
<td>13-23%</td>
</tr>
<tr>
<td><strong>Stage 3 sleep</strong></td>
<td>18%</td>
<td>15.4%</td>
<td>22.5%</td>
<td>3-8%</td>
</tr>
<tr>
<td><strong>Stage 4 sleep</strong></td>
<td>0.9%</td>
<td>7.8%</td>
<td>5.5%</td>
<td>10-15%</td>
</tr>
<tr>
<td><strong>REM sleep</strong></td>
<td>0.0%</td>
<td>14.8%</td>
<td>6.3%</td>
<td>20-25%</td>
</tr>
<tr>
<td><strong>Arousals</strong></td>
<td>22</td>
<td>27</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>21</td>
<td>23</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Technician</td>
<td>1</td>
<td>4</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Completed sleep cycles</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

*Normal values are adapted from Carskadon and Dement (2000) and reflect expected values for a healthy young adult.*
Table 6.

Sleep Quality Expressed in Minutes: John Doe

<table>
<thead>
<tr>
<th></th>
<th>Night One</th>
<th>Night Two</th>
<th>Night Three</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1 sleep</td>
<td>26.5</td>
<td>36.5</td>
<td>34.5</td>
</tr>
<tr>
<td>Stage 2 sleep</td>
<td>115.5</td>
<td>74</td>
<td>54.5</td>
</tr>
<tr>
<td>Slow wave sleep</td>
<td>33</td>
<td>41.5</td>
<td>38</td>
</tr>
<tr>
<td>Stage 3 sleep</td>
<td>31.5</td>
<td>27.5</td>
<td>30.5</td>
</tr>
<tr>
<td>Stage 4 sleep</td>
<td>1.5</td>
<td>26.5</td>
<td>7.5</td>
</tr>
<tr>
<td>REM sleep</td>
<td>0</td>
<td>26.5</td>
<td>8.5</td>
</tr>
</tbody>
</table>

John Doe received very little sleep in terms of quantity. His SEI on the first two nights was 38%, well below the expected 96% for a young healthy adult. His SEI on the third night was even worse at 28%. However, it is known that sleep quantity declines with age, although the extent to which this occurs is profoundly variable individually. John Doe was 57 years old at the time of this study, certainly not elderly by today's standards, but certainly old enough to have experienced age-related changes in sleep architecture. According to Bliwise (2000), a man this age would be expected to have a SEI of 80 to 85%. Age similarly is associated with an increase in awakenings from sleep (Dement, 2000). Interestingly, John Doe had the fewest awakenings the night he experienced agitation and confusion. This may have been related to the sedative medications he received during the periods of restlessness and agitation. It may have been that without the sedatives he received that night, his SEI might have been
substantially lower, and presumably, his number of awakenings even greater, although this is purely speculation.

John Doe's sleep quality over the course of the three study nights was poor, overall, although he did have some improvement on the second and third nights. On the first study night, the participant spent most of his sleep time in Stage 1 and Stage 2 sleep. The amount of Stage 2 sleep (66%) he obtained was somewhat higher than what is present in normal sleep architecture (45 to 55%), and the amount of Stage 1 sleep (15.1%) was markedly higher than normal (2-5%). Although these findings are consistent with previous authors' descriptions of sleep in critically ill adults (Cooper et al., 2000; Freedman et al., 2000; Hilton, 1976), given the episode of ICU psychosis and resultant agitation the participant experienced, it is remarkable that he received any sleep at all. The “light” sleep John Doe received on the first night likely reflects the administration of midazolam and morphine, both of which promote the lighter sleep of Stages 1 and 2 (Krachman et al., 1995; Schweitzer, 2000). The participant experienced some SWS on the first night, although not in the proportion that it normally occurs. Slow wave sleep is comprised of Stages 3 and 4 sleep. Stage 3 sleep usually comprises 3 to 8% of total sleep time in a healthy young adult, and Stage 4 usually comprises 10 to 15% (Carskadon & Dement, 2000). John Doe spent 18% of total sleep time in Stage 3, with only 0.9% in Stage 4. This may reflect the influence of multiple factors. First, as humans age, the proportion of SWS normally markedly decreases. In fact, in some people, particularly men, SWS can be virtually absent by the age of 65 or 70 (Bliwise, 2000). Second, John Doe received a significant amount of benzodiazepines (midazolam) over the course of the night. As stated earlier, benzodiazepines are known to reduce SWS, and can even abolish
it with repeated use (Schweitzer, 2000). Third, John Doe had several technician related arousals over the course of the night (21), which may have prevented him from staying in SWS. John Doe did not have any REM sleep or experience any complete sleep cycles on the first study night.

On the second study night, the participant had an even greater amount of Stage 1 sleep (20.4%), but also had a greater amount of SWS (23.2%). Stage 2 sleep decreased proportionally (41.5%). Of note, John Doe did not receive any narcotics or benzodiazepines this night, and also received a smaller dosage of zopiclone. REM sleep also increased dramatically to 14.8% of total sleep time on this night. This proportionate increase in both SWS and REM sleep is likely indicative of recovery sleep. The participant had a greater number of arousals this night (27), and notably the majority of these were spontaneous (23). However, he did complete one entire sleep cycle. It should be noted that this night of improved sleep also was the night in which the earplug intervention was applied.

On the third night, the participant had a greater increase in Stage 1 sleep (25.5%), but also experienced an increase in SWS (28%). He also experienced a reduction in REM sleep (6.3%). John Doe completed a sleep cycle this night. The relative increase in SWS and relative decrease in REM sleep may be partially explained by the fact that SWS predominates in the earlier part of the night, while REM sleep predominates closer to morning (Carskadon & Dement, 2000; Krachman et al., 1995; Urden et al., 2002). During the third night, John Doe was awake more frequently and for longer periods near the end of the night than earlier in the night. It is worth noting that he had less frequent arousals on this night, none technician related. It is also important to note that he
received the same dosage of zopiclone as he did on the second night, so in theory, there should have been no difference in sleep due to this drug's effects between the second and third night. Overall, John Doe had less sleep, of slightly poorer quality, on the third night.

No conclusions can be drawn about the effect of the earplug intervention on John Doe's quantity or quality of sleep. John Doe's baseline sleep measurement (Night One) was unstable due to his episode of ICU psychosis, which may have impacted his sleep on the second and third nights. Furthermore, John Doe had different amounts of sedative and narcotic medications on the three nights. However, it is interesting to note that the participant believed that the earplugs improved his subjective experience of sleep.

**Research Findings**

To facilitate the presentation of the results as they specifically relate to the research questions, the research questions themselves are used as an organizing framework. These findings are discussed in further detail in Chapter Five.

**The Number of Patients from the Main Intensive Care Unit That Met the Specified Inclusion Criteria in a 12-Week Period**

In the 12-week period from September 6, 2002 to November 30, 2002, a total of 32 patients were referred to the researcher. Only two of these referred patients were actually enrolled in the study. Of these referrals, 26 were ineligible. Reasons for ineligibility varied. The most common reason for ineligibility was not speaking and understanding English. Language barriers were present in eight of the referred patients.

Six patients were referred who were ineligible due to brain dysfunction. Of these patients, two had seizure disorders, one had hypoxic brain damage following a cardiac
arrest, one had a cerebellar infarction, one developed hepatic encephalopathy after a liver transplant, and one had Down’s syndrome. Most of these patients were referred to the researcher by their family members.

Four referred patients were ineligible because they were deaf or hard of hearing. All four of these patients did not have deafness or hardness of hearing noted anywhere in their medical or nursing history. This characteristic was only discovered when the researcher talked to the family members about the patient’s potential participation in the study.

Mechanical ventilation was discontinued in another four of the referred patients, which rendered them ineligible. Mechanical ventilation was considered a necessary inclusion criterion because the population of interest was mechanically ventilated, critically ill people.

A further three patients were not included in the study because they were expected to be discharged within 12 to 24 hours. It was decided a priori to not include patients who were expected to be discharged shortly for two reasons. First, most patients who are discharged from the ICU are no longer mechanically ventilated, thus eliminating their eligibility for this particular study. Second, these patients would no longer be in the same physical environment. Noise levels in the units they would be discharged to could not be assumed to be the same as in the ICU.

One patient was ineligible due to sleep apnea. This patient was intubated and partially mechanically ventilated, which usually would have circumvented any problems with arousals from sleep due to apneic periods. However, this particular patient was mechanically ventilated with continuous positive airway pressure only. There is no set
breath rate in this form of ventilatory support, which means that the patient can still suffer apneic periods.

The Participation Rate of Those Who Met the Inclusion Criteria

There were six patients referred who met the inclusion criteria. Of these six, four patients or their families as substitute decision makers refused to participate. One patient refused to participate for the reason that she did not want any more equipment attached to her. One patient refused to participate with no explanation. One family refused consent after expressing concern that the PSG equipment could paralyze their family member, despite reassurances to the contrary. One family refused consent on the basis that they did not believe in scientific research. Two patients or their family members agreed to participate. This translates to a 33% participation rate. It must be noted, however, that some family members were eager to participate in this study. In fact, these families referred their relatives to the researcher of their own volition. Unfortunately, none of these referred patients was eligible for various reasons, usually because of brain dysfunction. These have been documented earlier.

The Barriers Encountered in Identifying and Recruiting Patients Who Met the Inclusion Criteria

There were a number of barriers to identifying patients who met the inclusion criteria. Consistent with the policies of the Clinical Research Ethics Board of the University of British Columbia, the researcher was unable to directly identify or approach potential participants. Thus, it had been established prior to the commencement of data collection that the intensivist in charge of the ICU would identify potential participants as a part of daily rounds. Furthermore, the researcher conducted several information sessions about
the study with the staff nurses to encourage them to identify potential participants. Notices were placed in the nursing communication book, on notice boards, and were attached to frequently accessed items, such as the medication cupboards, computer monitors, and bedside tables. The charge nurses had also agreed to identify potential participants to the intensivist in charge. However, in practice, this system did not work smoothly. The researcher was not informed of any potential participants during the first two weeks of the data collection period, despite frequent telephone and email reminders to the intensivist in charge. It was later determined through communication with the charge nurse that two potential participants may have been missed in this period. It was then decided that the researcher would enter the unit on an almost daily basis, after rounds had been completed, and ask the intensivist directly to identify any potential participants. After instituting this activity, the number of potential participants that were identified increased substantially.

Recruitment notices were also placed in prominent places in all critical care waiting rooms. These notices resulted in six referral calls placed to the researcher directly by family members. Unfortunately, none of these patients was eligible to participate. Five had brain dysfunction that rendered them ineligible, while one had mechanical ventilation discontinued.

The largest barrier to recruitment was language. Languages spoken included Italian, Tagalog, Vietnamese, Cantonese, and Punjabi. The researcher in this case is only fluent in English. Since the intervention and monitoring required actual touching of the patient, it was necessary for the researcher to be able to communicate with each participant.
Therefore, those who could not communicate and understand English were ineligible. Fully 25% of referred patients were ineligible for this reason.

Refusal to consent to participate was another significant barrier to recruitment. Four of six eligible patients, or their families as substitute decision makers, refused to participate. As stated earlier, one patient provided no explanation as to why he refused, one family did not “believe in scientific research”, and two were concerned about additional equipment.

Of the Participants Recruited, the Number Who Completed the Study

Two participants were recruited for the study. Both participants completed the entire protocol; there was no attrition.

The Feasibility and Practicality of the Stellate Emerald System in Monitoring Polysomnography in the Intensive Care Unit

The Stellate Emerald system was easy to set up and transport for this study. All necessary equipment packed into a carrying case that could be carried easily by one person. The system did not require much physical space to set up. The entire system easily fit onto one bedside table with room to spare. Because the system had long cables that attached to the electrodes on the participants, the researcher could monitor the recording about four metres away from the bedside. This kept the researcher and the equipment out of the way of the patient’s medical equipment, as well as out of the way of the nurse caring for the participant.

The electrode application was somewhat difficult. The participants in this study had limited mobility, especially of the neck and head area, due to injuries, attached equipment, and muscle stiffness. This made access to different areas of their scalps
challenging. Successful application of the electrodes required assistance from the attending nurses for lifting and holding the participants' heads in position.

There was some difficulty in obtaining an adequate electrical signal from one patient (Jane Doe) due to high electrical impedances. These were likely high as a result of her extreme diaphoresis, although acceptable signals were obtained after recleansing her skin and reattaching the electrodes.

Overall, this system was highly practical for use in monitoring polysomnography in the ICU setting. The difficulties encountered with application of electrodes are not unique to this particular system, and do not impact the assessment of the use of this system. It is also worth noting that Stellate provides 24 hour technical support for this system.

The Feasibility and Practicality of Using Hear Tech World's Finest Brand Wax Earplugs

As stated earlier, this particular brand of earplug was selected for several reasons. Its wax structure is hypoallergenic, and it has the highest noise reduction rating (NRR 34 decibels) that is available with earplugs alone. The earplugs were easy for the researcher to apply. Because this particular type of earplug is molded to seal the entire external ear canal, it can be visually determined to be in proper position. These earplugs did not dislodge over the course of the study nights, but were still easily removed by the researcher. Only one participant was able to communicate well enough to comment on the earplugs' effectiveness and comfort. As stated earlier, this participant found the earplugs comfortable, and believed they were so effective he wished to keep them for his continued use during his hospital stay.
Contextual Factors that Affected this Study

The contextual considerations that affected this study are myriad. Some of the issues were anticipated, but others were not. Most relate to the relative lack of control a researcher has over the critical care environment.

The first contextual factor that affected this study is the difference in patient care activities for each participant, for each night of the study. For example, one study night Jane Doe's nurse decided to re-arrange all equipment that was attached to the participant, after she had been settled for sleep. This was a noisy and disruptive process that lasted at least 30 minutes. It was also a process that did not occur on the other two study nights with Jane Doe.

Another contextual consideration that threatens the internal validity of this type of study is the unpredictable presence of visitors. For example, on his first study night, John Doe had visitors from 0130 hours until 0220 hours. In this particular instance, John Doe was agitated and awake anyway, so the presence of visitors may not have affected his sleep. However, had he been asleep when the visitors arrived, there almost certainly would have been an impact on his sleep scores, particularly in terms of arousals, awakenings, and his sleep efficiency index. This particular ICU has a 24 hour visiting policy, and it is not unheard of for visitors to arrive in the middle of the night.

In terms of internal validity, another very significant issue was the change in sedating medications administered to the participants. There were changes in the amounts of administered narcotics, benzodiazepines, and bedtime sedatives almost every night.

Another contextual issue to consider is the fact that the researcher in this case was a former staff nurse with the unit. The familiarity the researcher had with the staff likely
increased cooperation with the study, notably with helping position the participants for electrode placement. However, the negative aspect of being this familiar with the staff, particularly the nurses, was that at times the researcher was expected to assist the nurses with the care of various patients. This would not have been a problem in and of itself, but assisting the staff nurses would have drawn the researcher’s attention away from monitoring the polysomnography recordings. It was essential to establish clear boundaries related to what the researcher’s role in the ICU was with respect to caring for patients.

In terms of contextual considerations, the greatest impact was caused by the dramatically changing status of the participants. John Doe experienced an episode of ICU psychosis the first study night, then rapidly improved. Thus, his first study night is not comparable to the other two nights as a result. Jane Doe’s clinical condition deteriorated significantly from the first night to the third, when she experienced a cardiac arrest. As a result, the change in her clinical condition may explain her differing PSG results rather than the intervention. Furthermore, her deterioration in condition necessitated the administration of further sedating medications, which certainly may have affected her PSG recordings, further confounding the interpretation of her PSG results.

Key Budgetary Considerations

There were many budgetary issues in undertaking this study. First was the acquisition of the necessary equipment to monitor polysomnography. The equipment in this feasibility study was loaned to the researcher free of charge, including shipping, by Stellate Systems. This was in exchange for sharing the polysomnography results. Normally this equipment rents for a cost of approximately $500.00 per month. Other
required equipment included female gold cup electroencephalography electrodes, and conducting medium (Ellefix). In this study, the researcher was required to purchase insurance for the loaned equipment as a condition of the loan.

The cost of the earplugs themselves must also be factored. Generally speaking, earplugs increase in cost when they are re-usable, and also with a higher NRR. The Hear Tech World’s Finest Brand wax earplugs used in this study cost $6.00 per pair. In a larger study, the cost for earplugs could be substantial, depending on the sample size.

The personnel costs involved with this study were the most significant. The researcher in this study did all aspects of the data collection, from obtaining consent once the potential participants had been identified and approached, to setting up and monitoring the polysomnography recordings. However, the PSG data were analyzed by a registered polysomnography technician, at a cost of approximately $30.00 per hour. This same technician also did the initial training the researcher required to use the PSG equipment.

The Effect of Hear Tech World’s Finest Brand Wax Earplug Use on the Sleep of Critically Ill Patients

This research question was a secondary question that was intended to be answered if enough data were collected to analyze statistically. Unfortunately, only two participants completed the study. No conclusions can be drawn regarding the effectiveness of the earplug intervention. However, it is interesting to note that the one participant who could communicate indicated that he believed that the earplugs helped his sleep, and requested to keep them for his continued use.
Summary

Two participants were recruited for this study. Both completed the entire study protocol, but both experienced physiological complications as a result of their critical illness processes that affected the polysomnography results. Both participants received very poor sleep in terms of both quantity and quality, over the course of the study period.

Recruiting participants proved to be very challenging. Only two participants from a total of 32 referrals were recruited. Most of the referred patients (n=26) were ineligible to participate on the basis of the inclusion criteria. The participation rate of those who were eligible was 33%. Barriers to recruitment included language, inappropriate referrals, and refusal to consent to participate.

The Stellate Emerald system proved to be highly practical for use in monitoring polysomnography in the ICU setting, despite some difficulties encountered with the application of the scalp electrodes. The Hear Tech World’s Finest Brand wax earplugs also proved to be very practical for use in the ICU setting.

There were many contextual issues that affected this study, both in terms of outcome and process. Most of these issues were entirely related to the relative lack of control a researcher has over the clinical environment, especially in the unpredictable critical care environment. Patient care activities differed from night to night, as did the amounts and types of sedating medications each participant received. Visitors were sometimes present at sleep times, although not consistently. Finally, the physiological status of each participant varied tremendously from night to night.
Budgetary implications included the supply of earplugs, and the access to the polysomnography monitoring equipment. The most significant implication for a budget for a larger study is personnel costs.

The sample size of this feasibility study was too small to generate any statistically significant effects from the use of earplugs in critically ill adults. However, one participant who was able to communicate believed the earplugs were effective, and requested to continue to use them throughout his hospital stay.
CHAPTER 5: DISCUSSION AND RECOMMENDATIONS

As stated in Chapter One, the primary purpose of this study was to examine the feasibility of studying the effect of earplug use on the sleep of critically ill patients. As a feasibility study, the researcher sought to gain an understanding of the suitability of the proposed intervention (earplugs), and the possibilities and potential difficulties with the selected data collection method as the primary component of the study. The researcher also intended to identify problems associated with the administration of the study protocol in this population and setting. Furthermore, the researcher hoped to gain an understanding of the projected costs involved with completing such a study with a larger sample. The secondary purpose of this study was to explore the impact of this intervention for a presumed sleep disrupter, noise, on the sleep of patients in the intensive care environment. This chapter presents an assessment of the feasibility of a larger study of the effect of earplugs on the sleep of critically ill patients, as well as recommendations for future research. Implications of the feasibility study for nursing are then identified.

Assessment of the Feasibility of a Larger Study

The assessment of the feasibility of a larger study of the effect of earplug use on the sleep of critically ill adults was directed toward determining the number of participants who could be recruited, the identification of barriers to recruitment and any logistical problems encountered in conducting the study, including the suitability of the research method. In addition, the utility of the Stealate Emerald polysomnography system was evaluated, as was the utility of Hear Tech World's Finest Brand wax earplugs. A review of the study data suggests that a larger scale study of the effect of earplug use on the sleep of critically ill adults is feasible if fiscal resources are adequate to hire staff to support
recruitment efforts and to perform the PSG monitoring, as well as to access language interpretive services. Because the costs may be significant, the rationale for hiring such personnel is discussed at some length. Recommendations are made to address logistical problems, and a discussion regarding the selected research method and inclusion criteria is presented.

The Need for Personnel

Identification and recruitment of participants proved to be one of the greatest challenges experienced during the study. This is clearly evidenced by the extremely small sample size. The nature of the challenges included a lack of identification of potential subjects by ICU medical and nursing staff, the existence of language barriers, and a relatively high rate of refusal. There were also a number of referrals made to the researcher who did not meet the inclusion criteria.

There are several reasons why identification and recruitment of participants were particularly challenging. The Vancouver General Hospital ICU is a tertiary level unit that functions as the province’s trauma center. The patient population has a relatively high proportion of brain injuries, which affected how many potential participants met the inclusion criteria. Furthermore, this unit has the highest acuity level in the province (H. Tholin, personal communication, January 28, 2002). This also influenced the number of potential participants for a study such as this, since sicker patients generally are more likely to require higher doses of narcotics and sedatives, and are more likely to have abnormal EEG findings that preclude sleep (Cooper et al., 2000).

The researcher was often not contacted when potential participants were available in the ICU. The reason for this lack of communication is not entirely clear, but maybe
related to the size and type of unit in which the study took place. As previously stated, this particular ICU is a large tertiary level unit. As such, there are many research studies and clinical trials taking place at any one time. It may have been difficult for the intensivists and nurses to remember to consider various patients for yet another study. Workload of both nurses and intensivists may have been another barrier to identification since one intensivist and one charge nurse indicated that identifying and contacting potential participants, and particularly substitute decision-makers, was too time consuming.

Another significant barrier to recruitment was refusal to consent to participate by either the patient him or herself, or by the family as substitute decision-makers. It is well known that the critical illness experience is highly stressful for both patients and family members. According to Urden et al. (2002), patients and families may experience reactions to the stress of the experience that include emotional turmoil, disorganized thought patterns, confusion, anger, hostility, inability to make decisions, and mental immobility. Anecdotal evidence suggests that obtaining agreement to participate in clinical research is very difficult in critically ill patients and their family members (F. Gagnon, Gagnon Research Associates, personal communication, August 8, 2002; J. Reimer-Kent, Personal communication, June 12, 2001). It is possible that the refusals to participate were not directly about this particular study at all, but rather were a reflection of the nature of the experience of being critically ill, or having a critically ill family member.

Some families were eager to have their critically ill relatives participate in this study, however. Recruitment notices resulted in six referral calls placed directly to the
researcher by family members. Unfortunately, none of these patients was eligible to participate. The majority (five) were ineligible due to brain dysfunction. The inclusion criteria for the study were clearly listed and explained on the recruitment notices. It is therefore interesting that many of these family referrals were ineligible for this reason, since brain dysfunction was a clear exclusion criterion. It may be that the explanation provided regarding the inclusion/exclusion criteria on the recruitment notices was not sufficiently comprehensible. An alternative explanation is that these particular families may not have had a solid understanding of the condition of their family members' neurological status.

One potential solution to the problem of a lack of identification of potential subjects by the ICU medical and nursing staff is to hire a research facilitator who could work collaboratively with interested clinical nurse specialists, clinicians, managers, and other resource staff. Tranmer, Kisilevski and Muir (1995) reported that utilizing a research facilitator increased staff interest in and commitment to a research project in a neonatal intensive care unit. Miller, Johnson, Mackay and Budz (1998) reported that a research facilitator role was very influential in generating support among staff for their research project. In a study of the effect of earplug use, the facilitator in conjunction with interested resource staff could market the study with managers, physicians, and staff nurses. With the unit "gatekeepers" on side, the facilitator could then work with the staff and other clinical contact persons to identify participants (Topolnicky, 1999). A research facilitator could assume responsibility for all recruitment activities. As reported earlier, some feedback from clinical personnel indicated that workload was a concern, and identifying and contacting potential participants or their families was too time-
consuming. Miller et al. (1998) advise that it is important to budget for resources to conduct a study in such a way that "nursing staff will not have to bear the burden of the study" (p. 215). It is unlikely that an adequate sample can be attained in future studies if recruitment efforts fall to overburdened clinicians. The research facilitator could also conduct data collection, if he or she is trained in PSG monitoring techniques. This would require about 20 to 25 hours of training, which of course would need to be funded. However, it must be noted that one research facilitator could not be available for all data collection. Because the data are collected over the course of three nights, it is not physically reasonable to be on call for data collection every single day. It may be necessary to employ other persons trained in the data collection technique, such as polysomnography technicians or electroencephalography technicians, to be on call for data collection, on occasion. This would provide the research facilitator with "time off" from data collection in a cost effective manner. An "on call" stipend for a PSG technician is usually paid at a rate of $1.00 per hour, with an hourly rate of pay of $25.00 per hour should there be data to collect (R. Polischuk, personal communication, February 5, 2003). Overall, the major advantage of utilizing a fulltime research facilitator is the likelihood of recruiting a greater number of participants.

Language barriers were also a significant impediment to recruitment. The relatively high proportion of ineligible participants due to language barriers probably reflects the geographical location of the study. Vancouver is a very multicultural city that is home to many diverse groups who speak different languages. Interpretive services are needed to increase recruitment in a larger study if it is to take place in the same geographic region. Some interpretive services are available at Vancouver General Hospital, but these are
provided by volunteers, mostly staff members, who are able to speak languages other than English. There is no guarantee that at any given time, there will be a volunteer who is fluent in the needed language. Since the data collection aspect of this type of study involves touching and manipulating a participant, the researcher needs to be able to communicate with the subject. Volunteer interpreters may not be available for three consecutive nights to help the researcher (or data collector) communicate with the subject, even if they are available to assist with informed consent. If family members are available, they may be a resource to assist with language, provided they are able to speak and understand English. It may be worthwhile for the researcher to develop a relationship with the volunteer interpretive service at the agency to discover if there are volunteers who may be willing to offer their assistance with language outside of their usual hours.

It is difficult to estimate the potential costs of a larger study without being aware of several important factors. These include the sample size and location(s). The larger the desired sample, the longer it will take to enroll the necessary subjects, which in turn, will increase personnel costs. This will also impact how much is required in terms of interpretive services. Of course, the study duration could be shortened if more than one site was used. However, this would result in the need for personnel coverage at two sites, so the cost differential may be negligible. Depending on where a second site was chosen, more, less, or the same amount of interpretive services may be required.

Addressing Logistical Problems

Logistical problems encountered during the course of this feasibility study included unstable patient conditions, changing medications, changing patient care activities, and
technical issues. The nature of illness trajectories in the critical care setting is
unpredictable at best. This characteristic would certainly impact how many participants
complete the study protocol, especially as it lasts for a three-day period. Given that both
participants experienced a major physiological event during the data collection that
impacted the findings (ICU psychosis and cardiac arrest), it is clear that a proportion of
referred subjects will likely also have major physiological events during data collection.
It is important that the sampling and data analysis plan be designed with this likelihood in
mind.

Due to the unpredictable and frequently changing nature of critical illness, it is not
realistic for participants to receive the same amounts of the same medications, especially
narcotics and sedatives, for three consecutive nights. This may be partly controlled for
by delineating a limit for the amount of narcotic and benzodiazepine a participant can
receive as an inclusion criterion. In this way, the effect of these medications can be
minimized, if not eliminated. However, since pharmacokinetics and pharmacodynamics
are difficult to predict in a critically ill patient, there really is no way of saying how much
effect these medications will truly have on PSG results (Cooper et al., 2000; Schweitzer,
2000). Furthermore, there is little or no information on the effects of other sedating
medications frequently used in the critical care setting, notably methotrimeprazine and
zopiclone, as in the case of this study’s participants.

The differences in patient care activities each participant experienced for each night of
the study were a logistical problem. This was clearly exemplified in Jane Doe’s case
when all the patient equipment was re-arranged after the patient had been settled for
sleep. The very nature of critical care is such that there often are unpredictable patient
circumstances that warrant changes in patient care activities. Most of these activities are necessary for the patient's well-being. However, some activities, such as the described re-arrangement of equipment, clearly are not. Changes in patient care activities undoubtedly threaten the internal validity of a study such as this.

Similar to changes in patient care activities, infectious diseases and relocation of patients are potential logistical problems that warrant discussion. These particular issues were not encountered in this feasibility study, but were readily apparent when the researcher was in the study setting. In terms of infectious diseases, many patients in the ICU were under isolation precautions for methicillin resistant staphylococcus aureus (MRSA). Precautions for patients colonized with this microbe include isolating them from other patients who are not colonized. Most of these patients are therefore cared for in isolation rooms that have walls and closed doors. Thus, these patients are not exposed to the same levels of ICU noise as patients who are not in isolation rooms. Furthermore, precautions for MRSA include wearing isolation garb when in the patient room (gown, gloves, and masks), and decontamination of any equipment that has been in the patient room with a bleach solution. Although these precautions would not preclude using polysomnography to monitor sleep in these patients, they would certainly impact the ease of use of the equipment.

Relocation of patients frequently occurs in this ICU setting. It is not uncommon for patients to be moved from one area to another within the ICU. This is usually done to accommodate staffing issues, such as patient coverage while nurses are on breaks. Patients may also be moved for other reasons, for example, isolation precautions may be
required. Depending on where and when a patient is moved, there may be different environmental conditions such as noise that would impact his or her sleep.

To appropriately address logistical considerations such as differences in patient care activities, relocation of patients, and infectious disease precautions, one needs to consider the level of support there exists for a research project such as this. These issues could be effectively minimized, and in some cases, eliminated, with a research facilitator in place. The research facilitator could be responsible for working with nurse educators, clinician, managers, physicians and medical residents to help ensure ongoing support for the study.

Technical issues included difficulties with electrode application and obtaining adequate electrical signals. The application of electrodes on the particular participants in this study was difficult due to their limited mobility, attached equipment, and muscle stiffness. Assistance from the attending nurses was required to lift and hold the participants’ heads in position for electrode application. The researcher was well-known as a former staff nurse in the unit in which the study took place, so accessing the attending nurses’ assistance was not an issue. However, if a researcher unknown in a particular unit was to attempt the same study, accessing help for positioning patients might be problematic. Again, utilizing a research facilitator may help gain nurses’ support for this type of study.

High electrical impedances resulted in difficulty obtaining an adequate electrical signal from Jane Doe. This problem may have been circumvented by using collodian, an adhesive substance that is used to firmly attach electroencephalography leads to patients. Collodian was not used in this study for two reasons. First, this substance has a very strong odour that some of the ICU staff found particularly objectionable. Second, when
collodian is used, it must be dried with the use of an air compressor. The researcher encountered difficulty in obtaining the use of an air compressor that could pass a biomedical engineering inspection for use in the ICU. For these reasons, and since the researcher was monitoring the equipment overnight and thus was available to deal with problems as they arose, it was decided not to use collodian.

Suitability of the Research Method

The overall suitability of the selected research method warrants discussion, since some of the difficulties in data collection were a result of the design itself. Additionally, a commentary on the appropriateness of the inclusion criteria is provided, given the challenges in recruiting participants.

The quasi-experimental, single-subject, time series design was chosen for this study for a number of reasons. A single subject design such as this requires each participant to serve as their own control, thereby eliminating the need for finding a comparable control group (Christensen, 1991). Using a single-subject design appeared to be the best choice, as the critically ill population is so diverse that it would be difficult, if not impossible, to obtain comparable intervention and control groups. The ABA design of this type of study required a baseline measurement, intervention measurement, then another measurement after the intervention to see if there was a return to baseline in the dependent variable of interest. This design was specifically chosen for two reasons. The pre- and post- measures allows for determining causality with greater strength than a post-test only design does. The design also required only three nights of study. A stronger design is the ABAB design, in which a fourth measurement is done with the intervention again applied. However, there were many logistical difficulties in
completing three nights of data collection, and adding a fourth night of data collection may not be possible. Furthermore, maturation is already a significant threat to internal validity with three nights of data collection, as is clearly seen with Jane Doe’s case. Adding a fourth night would only serve to increase the odds of a rival hypothesis, such as a change in the participant’s condition, causing an effect on sleep rather than the intervention. Increasing the number of nights of measurement would also serve to increase costs. Given the realities of patient conditions and illness trajectories in the critical care unit, collecting data over three nights is probably maximal.

One suggestion for a future larger study that would increase the strength of its design is to concurrently measure noise levels while PSG recordings are being monitored. The relationship of the noise levels to the PSG recordings would provide a stronger indication of the effect of earplug use in reducing noise to promote sleep. Measuring noise levels would also help control for the differences in noise levels from night to night. However, this additional aspect of a study would serve to increase its costs.

This research design did not allow for the accrual of data regarding the participants’ subjective experience of sleeping in an ICU with and without earplugs. Since a large part of the experience of sleep and sleep deprivation is individually perceived and evaluated (Hauri, 2000; Urden et al., 2002), it may be useful to gather qualitative information from the participants regarding their subjective experience.

The inclusion criteria chosen for this project included a Glasgow Coma Scale score of 10 or greater, English language proficiency, no history of deafness, no history of neurological dysfunction, no history of sleep disorders, receiving mechanical ventilation, having spent at least one night in the ICU, lorazepam equivalents of less than 10
Participants also had to be at least 18 years of age, and consent to participate was provided by the participant or family member. As discussed earlier, participants had to be proficient in the English language, because the researcher in this case is only fluent in English, and the monitoring and intervention required the researcher to physically touch the participants. Participants also had to be receiving mechanical ventilation, as this was the population of interest. Deafness was an exclusion criterion because the intervention, earplug use, requires that participants are able to hear in order to have an effect. Known sleep disorders would have confounded the interpretation of the PSG results, so it was decided that having a known sleep disorder was an exclusion criterion. The same rationale applied to neurological dysfunction, since the EEG portion of the PSG monitoring could be difficult or impossible to interpret in terms of sleep if the brain signals were not normal. Having spent at least one night in the ICU was considered necessary to reduce or eliminate the possibility of patients' not sleeping because of being in unfamiliar surroundings.

The remaining criteria were based on Cooper et al.'s (2000) description of critically ill patients in whom it is appropriate to study sleep. These authors developed their description after studying sleep in critically ill patients requiring mechanical ventilation. Their criteria specifically included the GCS score of 10 or greater, as well as an Acute Physiology and Chronic Health Evaluation (APACHE) score of less than 13, lorazepam equivalents of less than 10 mcg/kg/hour and morphine equivalents of less than 10 mcg/kg/hour. These cutoffs approximate the point estimate of patients who did not manifest sleep as it is normally defined. It was elected not to include the APACHE score
as an inclusion criterion in this study for two reasons. Since APACHE scores are not routinely assigned in the ICU in which the study took place, requiring scoring to be done would have added a cumbersome task to the identification of potential participants.

Second, the GCS is a heavily weighted component of the APACHE score, and as such, APACHE results often reflect changes in the GCS (Cooper et al., 2000; Lemeshow & Le Gall, 1994). Thus, the GCS alone should be adequate. However, if APACHE scores are available, including them as an inclusion criterion in a future study may be reasonable.

It was also decided to deviate from the authors’ suggestion that a maximum of morphine 10 mcg/kg/hour equivalents in the preceding 24 hours be used as an inclusion criterion. As discussed earlier, this upper limit is extremely low for a standard adult dose of morphine equivalents (J. DeLemos, personal communication, August 12, 2002), and as such, would have essentially precluded any patients who received any amount of narcotic from participating in the study. Furthermore, a discussion with one of the authors of the study revealed that the participants in whom the researchers were able to measure sleep received up to 30 mcg/kg/hour. The limit of 10 mcg/kg/hour was arbitrarily chosen (P. Hanly, personal communication, August 16, 2002). Therefore, it was decided in concert with the clinical critical care pharmacist that the upper limit of narcotic for this study would be 30 mcg/kg/hour (J. DeLemos, personal communication, August 17, 2002). In general, sicker critically ill patients require proportionately more narcotic medications than less sick critically ill patients (J. Fenwick, August 17, 2002). Increasing the upper limit of morphine equivalents to 30 mcg/kg/hour may allow some “sicker” patients to be included in the study whose physiologic statuses do not permit sleep study, or who may experience a major physiologic event, as in the case of Jane Doe. However, minimizing
the upper limit of morphine equivalents to 10 mcg/kg/hour may be problematic, since this dose is extremely low. It has been suggested by some of the same authors that about 50% of patients in a general ICU would meet these criteria (Gabor et al., 2001), but this was not this researcher's experience. Maintaining the upper limit of morphine equivalents at 30 mcg/kg/hour as an inclusion criterion in a future study would allow a greater number of participants to be eligible, and in theory should allow for proper PSG study. It may, however, be desirable to plan a study in such a way to allow for comparisons of participants' sleep based on the amount of morphine equivalents they receive.

**Implications for Future Research**

An extensive literature review and the process of this feasibility study have identified several important areas for future research. Despite the burgeoning field of sleep research and sleep medicine, there is a paucity of research that is directed at the sleep of critically ill patients. Although it is well documented that critically ill adults do not receive enough sleep, either in terms of quantity or quality, there is a scarcity of research that looks at interventions and strategies to improve sleep in this population. Other strategies may be more or less effective than earplug use, but until interventions are developed and tested, there is no way to know this. In the meantime, a larger, adequately powered study to test the effects of earplug use on the sleep of critically ill adults is warranted.

Studies are needed that clearly link sleep and sleep deprivation in critically ill adults with specific outcomes such as wound healing, immune function, respiratory function, and mental health. Studies should also be undertaken to explicate the relationships
between sleep and sleep deprivation in critically ill adults and other outcome measures such as morbidity, mortality, length of stay in critical care, and quality of life.

It has been well documented that the sleep of critically ill patients is frequently interrupted for patient care activities. This feasibility study clearly highlighted this finding, especially in Jane Doe's case. Many of these activities were either not immediately necessary for the participant’s health and well being, or they were uncoordinated so that the participant was disturbed multiple times. This observation is congruent with the assertion of some authors that despite being aware of the importance of sleep, critical care nurses do not necessarily differentiate between essential and nonessential tasks to permit uninterrupted time for sleep, nor do they attempt to control the environment to promote sleep (Morgan & White, 1983; Pulling, 1991). Study is needed to determine the reasons why nurses do not actively promote sleep within the critical care environment. Studies that explore the feasibility and effectiveness of changing the timing of patient care activities to promote sleep in critically ill patients are also needed.

Implications for Nursing: Points to Consider

While this feasibility study does not allow for any conclusions to be drawn as to the effectiveness of earplug use in the critical care setting, the study process itself and the findings have implications for critical care nursing. These are identified and briefly discussed.

The process of this feasibility study demonstrated that critical care nurses in this unit interrupt patients’ sleep on a frequent basis for patient care activities. Many of these activities are either not essential, or are not coordinated with other care activities. It is
important that critical care nurses consider the sleep of patients as a priority in their practice, so that they undertake actions that actively promote sleep. Some of these nursing actions include grouping patient care activities to allow for uninterrupted blocks of time for sleep, and distinguishing between essential and nonessential tasks to minimize disruptions when patients do sleep. These nursing actions are very basic, and are likely already well known by critical care nurses. However, since these actions are not regularly undertaken in practice, ways to present sleep needs as a priority for nursing care must be considered. To this end, clinical nurse educators, clinicians, and other resource personnel must actively educate critical care nurses about the importance of sleep, and provide support for changing practice to accommodate sleep as a priority. Currently, most teaching in relation to sleep in critically ill adults is anecdotal and not a large part of critical care nursing curricula (E. Shackell, personal communication, January 12, 2003). Little information describing the boundaries of the research based knowledge of sleep in critically ill adults is disseminated. For these reasons, few staff nurses and nurse educators and clinicians understand that rigorous studies of interventions impacting sleep have not been done. Thus, they may not fully appreciate the need for research and their role in facilitating this work. Nurse educators must inform themselves and others of the limits of existing knowledge, and the need to expand research based knowledge in this area.

The implementation of a large scale study of the effect of earplug use on the sleep of critically ill adults will require the cooperation and support of all levels of nursing administration in creating and fostering an environment that is conducive to, and
supportive of, this type of research. This includes fostering a collaborative working relationship with physicians.

Finally, although no conclusions regarding the effectiveness of earplug use in promoting sleep in critically ill adults can be drawn from this particular feasibility study, subjective anecdotal evidence from John Doe suggests that earplugs may improve some patients' perception of their experience of sleep in the critical care unit. Since earplug use has essentially no known contraindications or associated risks, it is reasonable for nurses to offer earplugs to critically ill patients as part of sleep promotion.

Summary

The feasibility of undertaking a study of the effect of earplug use on the sleep of critically ill adults has been studied. The need for a feasibility study was originally based on the author's clinical impression that critically ill patients were subjected to a variety of circumstances that would likely preclude sleep. This led to such questions as, "Is it possible to study the effect of an intervention on sleep?" and "Are there enough critically ill adults in whom it is possible to test an intervention to warrant a study?" The effect of earplug use on the sleep of critically ill adults was unknown. Also unknown were the pitfalls of sleep research as it related to the recruitment of subjects, the logistics of conducting such a study, and the suitability of both the intervention, earplugs, and the data collection tool, the polysomnography system as well as the research design.

A study designed to answer the questions of feasibility was implemented in the Vancouver General Hospital Main Intensive Care Unit. Prior to the implementation of the study, the protocol was submitted for ethical review to the University of British
Columbia and the Vancouver General Hospital Ethics Review Board. Two participants were enrolled in the study.

Analysis of the study data resulted in a number of recommendations for future studies. First, it was demonstrated that a larger study testing the effect of earplug use in critically ill adults is needed, and that it is feasible to conduct such a study if sufficient funding is obtained. Second, it is suggested that the use of a research facilitator be considered. This should not only resolve some of the barriers encountered in recruitment of subjects, but should also overcome some of the logistical issues with nursing and medical support for the project. Third, the access to interpretive services needs to be available if such a study is to be attempted in the same setting. Fourth, it is recommended that noise levels are measured concurrently with the polysomnographic sleep measurement. Fifth, the eligibility criteria needs to be carefully considered when planning a future study. It is recommended that the upper limit for morphine equivalents be set at 30 mcg/kg/hour, but the study design should allow for comparisons to be made of participants' sleep based on the number of morphine equivalents they receive. Finally, qualitative data regarding the participants' subjective experience of sleep should be collected to further inform the study.
References


