

Temperature Patterns in Pediatric Intensive Care Patients

By

Karen L. Sonnenthal, RN, BSN

A Master's Research Project

Presented to

Oregon Health Sciences University

School of Nursing

in partial fulfillment of

the requirements for the degree of Master of Science

May 12, 1997

APPROVED:



Linda Felver, Ph.D., R.N. Research Advisor



Brahm Goldstein, MD, FCCM, Committee Member



Marsha Heims, R.N., Ed.D., Committee Member



Beverly Hoeffler, R.N., D.N.Sc., F.A.A.N., Interim Associate Dean for Graduate Studies

ACKNOWLEDGMENTS

I wish to thank Dr. Linda Felver for her motivation and enthusiasm for research, and for her endless dedication to her students. I wish to thank Dr. Marsha Heims for her flexibility and motivation.

I also wish to thank the staff of the Pediatric Intensive Care Unit at Doernbecher's Children's Hospital, for their assistance with data collection, and their support during this past year.

Last, I wish to acknowledge and thank Dr. Brahm Goldstein for his friendship, his support, and his guidance.

ABSTRACT

TITLE: Body Temperature Patterns in Pediatric Intensive Care Patients
AUTHOR: Karen L. Sonnenthal, RN, BSN
APPROVED: [REDACTED]-----
Linda Felver, Ph.D., R.N.

ABSTRACT

Objective: The primary aim of this study was to describe the patterns of body temperature over forty-eight hours in selected patients in a Pediatric Intensive Care Unit. The secondary aim was to examine the relationship between identified patterns in body temperature fluctuations and the subject's severity of illness and outcome.

Design: A descriptive (time-series) design was used.

Setting: A 10-bed pediatric intensive care unit (PICU) in a tertiary care children's hospital.

Subjects: 8 PICU patients (3F / 5M) age 3 - 17 years (mean \pm SD = 8 \pm 4.70 years).

Measurements and Main Results: Bladder temperature was measured every 5 minutes for 22-53 hours using a Mon-A-Therm Foley Catheter with temperature sensor. The temperature data were graphed over time and a fast Fourier transform analysis was done to search for the major periodic components. The temperature fluctuations were then compared to the subject's reported prehospital customary sleep and awake times.

Remnants of the estimated prehospital temperature patterns were noted and compared to the severity of illness using the Pediatric Risk of Mortality (PRISM) score and to outcome.

Outcome was determined by a change in Pediatric Cerebral Performance Category or Pediatric Overall Performance Category from hospital admission to discharge. Three subjects retained their prehospital body temperature pattern during customary sleep time, and three subjects retained their prehospital body temperature pattern during customary awake time. However, only two subjects retained their prehospital body temperature

patterns during both customary sleep and awake time. Subjects with higher severity of illness and poor outcomes retained fewer components of their prehospital temperature pattern than subjects with lower severity of illness and good outcomes.

Conclusions: This study provided an initial description of body temperature patterns in pediatric patients in the PICU. Graphical analysis of the subjects according to their severity of illness and outcome indicated that there may be a relationship between alterations in body temperature patterns and severity of illness and outcome. Knowledge gained from research on body temperature patterns has potential clinical applicability in monitoring and assessment of critically ill patients in the PICU.

TABLE OF CONTENTS

	Page
APPROVAL PAGE	ii
ACKNOWLEDGMENTS.....	iii
ABSTRACT	iv
LIST OF TABLES.....	ix
LIST OF FIGURES.....	x
CHAPTER I: INTRODUCTION.....	1
Statement of the Problem.....	1
CHAPTER II: REVIEW OF THE LITERATURE.....	5
General Principles of Biological Rhythms.....	5
Endogenous Influences....	5
Exogenous Influences	6
External Desynchronization	7
Internal Desynchronization	8
Summary	8
Development of the Circadian System	9
Thermoregulation	11
Body Temperature and Thermoregulatory Rhythms	12
Temperature Rhythms in Infants and Children	14
The Environment of the Intensive Care Unit	16
Overt Temporal Patterns in the ICU	18
Severity of Illness and Outcomes in the Pediatric Intensive Care Unit	24
Research Question	26

CHAPTER III: METHODS	27
Design	27
Sample and Setting	27
Instruments	29
Bladder Temperature Measurement	29
Data Collection Forms	29
Data Collectors.....	30
Procedures	30
Protection of Human Subjects	32
Analysis of Data	32
Fast Fourier Transform and Periodogram Analysis	32
Graphical Analysis	33
CHAPTER IV: RESULTS	36
Sample	36
Statistical Analysis of Individual Subjects	42
Descriptive Statistics	42
Fast Fourier Transform and Periodogram Analysis	44
Graphical Analysis of Individual Subjects	44
Summary of Individual Analysis	58
Graphical Analysis by Groups of Subjects	58
Graphical Analysis According to Diagnosis	58
Graphical Analysis According to Admission PRISM.....	60
Graphical Analysis According to Outcome	62
Summary of Graphical Analysis of Groups	64
Statistical Analysis of Groups	64
Summary of Results	64

	viii
CHAPTER V: DISCUSSION	66
Analysis of Individual Subjects	66
Fast Fourier Transform and Periodogram Analysis	71
Analysis of the Study Group as a Whole	75
Significance to Clinical Nursing Practice	77
Future Research	79
Conclusion	80
REFERENCES.....	81
APPENDICES	88
A Informed Consent Form	88
B Subject Screening Tool and Subject Information Record.....	91
C Example of Environmental and Event Data Collection Form.....	94
E Worksheets Used in Graphical Analysis	96

LIST OF TABLES

Number		Page
1	Summary of Research on Body Temperature Rhythms in Adult Patients in an ICU: Medical Patients	20
2	Summary of Research on Body Temperature Rhythms in Adult Patients in an ICU: Surgical Patients	22
3	Summary of Subject Characteristics, Diagnoses, PRISM scores, and Outcomes	37
4	Summary of Temperature Ranges for each Subject.....	43
5	Patterns in Body Temperature Rhythms During Customary Sleep and Awake Times for Subject Group Divided by Diagnosis	59
6	Patterns in Body Temperature Rhythms During Customary Sleep and Awake Times for Subject Group Divided by PRISM Scores.....	61

LIST OF FIGURES

Number		Page
1	Temperature Graph: Subject 1	50
2	Temperature Graph: Subject 2	51
3	Temperature Graph: Subject 3	52
4	Temperature Graph: Subject 4	53
5	Temperature Graph: Subject 5	54
6	Temperature Graph: Subject 6	55
7	Temperature Graph: Subject 7	56
8	Temperature Graph: Subject 8	57

CHAPTER I

INTRODUCTION

Many physiological variables fluctuate over time in healthy individuals living in their standard environment. These biological rhythms have a period of around twenty-four hours, and are thus termed circadian rhythms. They are endogenous rhythms that are transiently effected by specific environmental stimuli (masking effect), and are entrained (synchronized) by the patterns of the external environment. In other words, the timing of the endogenous rhythm is synchronized by the timing and pattern of the environment. In humans, the most influential environmental rhythm is the alternation between day and night (Minors & Waterhouse, 1981).

An example of such a biological rhythm is the temporal fluctuation in body temperature, which has been well described in normal adults (Minors & Waterhouse, 1981), and infants (Schechtman & Harper, 1991). In nursing practice, body temperature is a variable that is measured frequently in clinical settings and its value is used as a basis for therapeutic decisions. Studies have shown that body temperature has a stable rhythm that adjusts slowly to changes in the environment. It is considered to be one of the most reliable rhythms and is therefore used as a marker rhythm in chronobiology (Clancy & McVicar, 1994). Because of its stability, it can be assumed that if the body temperature rhythm is altered, it is highly probable that other rhythms have also been altered.

Statement of the Problem

Changes in biological rhythms have been documented in adults (Minors & Waterhouse, 1986). When external time cues are altered or removed completely from the environment, shifts occur in the phase relationships between the individual's endogenous rhythms and the environmental cues. This process is termed external desynchronization (Felton, 1987). Internal desynchronization occurs when the internal rhythms take on periods that are distinct from that of other internal rhythms and occurs because internal

rhythms shift in response to a changed environment at different rates (Minors & Waterhouse, 1986).

Research studies on the environment of an adult Intensive Care Unit (ICU) have found that the ICU does not have standard day-night patterns (Wilson, 1987). When individuals are placed in an environment with time patterns that are different from the environment to which their biologic rhythms are entrained, their rhythms begin to resynchronize to the new environment. But because different biologic rhythms also re-entrain at different rates, they become out of phase with the other biologic rhythms. This causes internal rhythms to be internally desynchronized and clinically the individual may experience fatigue, malaise, gastrointestinal disturbances, and more frequent occurrence of infection. When this factor is combined with the patients' unstable physiology due to their illness or injury, there is increased chance of alterations in the biological rhythms of patients in ICUs (Felver, 1995).

Disruption of biological rhythms has been demonstrated in several studies of hospitalized patients. In a study of postoperative patients, Farr, Keene, Samson, & Michael, (1984) found that temperature and heart rate rhythms of these hospitalized subjects were altered. Felver (1991) found that temperature rhythms had a period of less than twenty-four hours in adult medical ICU patients. Moore-Ede, Czeisler, & Richardson, (1983) concluded that with the constant levels of noise, light, and activity, even patients with normal phase orientation on admission have some disruption after a few days. As noted earlier, due to the stability of the temperature rhythm, if it is altered it is highly probable that other rhythms have also been altered.

The significance of these results is suggested in studies of circadian rhythms in postoperative individuals by Farr, Keene, Samson, & Michael-Jacoby (1986) and in similar work in postoperative rats (Farr, Campbell-Grossman, & Mack, 1988). These studies found that humans and rats with postoperatively altered rhythms also had delayed

recovery from surgery. In addition, Moore-Ede, Sulzman, & Fuller (1982) noted that the body's circadian timing system influences the responsiveness of each system to challenges. When the system is desynchronized, it decreases the body's ability to respond to additional stressors.

Therefore, for the patient in an ICU, desynchronization of rhythms is likely to lead to delayed recovery (Farr et al., 1984), and an increased incidence of nosocomial infections (or increased susceptibility to infection (Felver, 1995). These factors could then lead to increased length of stay, increased time away from family, and family stress. An additional consideration in children is that an increased length of stay may adversely affect the child's growth and development.

The significance of these findings to nursing lies in the following. Assessment and interpretation of temperature is an important nursing function and responsibility in ICU's (Felver, 1991). One of the primary objectives of continuously monitoring physiological parameters is the detection of changes indicative of homeostatic instability so that corrective measures can be implemented. Another objective is to detect patterns or trends which might be predictive of clinical outcomes (Lanuza, Robinson, Marotta, & Patel, 1989). Knowledge of an individual patient's baseline fluctuation in temperature is important in interpreting vital signs and in making clinical decisions based on that interpretation (Woods, 1991). If the nurse is aware of the potential for altered circadian rhythms, then close assessment for altered signs and symptoms (such as sleep disruption, gastrointestinal disturbances, and a general feeling of malaise) in order to determine the individual's needs and anticipate problems arising from circadian disturbance can be implemented (Farr et al., 1988). Spencer and Bale (1990) suggest that circadian patterns can influence the healing of surgical wounds. Nursing measures such as grouping activities during the night to limit sleep disturbance, and providing environmental cues which are appropriate to the individual's prehospitalization life pattern may help to prevent the disruption and or

facilitate a return to the individual's rhythmic profile. The end goal is to promote the rapid restoration of the patient to an optimum level of functioning (Ballard, 1981).

The results from adult studies indicate the occurrence of alterations in temperature rhythms in adult patients in an ICU. To date, little research has been done in this area in a pediatric population. Descriptive studies which would identify alterations in circadian rhythms of pediatric patients in a Pediatric Intensive Care Unit are therefore needed.

CHAPTER II

REVIEW OF LITERATURE

General Principles of Biological Rhythms

A rhythm is defined as a sequence of events that repeats over time in the same order and at the same interval. A period is the time to complete one cycle. Biological rhythms are rhythmic changes in variables that arise from within the organism. If the period of a biological rhythm is twenty to twenty-eight hours, it is termed a circadian rhythm. If the period is less than 20 hours, it is termed an ultradian rhythm; if longer than 28 hours, it is termed an infradian rhythm. The term phase is used to describe the position of the rhythm in time, or in relation to another rhythm. The acrophase of the rhythm is the timing of the peak of the rhythm (Minors & Waterhouse, 1981).

Endogenous Influences

Circadian rhythms have been documented in almost every species (Minors & Waterhouse, 1986). For most living organisms, when in their customary environments, their predominate rhythms seem to oscillate with a frequency which corresponds to a major environmental periodicity. For man, the most evident environmental rhythm is the alternation between day and night (Minors & Waterhouse, 1981). Circadian rhythms, however, do not originate from external influences on the organism. Instead, they are endogenous rhythms which are under the innate control of the organism. Proof of this came from past research where subjects were maintained in constant conditions--i.e. the noise, temperature and lighting of their environment was held constant, they were kept awake, and they were fed small meals every hour to eliminate the rhythmic effects of normal mealtimes. These studies found that most circadian rhythms did continue to oscillate, proving that they were not simply due to changes in the environment (Minors & Waterhouse, 1986).

Due to the nature of these studies, however, their duration was limited. Another line of research maintained the individual subjects in isolation units which eliminated all environmental time cues. They were also allowed to eat and sleep which eliminated the intervening effects of sleep deprivation but were offered no cues as to when to eat or sleep. It was assumed that in the absence of external time cues, the sleep-wakefulness cycle and other rhythms would be determined by endogenous influences alone. As in the previous studies, these experiments found that the circadian rhythms persisted. In addition, however, they noted that the rhythms had a mean period of twenty-five hours and were no longer synchronized to the solar day. Biological rhythms with periods that deviate from an exact twenty-four hours and are therefore no longer synchronized to the solar day are said to be 'free-running' (Minors & Waterhouse, 1986). The significance of this occurrence will be discussed later.

Further evidence of the endogenous nature of biological rhythms can be found in the animal studies done by Moore and Len (1972), and Moore and Eichler (1972). These early studies found that destruction of the suprachiasmatic nuclei resulted in the apparent loss of certain circadian rhythms in rodents (Moore-Ede, Czeisler, & Richardson, 1983). Since then, more sophisticated studies on mice by other investigators have supported their results (Minors & Waterhouse, 1986).

Exogenous Influences

Circadian rhythms, however, are not completely independent from external stimuli. Instead, they can be influenced by the environment in two ways. The first effect is seen when the external stimulus has its own rhythmic quality. These rhythmic environmental influences, or zeitgebers, can synchronize the body's endogenous rhythms (Minors & Waterhouse, 1986). The process of synchronization by zeitgebers is termed entrainment (Minors & Waterhouse, 1981). The most reliable and potent zeitgeber is the regular diurnal

alternation of light and darkness although social cues and the timing of meals also act as strong zeitgebers (Moore-Ede, et al., 1983).

The second type of environmental influence produces an exogenous component of the rhythm as well as changes in the rhythm that are transient. These effects occur in response to some environmental stimulus. In man, the main exogenous influence is the normal rhythm of sleep and wakefulness (Minors & Waterhouse, 1986). Sleep produces direct changes in physiological variables such as the secretion of growth hormone and cortisol, as well as changes in body temperature. As a result, sleep is said to 'mask' the effects of the internal clock (Minors & Waterhouse, 1986). In addition, environmental stimuli can exert transient changes. Examples of this stimulus-response type of environmental effect include an increase in heart rate that occurs with pain, or a loud noise which disrupts sleep (Folger, 1995). In summary, external rhythmicities exert effects on circadian rhythms in two ways: by entraining the internal rhythm, and by producing an exogenous component. In addition, many of the zeitgebers for man are the very same influences that are responsible for the exogenous component of the rhythm (Minors & Waterhouse, 1986).

External Desynchronization

Under normal circumstances, exogenous and endogenous influences are in phase with one another. An example of this can be found in the rhythm of body temperature which peaks in the daytime as a result of the body clock entrained and accentuated by diurnal activity. When environmental time cues are altered, however, shifts occur in these phase relationships. Examples of this process occur during travel into a different time zone, or when workers vary shifts between days and nights. During these situations, the measurable consequences associated with the change of sleep and activity patterns are that internal rhythms shift out of phase with external cues such as social routines and the sleep-wake cycle which normally act to synchronize the internal circadian rhythms. This process

is termed external desynchronization (Felton, 1987). Clinically, the individual experiences sleepiness and wakefulness at environmentally inappropriate times.

Internal Desynchronization

When internal desynchronization occurs, an individual's rhythmic structure can change so that some internal rhythms show a period distinct from that of other internal rhythms and the phase relationship between groups of rhythms is lost. This can take place within one to two days, and occurs because internal rhythms shift at different rates. This process is termed internal desynchronization (Minors & Waterhouse, 1986). In this case, the individual may experience fatigue, malaise, decreased mental alertness, impaired performance, and more frequent occurrence of infection (Felver, 1995).

Internal desynchronization was also noted in the research studies of individuals in isolation, where their internal rhythms were free-running due to the lack of environmental cues. These experiments found that when rhythms were left to be free-running, there was spontaneous internal desynchronization, where two rhythms in the same individual continue oscillating, but with different periods. In summary, endogenous rhythms can be desynchronized from the environment (external desynchronization) as well as from other rhythms (internal desynchronization) (Minors & Waterhouse, 1986).

Summary

In summary, circadian rhythms are endogenous rhythms which are both synchronized and modified by the environment. In a person's usual environment, the endogenous and exogenous components are in phase with each other. When the individual's environment is altered, desynchronization between the rhythms and the environment as well as between the rhythms themselves can occur. Many circadian rhythms regulate physiologic function, and therefore influence health and susceptibility to disease (Sorensen & Luckmann, 1986). In humans, examples of physiological rhythms include body temperature rhythms, heart rate rhythms, blood pressure rhythms, hormone

and enzyme rhythms, and urine flow rhythms (Dobree, 1993). The body temperature rhythm is very stable, and is not readily affected by changes in the environment. Because of this, it is often used as a marker rhythm in chronobiology studies (Samples, Van Cott, Long, King, & Kersenbrock, 1985).

Development of the Circadian System

The development of the circadian system has been studied by looking at biological rhythmicity in full term and preterm infants. Hellbrugge's (1974) studies in the 1960's revealed several important principles: 1. The development of physiological and behavioral rhythms in infants is characterized by a shift from polyphasic to monophasic cycles; 2. Different physiological functions develop circadian rhythmicity independently; and 3. The amplitude of rhythmic oscillations increase with increasing postnatal age. Since then, numerous studies of preterm and full term infants have demonstrated circadian variation in body temperature, blood pressure, and the rest-activity cycle (Schechtman & Harper, 1991). These rhythms were also present in very premature infants, although the rhythms appeared and disappeared in successive weeks, indicating minimal development of circadian rhythmicity at this age (Tenreiro, Dowse, D'Souza, Minors, Chiswick, Simms, & Waterhouse, 1991). In addition to these infant studies, fetal studies found evidence of circadian rhythms in utero (Schechtman, & Harper, 1991). Results of these studies showed circadian rhythms during the last trimester in several variables including body movement, respiratory movements and heart rate. It is believed that such rhythmicity is not totally generated by the fetus' own endogenous mechanisms, but is primarily entrained by the maternal environment (Tenreiro, et al., 1991). However, the fact that acrophases of rhythms in neonates may differ from those of their mothers indicates that the endogenous oscillators may already be effective in neonates (Sitka, Weinert, Berle, Rumler, & Schuh, 1994).

After birth, circadian rhythms develop slowly, and at different post-natal ages. Studies of preterm infants found low amplitude circadian rhythms for rectal and skin temperatures, as well as heart rate and activity. The rhythmicity in each variable was dominated by ultradian periodicities that were coincident with feedings and related interventions (Glotzbach, Edgar, & Ariagno 1995). In contrast, Glotzbach, Edgar, Boeddiker, & Ariagno (1994) studied ten healthy full term infants in the infant's home for three consecutive days at one month and three months of age. Their results demonstrated circadian periodicity for rectal temperature, heart rate, and activity at one month of age with a significant increase in the amplitude of these rhythms at three months. These differences were highlighted by an increase in activity during the day, and a decrease in the rectal temperature during the night at three months compared to one month. At the same time, there was an increase in the interval between feedings at three months of age which corresponded with an increased consolidation of sleep during the night. They concluded that the subtle changes in the nature and interaction of infant variables may reflect maturation of the circadian system and its coupling with homeostatic effector systems that are developing in parallel (Glotzbach et al., 1994). Weinert, Sitka, Minors, & Waterhouse (1994) found similar results in their study of eleven healthy, full term babies on the second day after birth and then at one month of age. Their study noted that while circadian rhythms in heart rate and blood pressure were poorly developed at day two and at one month of age, there was a circadian rhythmicity in rectal temperature on day two which increased in amplitude by one month. This corresponded to an increase in deep sleep between day two and one month. They concluded that during the first four weeks of life, both endogenous and exogenous components of the circadian rhythms increased in amplitude and the rhythms became more synchronized. In addition, they concluded that their results support the evidence that the suprachiasmatic nuclei are present and functional

towards the end of a normal pregnancy and that the process of entrainment develops separately (Weinert et al., 1994).

Thermoregulation

Body temperature is the result of a balance between heat production and heat loss. Information on body temperature is provided by thermoreceptors throughout the body in addition to temperature-sensitive neurons in the anterior hypothalamus. In a normal person, the set point in the hypothalamus determines body temperature at any particular time. Inputs from the thermoreceptors are integrated by the hypothalamus which stimulates behavioral and physiological mechanisms to regulate body temperature by adjusting heat production and heat loss (Cohen, & Sherman, 1988). Behavioral regulation involves conscious, voluntary efforts such as the application or removal of clothing, obtaining shelter, or regulation of one's own environment. Physiological regulation is under the control of the autonomic nervous system. When sensors indicate that the body is too cool, heat is produced by metabolism, increased muscle activity, the effect of increased thyroid hormones, and increased activity of the sympathetic nervous system. Heat loss is restricted by the autonomic responses of piloerection and constriction of cutaneous vessels. In contrast, when the body is too warm, heat loss occurs by sweating and dilation of the cutaneous vessels which enhances the processes of radiation, convection, conduction, and evaporation.

Under varying environmental conditions, behavioral and physiological regulation occur via these mechanisms in order to balance heat loss and heat production and thereby maintain body temperature. For example, if a person is cold, heat production may be increased and heat loss diminished by putting on clothing, along with increased skeletal muscle activity, cutaneous vasoconstriction, and shivering. In contrast, if the person is too warm, heat loss can occur by removal of clothing, dilation of blood vessels, and sweating (Cohen, & Sherman, 1988).

Body Temperature and Thermoregulatory Rhythms

A review of the research on temperature rhythms shows that the temperature of humans living in a standard environment fluctuates over a 24 hour cycle that is influenced by the individual's daily routine of work, meals, rest, and sleep (Minors & Waterhouse, 1981). Specifically, these studies noted that when the sleep-wake schedule was stable, oral temperature was lowest at midsleep, started to rise before waking, peaked eleven to fifteen hours after midsleep, and then began to fall before the onset of sleep.

As with other circadian rhythms, the temperature rhythm at any instant is derived from both its endogenous and exogenous components (Minors & Waterhouse, 1981). The endogenous component was identified during the free-running experiments that were discussed earlier. As indicated, these studies found that the rhythm of body temperature did not disappear when external time cues were eliminated but rather continued with a period of about twenty-five hours (Minors & Waterhouse, 1986). Moore-Ede et al., (1983), however, have also noted that the endogenous component of the temperature rhythm is very stable and does not readily respond to changes in the environment, unlike other rhythms such as heart rate which are easily influenced by environmental stimuli. It is therefore deduced, that the overt rhythm of body temperature has a large endogenous component (Woods, 1991).

In the presence of environmental stimuli, however, the temperature rhythm is synchronized to the environment by the patterning of environmental variations and altered transiently by changes in environmental stimuli (Moore-Ede et al., 1983). In man, the environmental variations that synchronize the endogenous rhythm are the same influences that are responsible for the exogenous component of rhythms. These include the alternation of light and dark, feeding and fasting, presence and absence of social cues, and sleep and activity. Past studies reported the pattern of sleep and wakefulness as the primary exogenous component responsible for synchronization to the environment (Minors

& Waterhouse, 1986). Czeisler et al (1986), however, showed that exposure to bright indoor light can reset the human circadian pacemaker even when the timing of the sleep wake cycle is constant. In addition, more recent work by Boivin et al (1996) demonstrated that light of even low intensity significantly phase-shifts human circadian rhythms. Because most people are generally exposed to low-intensity light, they conclude that exposure to low-light intensities (such as those from artificial light) is probably the principal synchronizer of human circadian rhythms.

As described in the previous section, the hypothalamus monitors body temperature and maintains it by controlling thermoregulatory mechanisms (Cohen, & Sherman, 1988). When this process is extended over time, it becomes apparent that the body temperature results from the balance between heat gain and heat loss. In their review, Minors & Waterhouse, (1981) indicated that it was assumed that since moderate changes in metabolic rate had little effect on temperature rhythms, it was the circadian variation in heat loss that must account for the circadian rhythm in temperature. Previous research, however, argued against this. In his work on thermoregulatory response, Hildebrandt (1976), noted that humans in a normal day routine exhibited two thermoregulatory adjustments to the course of their body temperature rhythm. The first adjustment is the heating up phase which begins as the sleep period ends and lasts into the activity period. During this phase, peripheral vasoconstriction occurs in order to increase the internal body temperature. At the end of the activity period, the cooling-down phase begins and lasts until midsleep. At this point, peripheral vasodilation occurs in order to decrease body temperature. These observations of the rhythmic changes in thermoregulatory mechanisms led to the deduction that the circadian rhythm of body temperature is the result of a changing thermoregulatory set-point in the hypothalamus which is higher in the afternoon than in the morning in day-active subjects (Hildebrandt, 1976, & Minors & Waterhouse, 1981). Hildebrandt (1976) further concluded that at any given time, the physiological response to thermal stimuli was

influenced by the phase of the thermoregulatory adjustment. In brief, Hildebrandt's work showed that rhythmic changes in thermoregulatory mechanisms due to changes in the thermoregulatory set point influence the degree of response to thermal stimuli, and therefore result in a rhythmic variation in body temperature.

In summary, the adult literature has documented well-characterized rhythmic variations in body temperature in healthy adults living in standard environments (Felver, 1995). Various studies have shown that when in a customary environment, an individual's temperature rhythm is synchronized to the environment by the patterning of environmental variations (Moore-Ede et al., 1983) and is influenced by the daily routine of work, meals, rest, and sleep (Minors & Waterhouse, 1981).

Temperature Rhythms in Infants and Children

Little work was found on temperature rhythms in children. Instead, most of the pediatric studies focused on the documentation of temperature rhythms in infants, particularly newborns. This section will present some of the studies done on infants. A brief summary of the available adult literature was presented in the previous section.

Early studies done on newborn infants found that temperature rhythms are absent in the first weeks after birth, then progressively increase in amplitude over about the next five years until the adult value is reached (Minors & Waterhouse, 1981). Subsequent research, however, has produced contradictory results. Lodemore, M., Petersen, S., & Wailoo (1992) noted that changes in rectal temperatures correspond with sleep cycles from as early as the sixth week of life regardless if the infant was asleep during the day or night. However, at around ten weeks of age, the temperature variation increased in amplitude with more variation noted during sleep at night than during sleep which occurred during the day. Brown, Dove, Tuffnell, & Ford (1992) found similar results, however, their study noted an initial decrease in rectal temperature with sleep at four weeks of age. Sitka et al., (1994) found the presence of a circadian rhythm in body temperature in infants as early as age two

days. They further noted that the variance of acrophases for the temperature rhythm decreased with increasing postnatal age and therefore indicate a beginning synchronization with the environment. The differences between the results of these studies can be accounted for by the simple fact that they all studied infants of different ages. All of the authors used full-term infants, however, Lodemore et al., (1992) studied infants from age six weeks, while Brown et al., (1992) looked at infants from age two weeks, and Sitka et al., (1994) studied infants as young as two days of life. This was the only difference in the methodology between the studies since they all used continuous recordings of rectal temperature in order to document the infant's temporal patterns.

In summary, these studies show the presence of a body temperature rhythm from as young as two days postnatally with an increase in the amplitude with increasing age. All of the infants studied were healthy, full-term infants, and recordings were made in their home under standard living conditions. No work was found on the temperature rhythms of infants hospitalized in an intensive care unit, and therefore the effect of the environment of an intensive care on the temperature rhythm is unknown.

The adult literature has documented well-characterized rhythmic variations in body temperature in healthy adults living in standard environments (Felver, 1995). Specifically, a review of the studies noted that the temperature of humans fluctuates over a diurnal cycle that is influenced by the daily routine of work, meals, rest, and sleep (Minors & Waterhouse, 1981). In other words, when in a customary environment, an individual's temperature rhythm is synchronized to the environment by the patterning of environmental variations (Moore-Ede et al., 1983). As noted earlier, however, in the presence of a changed environment the temperature rhythm is noted for its stability, and therefore, when altered, is indicative that other rhythms have been altered as well.

The Environment of the Intensive Care Unit

The role of the environment in relation to human circadian rhythms has been discussed in earlier sections. As part of the discussion, the disruptions in these rhythms (i.e. external and internal desynchronization) which occur as a result of changes in the environment were also noted. Up to this point, the discussion has centered on normal, healthy individuals. When a person becomes ill, however, his / her underlying circadian rhythm may be altered by the effects of severe illness, surgery, or trauma (Moore-Ede, et al., 1983); or by their response to therapy (Felver, 1995). In addition, during severe illness the individual is removed from his /her familiar environment in which the rhythms were entrained, and placed in an environment that lacks customary time cues (Felver, & Pike, 1990). This section will discuss the environment of an Intensive Care Unit (ICU) and conclude with a summary of the impact of an ICU environment on biological rhythms. Most of the studies reviewed were conducted on adults in an Adult ICU since little work has been completed on the environment of a Pediatric Intensive Care Unit (PICU).

The ICU environment is known for its noise level, constant lighting, and lack of patient control. Wilson (1987) noted that patients in an ICU identified the noise level and the inability to track time as positive stressors of the ICU. In an early study of noise, Falk & Woods (1973) documented the noise levels in infant incubators, and in rooms of an ICU. They used sound level meters to record the noise levels in these areas, in order to determine if they exceeded fifty decibels which is a level that has a twenty-five percent probability of affecting sleep. Their recordings found that the noise levels in the incubator and in the ICU exceeded fifty decibels. In a similar study of the noise level in adult ICUs, Hilton (1985) found that the noise levels continued to range between forty-eight and sixty-eight decibels. Felver & Pike (1990) recorded measurements of the sound level near the head of their subject every minute in their 24-hour study of the relationship of physiological rhythms of a mechanically ventilated patient to environmental variables in an intensive care

unit. Their measurements showed rhythmic variations in the sound level with a 24 hours mean / time (mesor) sound level equal to 62 dB.

In addition to these results, Topf's (1992) review of studies on the environment of ICUs noted that the sound levels in the ICU negatively affected the patient's ability to sleep. Specifically, sleep deprivation occurred as a result of a longer latency to sleep, more stage shifts, and less sleep during the night due to the noise levels. In their study of sleep patterns of seven male patients in an ICU, Felver & Hoeksel (1993) found that the mean total amount of uninterrupted time available for sleep during the patients usual sleeping time was 4 hours 37 minutes (\pm 69 minutes). In addition, they found that only three of the seven subjects had interrupted sleep intervals that were greater than sixty minutes. The other four subjects had uninterrupted sleep intervals that were greater than sixty minutes. The other four subjects had uninterrupted sleep intervals that were less than forty-five minutes. For all subjects, sleep was interrupted by patient monitoring activities, interventions, and standard care procedures of the unit. These results indicated that patients in an ICU had no more uninterrupted time during their usual sleeping time than during their usual awake time, and that some patients do not even have the opportunity to complete a standard sleep cycle of sixty to ninety minutes.

Baker (1984) noted that in addition to the noise level, the various environmental stimuli in an ICU such as continuous lighting, crowding with unfamiliar people, unpleasant smells, and disturbing or painful touch, contribute to sensory overload. This can then disrupt the patient's processing and decrease the meaningfulness of the environment. Last, Campbell, Minors, & Waterhouse (1986), described the ICU environment as one that is monotonous with severely limited visual and auditory inputs.

From these studies and others, most investigators have concluded that the environment of various ICU's contributes to sleep and sensory deprivation (Wilson, 1987). In addition, the environmental patterns that entrain endogenous rhythms are either

absent or altered which produces an environmental time shift (Felver, 1989). The implication of this is found in research which has looked at the impact on biological rhythms when a person who is entrained to the patterns of one environment enters another environment that has different time patterns.

Mills, Minors, and Waterhouse (1978) looked at the effects of altered sleep schedules in adults in an isolation unit. Their results demonstrated the presence of free-running rhythms when the individual was subjected to constantly changing sleep times. A similar study also noted that the period of the individual's temperature rhythms increased beyond twenty-four hours (Minors, Nicholson, Spencer, Stone, & Waterhouse, 1986). The effect of interrupted sleep-wake cycles has also been noted in studies that focus on travel across time zones. An overview of this research indicated that when people are confined to an environment with different time patterns, their biological rhythms begin to resynchronize to the new environment (Redfern, Minors, & Waterhouse, 1994). But since different biological rhythms re-entrain at different rates, internal desynchronization occurs and the person may experience the symptoms of "jet lag" which include: fatigue, gastrointestinal disturbance, malaise, decreased mental alertness, and more frequent occurrence of infection (Winget, Deroshia, Markeley et al., 1984).

Overt Temporal Patterns in the ICU

Illness in general is often associated with abnormal rhythms (Lanuza et al., 1989). As noted in the previous section, when an individual is in an ICU, this factor is added to the fact that they are in an environment which lacks normal rhythmic cues and causes disruption in their sleep-wake cycle (Campbell et al., 1985).

The overt temporal pattern of a physiological variable is the net result of the endogenous rhythm that is entrained by the environment and influenced by transient environmental stimuli (Weinert et al., 1994). For patients in the ICU, Felver (1995) expanded this definition to include the following three factors: (1) possible alterations in the

expression of biologic rhythms because of the patient's pathology or pathophysiology, (2) possible partial re-entrainment by patterns in the critical care environment, and (3) changes in physiologic status because of progression or resolution of pathophysiology.

Regular sleep-wake schedules help to synchronize biological rhythms. In an environment that lacks standard day/night patterns, four hours of sleep during the customary sleeping time can keep the biological rhythms synchronized (Minors & Waterhouse, 1983). The ease with which a biological rhythm adapts to a new environment depends in part on the stability of the rhythm. The body temperature rhythm is an example of a stable rhythm that adjusts more slowly to environmental changes. The following tables provide an overview of the research done on body temperature rhythms of adult patients in an ICU. Results from studies of surgical intensive care patients cannot be generalized to medical intensive care patients since surgery and anesthesia have been demonstrated to alter body temperature rhythms in patients (Farr, L., Gaspar, T., & Munn, D., 1984). The studies are therefore divided into two groups: studies of surgical intensive care patients (Table 1), and medical and cardiac patients who have not had surgery or general anesthesia (Table 2).

Table 1. Summary of Research on Body Temperature Rhythms in Adult Patients in an ICU: Medical Patients

Author, Year	Subjects	Temp. Msrmt Route	Frequency & Duration of Msrmt	Period	Peak
Gervais et al., 1973	N=20, Toxic Comas	Rectal	q4 h x 24h	>24 hours	1732
Bell et al., 1984	N=5, Unspecified Diagnosis	Axillary	q 1h Duration: NR	>24 hours	NR
Goswami et al., 1985	N=8 Spinal Cord Trauma	Rectal	q 1h x 24h	>24 hours	2000
Okawa et al., 1986	N=12 Brain Injury	Rectal	Continuous x 24 h	24 hours	1500
Rigaud et al., 1988	N=12 Respiratory Failure (MV)	Axillary	q 2h x 24h	>24 hours	NR
Tweedie et al., 1989	N=15 Unspecified Diagnosis	Rectal	q 1h x 8-26 days	24 hours	varied
Lanuza et al., 1989	N=10 Acute Head Injury	Rectal	q 2h x 2.5-9 days	4 / 10 exhibited rhythms of ~12, 16, and 24 hours	Varied, not at expected time of healthy day-time individual
Felver, 1990	N=6 Respiratory failure due to pneumonia; N=5, and COPD; N=1	Oral	q 1h x 24h	2/6 had 24 hour rhythms; 1 subject had 6 hour rhythm	NR

Felver, 1991	N=8 Respiratory Failure	Oral	q 1h x 24h	<24 hours	NR; but reported close timing of temperatu re and heart rate rhythm peaks.
--------------	-------------------------------	------	------------	--------------	--

Note: NR = not reported; MV = mechanical ventilation

Table 2. Summary of Research on Body Temperature Rhythms in Adult Patients in an ICU: Surgical Patients

Author, Year	Subjects	Temp. Msrmt Route	Frequency & Duration of Msmt	Period	Peak
Leach et al., 1983	N=1 Comatose after coronary bypass surgery	Rectal	Frequency: NR Duration: x 10 days	24 hours	NR
Carli & Aber, 1987	N=14 Major elective abdominal, cardiac, orthopedic, or pelvic surgery	TM	q 2-4 hr x 48 h	>24	NR
Hoeksel, R., 1991	N=6 Coronary valve surgery (n=1); Coronary artery bypass (n=4); Both (n=1)	PA	q 15min x 24-48 hours	Significant rhythms noted at: 4h (1/6) 6 h (2/6) 12 h (6/6) 24 h (6/6)	Varied from healthy subjects
Woods, S., 1991	N=6 Same patients as Hoeksel study	PA	q 15 min x 24 -48 hours	24 hours + periods <24 hours	NR; noted that the peak times in 24h temp and HR rhythms were not similar

Note: NR = not recorded; TM = tympanic membrane; PA = pulmonary artery.

Results across the above studies vary in whether or not body temperature rhythms were demonstrated. All but four of the medical studies (Okawa et al., 1986; Tweedie et al., 1989; Lanoza et al., 1989; & Felver, 1990) reported a change and / or loss of the body temperature rhythm. These results may therefore indicate that either the patient's illness and / or the effects of the ICU environment (including medical treatment) have disrupted their normal body temperature rhythm. Discrepancies across the studies may be due to the differences in chosen measurement sites. For those which recorded axillary temperatures, it is possible that axillary measurements did not provide an accurate marker of the core body temperature and therefore interfered with the identification of body temperature rhythms (Hoeksel, 1991). In addition, the type and acuity of patient varied. Another factor which may have obscured the results was too infrequent sampling of body temperature. According to Minors & Waterhouse (1986), a sampling frequency of 48 hours is necessary in order to detect rhythms of 24 hour period. In addition, most of the studies listed above, did not describe simultaneous environmental and other physiological variables over time and so exogenous influences that affected the body temperature rhythm were not identified. In summary, the body temperature rhythm of patients in an ICU is not well-characterized (Felver, 1989).

Of primary significance to nursing, is the work by Farr et al., (1986), and the work by Farr, Campbell-Grossman, & Mack, (1988) (these studies were discussed earlier and were not included in the above table since the subjects in the first study were not in the intensive care unit; and the second study was done on a rat model). In these studies on postoperative patients, they found that in most patients, circadian patterns of body temperature, blood pressure, heart rate, and urinary excretion of catecholamine metabolites were altered after the operation. In addition, and more importantly, the patients with altered rhythms had a delayed recovery from their surgery.

Severity of Illness and Outcomes in the Pediatric Intensive Care Unit

The increasing complexity of illness and the increasing cost of healthcare has resulted in a need for methods to quantify patient status, define prognosis, and evaluate treatment.

Severity of illness measures emphasize the course of the disease in order to assess prognosis. Specific measurements are based on the hypothesis that physiologic instability directly reflects mortality risk (Pollack, M., Ruttimann, U., & Getson, P., 1988). The Pediatric Risk of Mortality Score (PRISM) is an index which objectively looks at physiological variables (e.g., heart rate and blood pressure, and laboratory values such as arterial blood gases and electrolytes) and their contribution to mortality risk. The PRISM score provides a quantitative and unbiased assessment of severity of illness which has several applications in a PICU. In addition to identifying patients at risk for mortality, severity of illness scores are useful in clinical research, clinical evaluation of new therapies, quality of care assessments, and utilization review (Goldstein, Fugate, & Todres, 1996).

In addition to identifying mortality, health outcomes analysis has evolved from the need to classify patients based on resulting morbidity and mortality. These classifications would then be useful in clinical research and epidemiological studies, and for assessments of the continuous quality improvement processes (Fiser, 1996). Health outcomes can be classified in several ways. These include classifications based on quality of life, degree of impairment (morbidity), and mortality. A comprehensive method would consider all three classifications. In children, health outcome may be defined as the continued ability to participate fully in developmentally appropriate activities physically, psychologically, and socially (Fiser, 1992). Health outcomes can be looked at in relation to status at hospital discharge, or long-term status as a means of quantifying the result of rehabilitation. The Pediatric Cerebral Performance Category Scale (PCPC) and Pediatric Overall Performance Category scale (POPC) are scales developed to describe the short-term outcome of pediatric

intensive care by quantifying overall functional morbidity and cognitive impairment (Fiser, 1992). They incorporate morbidity and mortality by quantifying the degree of impairment or death as a result of overall systemic insult or neurologic injury.

Research Question

Variations in the circadian rhythm of temperature have been documented in adults in the ICU. The implications of these variations have also been noted and include delayed recovery in postoperative patients (Farr et al., 1988), increased susceptibility to infection, increased length of stay, and increased patient and family stress.

The review of the literature, however, produced few studies on variations in the circadian rhythms of children. There were no studies on children in an intensive care unit. Therefore, the purpose of this study, is to provide a beginning to this area by documenting the temperature rhythms of children in an intensive care unit.

The specific aim is to answer the following question. What are the patterns of body temperature over forty-eight hours in select patients in a Pediatric Intensive Care Unit? A secondary aim was to look at any identified patterns in relation to the subject's severity of illness as measured by their Pediatric Risk of Mortality (PRISM) score, and in relation to their overall functional performance (Pediatric Overall Performance Category) and cerebral functional performance at discharge (Pediatric Cerebral Performance Category).

CHAPTER III

METHODS

This chapter will review the design of the study, the sample and setting, the instruments, and the data collection procedures. A brief description of the analysis will conclude this chapter. This design is adapted from the research design used in temporal pattern studies in an adult ICU by Felver (1990, 1991).

Design

A descriptive (time-series) design over time was used. This design allowed characterization of patterns over time in addition to analysis of specific fluctuations of a variable over time (Woods & Catanzaro, 1988). All consecutively admitted consenting patients who met the study criteria during the months of data collection were entered into the study until an N of eight had been reached. These subjects were followed continuously for forty-eight hours beginning on day one, two, or three after admission to the PICU. An effort was made to start everyone on the same day, however, the range of days occurred due to variations in the amount of time needed by the medical and nursing staff for standard PICU care. Data collection did occur, however, within the first five days of admission to the unit so that the data did not reflect possible re-entrainment of body temperature rhythm to the ICU time schedule, or conversion to a free-running rhythm (Felver, 1995). The interval of forty-eight hours was selected in order to ensure capture of two complete cycles of a twenty-four hour body temperature rhythm.

Sample and Setting

The PICU is a ten-bed medical and surgical unit of a tertiary care hospital. It is one of only two PICUs in the state of Oregon, and is one of the two referral centers for pediatric trauma. Six of the rooms are private rooms, the other two can accommodate two patients although the staff makes every effort to keep these private as well. All rooms have windows to the outside with blinds which can be opened or drawn to alter the amount of

daylight that filters into the room. There are curtains for the glass sliding door that leads into the room, which can be opened or closed as well. The PICU has an average of forty admissions per month.

A convenience sample of 8 subjects that met criteria was selected from all of the admissions to the Pediatric Intensive Care Unit (PICU) during the months of data collection. Data collection proceeded over three months. Consent for participation was obtained from the parent or legal guardian. The child was also asked if he / she wanted to participate. Subjects included male and female children of any ethnicity between the ages of birth to 18 years.

Subject number was not determined from a power analysis as this would not be appropriate for this type of study. In rhythm studies, the size of the group is not a factor since each individual is analyzed independently. Instead, the sampling frequency is the primary concern since it must be adequate (i.e., there must be an adequate number of data points) in order to complete the analysis of the data and detect a period of four to forty-eight hour length. It was necessary to be able to detect this wide range of periods since we did not know what the body temperature patterns would be in the pediatric patient in the PICU. To facilitate our ability to identify periods, temperature measurements were recorded every 5 minutes.

Exclusion criteria included:

1. Patients greater than 18-years-old since they would not be considered within the pediatric population;
2. Patients who were not predicted to remain in the ICU for at least 48 hours since this would not provide the minimum time frame needed for data collection;
3. Patients transferred to the PICU from the ward or another ICU if their total hospital stay had already exceeded five days due to the possibility that their body temperature rhythms would have been re-entrained to the environment after 5 days;

4. Patients diagnosed with acute spinal injury with cord transection or quadriplegia, malignant hyperthermia, cold-water drowning, hypo-or hyperthyroidism, or Down syndrome due to the effects these conditions have on the thermoregulatory system;
5. Patients admitted solely for a procedure (including EEG monitoring);
6. Patients whose medical condition did not indicate the need for placement of a foley catheter since this would not allow measurement of bladder temperature.

Instruments

Bladder Temperature Measurement

Bladder temperature was measured every five minutes with a Mon-a-therm Foley Catheter with temperature sensor in patients who had an indwelling catheter in place or whose condition warranted placement of the catheter. The measurements were displayed on the patient's bedside monitor. These thermometers had an accuracy of $\pm 0.2^{\circ}$ C over the range of 1.0° C - 50.0° C. Bladder temperature measurements have been highly correlated with pulmonary artery temperatures which are considered the gold standard of core body temperature measurement (Sessler, 1994). Lilly, Boland, & Zekan (1980) reported strong correlations ($r = 0.96$) between bladder and pulmonary artery temperature on 31 subjects. In addition, Hoeksel (1991) also found strong correlations ($r = 0.70$ to 0.97) in postoperative open-heart surgery patients.

Data Collection Forms

Age, gender, a brief medical history, current diagnosis, medications, and operative procedures were noted on all subjects from their medical record. In addition, the patient's baseline POPC and PCPC scores, and customary prehospital sleep and wake schedule (including naps) was obtained from the parents or the primary caregiver. To facilitate data collection and analysis, all data was entered directly into standardized data collection worksheets on a Macintosh Powerbook 5300cs.

As noted above, continuous temperature readings were displayed on the bedside monitor. Printouts of this display was obtained from the monitor every 4 - 8 hours for 48 hours (less if the bladder catheter was removed early) with the temperature readings displayed in five minute intervals. In order to identify differences and similarities between subjects during the analysis section, environmental and in-room events were also recorded on separate flowsheets by the bedside nurse. Environmental data included the patient's room number, and time cues such as the amount of light in room (i.e. curtains open -- natural lighting, curtains closed--lights on, or curtains closed and lights off). Event data included noting if the patient was awake and active; awake and quiet; sleeping; sedated; sedated with muscle relaxants, the presence of visitors in the room, and noting if the TV or radio was on. The use of warming or cooling blankets was also noted.

Data Collectors

The data collectors consisted of the researcher and the Nursing staff of the Pediatric Intensive Care Unit. Guidelines were adhered to by all collectors. The bladder catheters were inserted by the nursing staff according to the protocol described in the PICU's Policy and Procedure Manual.

Procedures

Permission to complete the study was obtained from the medical director and the nursing director of the unit prior to initiation of the study. Inservices for the nursing staff, and managers of the PICU was conducted before data collection began in order to provide an overview of the study and data collection procedures.

The PICU admission log was screened daily for potential subjects. The patient's nurses were consulted and charts reviewed to determine if eligibility criteria had been met. Then the patients and their parents or legal guardians were approached. The study purpose and procedures were explained and questions were answered. Verbal consent was obtained from the child (when appropriate) and written consent was obtained from the

parents or legal guardians. After consent was obtained, the bladder catheter was placed or the existing catheter changed to the Mon-A-Therm catheter if needed. The bedside monitor was set to include the temperature data and the table display was set to display the readings in 5-minute intervals. Then flowsheets were provided to the bedside nurse for recording hourly environmental and event data. The actual start time of data collection was coordinated with the nursing and medical staff in order not to interfere with standard patient care.

The temperature data were collected for all subjects by the investigator, her advisor, and the nursing staff during their shifts following the written protocol for obtaining the data from the bedside monitors. Printed temperature data was placed in the patient's research folder at the bedside. All gaps in temperature and environmental data collection were noted by the investigator.

At this point, most of the background information was obtained from the chart, and if the parents were available, a short interview with the parents or guardians was conducted. Specifically, the parents or guardians were asked about the subjects' normal level of functioning prior to the current illness or injury in order to establish the subject's baseline POPC and PCPC scores. The parents were then asked the following questions in order to estimate as accurately as possible the subject's customary sleep-wake schedule: 1. What time does your child regularly awaken in the morning?; 2. Does your child nap and if so, at what time and for how long?; and 3. What time does your child regularly go to sleep at night? This information was then used to calculate time of midsleep (time of midsleep = bedtime + $1/2$ (total hours of sleep)).

Last, the subject's chart and nurse were consulted in order to identify the subject's POPC and PCPC scores at discharge. The subject's outcome at PICU discharge was determined by noting if there was a change in discharge POPC and / or PCPC scores from their baseline values.

Protection of Human Subjects

Approval for this study was obtained from the Oregon Health Sciences University Committee on Human Subjects. Approval was also obtained from the medical and nursing directors. Written, informed consent was obtained from each subject's parents or legal guardians.

Temperature measurements are normally recorded every two to four hours as part of the standards of care for all patients admitted to the PICU. Bladder temperature measurement was only obtained if the patient's medical condition warranted the placement of an indwelling catheter. Data collection ended if the bladder catheter was removed before the end of the 48 hour time period.

Individual subjects were assigned subject numbers in order to preserve confidentiality. Study data was stored in locked files on a Macintosh PowerBook 5300cs that was designated only for use in research studies in the PICU.

There was no predictable risk to the patient with participation. There may or may not have been any personal benefit from participating either. However, by serving as a subject, the participant may have contributed to new information which may benefit patients in the future.

All patients were free to withdraw from the study at anytime.

Analysis of Data

The demographic data was managed using a Macintosh Powerbook with Cricket Graph graphing and spreadsheet software. The demographic data was analyzed using descriptive statistics with Systat 5.2 statistical software.

Fast Fourier Transform and Periodogram Analysis

A Fast Fourier Transform (FFT) and Periodogram Analysis was completed on each subject using Systat statistical software in order to determine the major periodic components in the graphed data. FFT analysis is a way to describe the curve

mathematically by a series of sine and cosine waves. In other words, the curve that comprised the graphed data was divided into its sine and cosine counterparts. The magnitude is equal to one-half of the amplitude of the curve at that frequency. The frequency is the inverse of the period. So, a large magnitude would indicate that the corresponding wave with the identified period length was a major component of the total curve. If the reported magnitudes are small, then no individual wave exerted more of an influence on the shape of the curve than any of the other waves that together comprise the curve. The periodogram is a graph of the magnitude squared and provides a visual display of the individual wave components.

Graphical Analysis

Next, the temperature data was analyzed separately for each subject by the following methods. First, data were graphed over time in order to identify visible period lengths. Second, environmental data that was thought to affect body temperature was noted on the graphs. This included the following documented procedures: 1. Arterial line or central line placement; 2. Endotracheal tube suctioning; 3. Duration of ventilation and time of extubation; and 4. Use of wet washcloths to facilitate cooling febrile patient. Next, the subject's reported customary prehospital sleep and wake schedule was noted along the X-axis using vertical lines labeled B, M, and A for bedtime, midsleep, and awake, respectively. Documented medications were also noted on the graphs. This included all blood products and continuous drip medications. Acetaminophen was noted near the X-axis with the approximate duration of action noted by vertical and horizontal lines.

Last, the graphs were inspected for the presence of remnants of the subject's customary prehospital sleep and wake patterns. This was done by noting if the graphed data exhibited the following trends. The first three questions looked at body temperature patterns associated with sleep. First, was there a decrease in temperature noted around the subject's reported prehospital bedtime? 2. Did the subject's temperature reach a trough

around the calculated midsleep? 3. Did the subject's temperature remain decreased during the customary sleep time? The remaining four questions looked at body temperature patterns associated with awake periods. 4. Was there an increase in temperature noted around the reported customary awake time? 5. Did the subject's temperature remain increased during the customary awake time? 6. Did the subject's temperature reach a peak in the second half of the customary awake time? 7. Did the peak occur within the range of 11-15 hours after midsleep? Positive answers in 2 of the first 3 questions indicated that the subject retained remnants of their customary body temperature pattern during their reported sleep period. Positive answers in 3 of the last 4 questions indicated that the subject retained remnants of their customary body temperature pattern during their reported awake period.

Subjects were then placed into groupings based on their diagnosis, PRISM score, and Outcome scores. The major diagnostic groups were: elective postoperative, trauma postoperative, nonoperative trauma, meningococemia, and hepatic failure. The PRISM group was divided into: Low PRISM (1-9), Moderate PRISM (10-15), and High PRISM (≥ 15). Outcome groups were: Good Outcome, Poor Neurological Outcome, and Poor Overall Function. Good outcome was defined as no change in discharge POPC or PCPC scores from baseline, or a change from a baseline of normal / healthy to mild disability in either POPC or PCPC. Poor Neurological Outcome was defined as a change from normal / healthy or mild disability at baseline to moderate disability or higher in PCPC at discharge. Poor Overall Function was defined as a change from normal / healthy or mild disability at baseline to moderate disability or higher in POPC at discharge. The presence of or deviations in each subject's prehospital body temperature patterns were then noted within each group. Last, the total number of subjects within each group who maintained remnants of their prehospital body temperature patterns during their reported customary sleep and awake times was determined in order to identify similarities and differences across groups.

Frequency tables and Pearson's Chi Square was completed on each group to identify the significance of associations noted between presence or absence of prehospital body temperature patterns and PRISM and Outcome Scores. The Pearson's Chi Square is a nonparametric test on a comparison table of 2 variables x 3 variables. It was used with this sample because of the small sample size. A Power Analysis was performed to determine sample size needed for definitive results.

CHAPTER IV

RESULTS

Sample

Eight patients (3F / 5M) admitted to the pediatric intensive care unit (PICU) between February, 1997 and April, 1997 were studied. The age range was from 3 to 17 years (mean \pm SD = 8 ± 4.70 years). Diagnoses included elective postoperative patients (N=2), trauma-postoperative (N=2), nonoperative trauma (N=1), meningococemia (N=2), and encephalopathy / hepatic failure (N=1). There were seven survivors and one nonsurvivor. Seven of the 8 subjects were studied within 24 hours of admission to the PICU. One patient was studied on PICU Day three. All postoperative patients were studied within 3 hours of the end of their operation. All subjects were on a mechanical ventilator for at least part of the time during data collection. Table 3 summarizes the characteristics of each patient.

Subject 1 was an 8-year-old female admitted following lumbar kyphectomy for severe kyphosis. Her medical diagnoses included scoliosis, spina bifida and developmental delay. Prior to this hospitalization, her health was limited by the spina bifida and kyphosis. She required orthotics for ambulation, and she was in a special education classroom at school. Her prehospital sleep schedule included customary bedtime at 2030 hours, and customary being awakened at 0700 hours. She did not normally take naps during the day. The 8.5 hour surgery was performed on the day of admission. She was admitted to the PICU directly from the operating room at 1620 hours; data collection began at 1855 hours --2 hours and 50 minutes after the end of the operation. Admission PRISM score was 6. Her baseline (i.e. prior to hospital admission) Pediatric Overall Performance Category (POPC) and Pediatric Cerebral Performance Category (PCPC) were 4 and 2, respectively. She was ventilated and sedated for the first 14 hours of admission. Her temperature readings indicated that she was febrile (temperature range: 37.8 - 38.8⁰C),

Table 3. Summary of Subject Characteristics, Diagnoses, PRISM scores, and Outcomes.

SUBJECT	AGE	GENDER	DIAGNOSIS	POSTOP	PRISM	Δ POPC	Δ PCPC
Sub 1: EP	8	F	SCOLIOSIS	Y	6	0	0
Sub 3: EP	17	M	SCOLIOSIS	Y	15	0	0
Sub 5: TP	13	M	TRAUMA	Y	5	1	0
Sub 6: TP	3	M	TRAUMA	Y	6	2	0
Sub 8: T	4	M	TRAUMA	N	10	1	1
Sub 2: S	6	M	MENINGOCOCCEMIA	N	45	5	5
Sub 7: S	6	F	MENINGOCOCCEMIA	N	33	0	0
Sub 4: H	8	F	HEPATIC FAILURE	N	14	1	1

Note. EP = Elective Postoperative; TP = Trauma Postoperative; T = Trauma, not Postoperative; S = Septic; and H = Hepatic Failure.

Y under the Postoperative column indicated the subject was admitted after surgery.

PRISM: Admission Pediatric Risk of Mortality Score

Delta POPC and PCPC: Computed difference in discharge Pediatric Overall Performance

Category or Pediatric Cerebral Performance Category from baseline scores.

however, antipyretics were not administered. She was discharged to the ward the following day (26 hours after PICU admission) in good condition and without a change in her POPC and PCPC scores. A total of 22.9 hours of data was collected.

Subject 2 was a 6-year-old male admitted for sepsis and septic shock due to meningococemia. He had an unremarkable past medical history prior to this hospitalization. His prehospital sleep schedule included a customary bedtime at 2030 hours and awakening at 0700 hours. He did not nap during the day. His POPC and PCPC scores were both 1. The day before hospital admission, he had complained of a sore throat and headache, had come home early from school, and had gone to sleep around 1930 hours. He was taken to an Emergency Department at an outside hospital around 0130 hours the following morning after his mother noted him to have decreased responsiveness and a spreading rash. When he first arrived at the referring hospital, he was alert and able to follow commands but was lethargic and disoriented to time and place. His blood pressure was low, and he was in renal failure. He was transported to the PICU at the study site at 0930 hours after his condition continued to deteriorate. Prior to transport, he was paralyzed and sedated and placed on a mechanical ventilator for impending respiratory failure. He arrived at the PICU at 1055 hours that day, and data collection began at 1150 hours. His admission PRISM score was 45. His temperature on admission was 39.3⁰C. Acetaminophen and antibiotics were started along with dopamine and dobutamine, epinephrine, and methylprednisolone. At approximately 1230 hours, he experienced two generalized tonic clonic seizures. Immediately following his pupils became unequal, the left was dilated and he was no longer responsive to stimuli. He died the next day, approximately 28 hours after admission. A total of 26.9 hours of data was collected.

Subject 3 was the oldest subject in the sample. He was a 17-year-old male with a past medical history of muscular dystrophy that was diagnosed at age 13 months with progressing scoliosis since age 9 years. His customary prehospital sleep / wake schedule

included going to sleep around 2130 hours and waking up at 0600 hours. He was admitted to the hospital the morning of surgery for spinal fusion with rod placement to correct the scoliosis. The surgery took 13.5 hours. He was admitted to the PICU at 2245 hours directly from the Operating Room. Data collection began at 2442 hours-- 2 hours and 45 minutes after the operation. His baseline POPC score was 3 (he has been wheelchair dependent since age 4 years), and his baseline PCPC score was 1. His admission PRISM score was 15. His temperature ranged from 36.9⁰C to 38.9⁰C. Medications received included acetaminophen, cefazolin, lorazepam, and ranitidine. He had two chest tubes placed during the operation and he was kept on a ventilator until PICU Day 8. His PICU stay was prolonged due to respiratory distress and difficulty with extubation. He was discharged to the ward 8 days after PICU admission with no change in his POPC or PCPC scores. A total of 59 hours of data was collected.

Subject 4 was an 8-year-old female with an extensive and complicated medical history which included: arthroglyposis, seizure disorder, paraesophageal hernia, status-post bowel perforation in February, and mental retardation. She has had numerous hospitalizations in the past, the last one in February, 1997. When she is not in the hospital, her customary bedtime is 2030 hours, she wakes up around 0500 hours, and she takes about a three hour nap between 0800 hours and 1100 hours. She was admitted from home directly to the PICU at 2215 hours for hepatitis and fulminant hepatic failure due to accidental acetaminophen overdose and seizure. Data collection began at 0855 hours the following morning. Her baseline POPC and PCPC scores were both 4 due to her pre-existing conditions. Her admission PRISM score was 14. Medications administered during her hospital stay included epinephrine, dopamine, dobutamine, acetylcysteine, antibiotics, lorazepam, phenobarbital, phenytoin, neurontin, and acetaminophen. After several days, her liver function started to improve. However, she was no longer responsive to stimuli. Her PICU stay was further complicated by a need for tracheostomy

due to failure to extubate from mechanical ventilation. She remained in the PICU for a total of 29 days and was transferred to the ward in a persistent vegetative state with a POPC and PCPC score of 5. A total of 57 hours of data was collected.

Subject 5 was a previously healthy 13-year-old male admitted postoperatively following repair of extensive facial lacerations and skull fractures received in a wood shop accident. His customary sleep schedule included going to sleep at 2300 hours and waking up at 0630 hours. He did not nap during the day. The repair of the lacerations took 3 hours and 35 minutes. He was admitted directly to the PICU from the operating room at 2300 hours. Data collection began at 2442 hours. His admission PRISM score was 5, and baseline POPC and PCPC scores were both 1. He received acetaminophen for increasing temperature twice. Other medications included: antibiotics, phenytoin, methylprednisolone, and fentanyl. His PICU stay was uncomplicated and he was extubated 12 hours after admission. He was discharged on Day 2. His POPC and PCPC scores on discharge remained 1. Data collection stopped after 40.5 hours when the indwelling catheter was removed.

Subject 6 was a 3-year-old previously healthy male admitted for treatment of injuries sustained in an auto accident. He was an unrestrained back seat passenger in a car that was struck head on by a second car. Both vehicles were traveling around 40-50 miles per hour. His prehospital sleep schedule included going to bed at 2100 hours and getting up at 0800 hours. He does not usually nap during the day. His injuries included trauma to the spinal cord at the T6 level, and a perforated cecum that was not detected until Day 3 of his hospital stay. He was taken to the operating room for an exploratory laparotomy with a bowel resection and colostomy on Day 3. The operation lasted 2 hours 45 minutes. He returned to the PICU directly from the operating room at 1700 hours. Data collection began at 1725 hours. His baseline POPC and PCPC scores were 1 and 1. His admission PRISM score was 6. His temperature varied greatly from minimum temperature of 37.05

to a maximum temperature of 39.0⁵. He received acetaminophen three times per day on Days 1 and 2. His PICU stay was complicated by multiple abdominal abscesses that required additional surgery on PICU 10, and the persistent paralysis he experienced from the nipple line down despite lack of cord injury identifiable on repeated magnetic resonance imaging. He was extubated on PICU Day 5 and discharged to the ward 15 days after admission. Discharge POPC and PCPC scores were 3 and 1, respectively. A total of 49.9 hours of data was collected.

Subject 7 was the second subject admitted with the diagnosis of meningococemia. She was a previously healthy 8-year-old female who had complained of sore throat and headache approximately 12 hours prior to admission. Her customary prehospital bedtime was at 2100 hours and she woke up at 0600 hours. She did not customarily nap during the day. She was rushed to an Emergency Department at an outside hospital around midnight after her mother noted decreased level of consciousness and a purple rash on her trunk and extremities. On arrival to the Emergency Department, her systolic blood pressure was 48 mmHg, and she was febrile with a temperature of 40.8⁰C. She was paralyzed and sedated for intubation and mechanical ventilation. She was started on vasopressors including dopamine, dobutamine, and epinephrine to maintain her blood pressures and was immediately transferred to Oregon Health Sciences University (OHSU). She was admitted to the PICU at 0430 hours. Data collection started at 0550 hours. On arrival to the PICU her Glasgow Meningococemia Prognostic Score (GMSPS) score was 15 (range for score is 1-15, with 15 being the most severely ill). Her admission PRISM was 33, and her baseline POPC and PCPC scores were both 1. Central venous and arterial catheters were placed to monitor her hemodynamic status. She received continuous dopamine, dobutamine and epinephrine drips to sustain her blood pressure, as well as cefotaxime, furosemide, methylprednisolone, ranitidine, vecuronium, lorazepam, and fentanyl for pain. She received one dose of acetaminophen for an increase in temperature to 39.3 ⁰C on

PICU Day 2. She responded well to treatment, was extubated on PICU Day 8, and was discharged 9 days after admission. Her discharge POPC and PCPC remained at 1. A total of 53 hours of data was collected.

Subject 8 was the second trauma subject. He was a 4 year-old-male with no prior medical history. He was admitted for head injuries and fractures received after being struck by a car. He was initially treated at an outside hospital, was placed on a ventilator for decreased neurological status due to possible subdural hematoma, and was then transferred to the study facility for evaluation and management by the Division of Neurosurgery. His customary bedtime was 2000 hours, while 0700 hours was his customary awake time. He occasionally napped between 1230 and 1430 hours. He arrived at our facility at 2430 hours. Data collection started the next day at 2006 hours. His admission PRISM was 10. Baseline POPC and PCPC scores were both 1. An intracranial monitor was placed to monitor his intracranial pressure along with an arterial line to monitor hemodynamic status. He remained sedated and on the ventilator for a total of 72 hours. He was discharged to the floor on PICU Day 3 and then later transferred to a rehabilitation center secondary to decreased cognitive function and left-sided weakness. His discharge POPC and PCPC were both 2. Data collection was shortened due to removal of the indwelling catheter. A total of 43.8 hours of data was collected.

Temporal Pattern of Body Temperature

Statistical Analysis of Individual Subjects

Descriptive Statistics

Seven of the eight subjects were febrile during data collection. Mean temperature ranged from 36.9⁰ C to 39.1⁰ C (SD range 0.18⁰ C to 0.72⁰ C respectively). Table 4 summarizes the temperature ranges for each subject.

Table 4. Summary of Temperature Ranges for each Subject.

SUBJECT	MINIMUM TEMP	MAXIMUM TEMP	MEAN TEMP	SD	RANGE
	°C	°C	°C		°C
Sub 1 EP	37.5	38.8	38.4	0.18	1.3
Sub 3 EP	36.9	38.9	38.3	0.51	2.0
Sub 5 TP	36.5	37.9	37.3	0.32	1.4
Sub 6 TP	37.5	39.5	38.5	0.43	2.0
Sub 8 T	36.3	38.9	37.3	0.50	2.6
Sub 2 S	37.7	39.4	38.5	0.53	1.7
Sub 7 S	37.5	39.3	38.2	0.42	1.8
Sub 4 H	36.3	40.8	39.1	0.72	4.5

Note. EP = Elective Postoperative; TP = Trauma Postoperative; T = Trauma, not Postoperative; S = Septic; and H = Hepatic Failure.

Fast Fourier Transform and Periodogram Analysis

A Fast Fourier Transform (FFT) and Periodogram Analysis was completed on each individual subject using Systat (Wilkinson, Hill, & Vargi, 1992) statistical software for the Macintosh computer in order to determine the major periodicities in the curve. Results revealed waves with small magnitudes. The largest magnitudes noted were 1.84 and 1.77, which corresponded to periods of 10.6 and 7.1 hours, respectively. However, since the magnitudes were small, the analysis did not support the presence of any major periodic component for any subject. These results were consistent with the FFT being comprised of multiple sine and cosine waves of different frequencies which, when taken together in near equal parts, comprised the data curves.

Graphical Analysis of Individual Subjects

The amount of data obtained from each subject varied between subjects due to variations in the amount of time the patient had an indwelling catheter inserted and differences in the total length of stay in the PICU. In addition, several data points were deleted from Subject 1 and Subject 4. In Subject 1, the first hour of data revealed a sharp and steady increase in temperature. The temperature rose from 37.5⁰ C to 38.5⁰ C within 50 minutes. It was thought that this patient was at risk for induced hypothermia given the length of the surgery (8 hours and 20 minutes) and the type of surgery (spinal fusion with rod placement). If the patient had become hypothermic during surgery (the last temperature recorded in the operating room was taken 2 hours before the end of the operation and revealed a temperature of 36.5⁰ C), then the sharp and rapid elevation in temperature represented a rewarming trend after surgery which would affect the overall pattern if included in data for analysis. The data was therefore deleted. Subject 4's indwelling catheter was irrigated when his urinary output decreased. There was a notable decrease in temperature recorded at the time of irrigation and these data points were therefore deleted so as not to affect the overall pattern of the data. There were other missing data from other

subjects that was due to accidental error by the bedside nurse when collecting data from the bedside monitor. This is represented on the individual graphs by a straight line.

Bladder temperature data were graphed over time for each subject (Figures 1 through 8). The size of each graph and range of both axes were standardized to allow comparisons between subjects. The patient's customary sleep and wake schedule was added to each individual graph along with medications received and treatments completed. Lastly, the nursing records and the collected environmental data were compared to the graphs and potential associations with small fluctuations in temperature were noted. Of interest were the small fluctuations ($< 0.5^{\circ}\text{C}$) noted in 6 / 8 ventilated patients during endotracheal suctioning. Most of these fluctuations lasted for only a short duration (< 1 hour) and were noted regardless of the presence of sedating medications. There was no detectable difference in the size of the temperature fluctuation in relation to whether it occurred at the peak or trough of the curve. Similar small fluctuations were noted in 2 subjects during placement of either central venous catheters or arterial catheters.

As indicated above, seven of the eight subjects were febrile at the start of data collection. Subject 3 started with a temperature of 36.9°C but then quickly rose to 38.4°C within 6 hours. Visual inspection of all individual temperature graphs revealed that there were fluctuations in temperature for all subjects, however the degree of fluctuation varied from a range of 1°C to 4.5°C . Changes in the temperature curve's baseline or overall pattern from Day 1 to Day 2 were noted in four of the six subjects (there was not enough data for two of the subjects to make this comparison). Three subjects showed a decrease in baseline but no overall change in their pattern of temperature fluctuation. However, Subject 4 showed a small decrease in baseline but a significant change in overall pattern from Day 1 to Day 2. On Day 1, the temperature graphed indicated the temperature was increasing to a peak between the subject's customary bedtime and her customary time at

midsleep. But, by Day 2, this pattern was no longer present. Instead, there was little to no fluctuation in temperature between her customary bedtime and customary awake time.

A final visual inspection was done in order to identify any gross similarities in the pattern of temperature fluctuations. Individual body temperature graphs were superimposed on each other and sleep / wake schedules were aligned. The most striking finding from this inspection was the similarities noted between Subject 4 and Subject 8. Both subjects had marked fluctuations during the first 24 hours of data collection followed by a steady decrease in body temperature on Day 2. At each subject's customary bedtime, the body temperature reached a plateau and remained there with only small ($\leq 0.5^{\circ}\text{C}$) fluctuations until 9 hours after customary awake time.

Further analysis of individual graphs was completed using the questions outlined in the methods chapter. Results from this individual review are discussed below.

Subject 1

Data collection for this subject began around her customary bedtime. At this time, her temperature was increasing and remained elevated until ~2 hours after her customary bedtime when it started to decrease. Her temperature did reach a trough around her customary time of midsleep and remained decreased during her customary sleep time except for the 0.3°C increase that corresponded to her being turned and suctioned by her nurse. It was also noted that she had complained of pain at this time. Her temperature then started to increase around her customary awake time. However, it did not peak during the second half of her awake time (i.e. 11 - 15 hours after midsleep). It also did not remain elevated during her awake period but decreased back to the same temperature noted during her customary sleep period. Therefore, there were remnants of a normal prehospital circadian pattern during her customary sleep period but not during her customary awake time.

Subject 2

Subject 2's body temperature was greatly elevated at the start of data collection and then sharply decreased over the next 6 hours. However by the time of his customary bedtime, his temperature had again begun to increase (even though he was given acetaminophen at this time), reached a peak at midsleep, and then decreased again before his customary awake period. His temperature continued to decrease for the next 5 hours and then started another increase when data collection was halted. There were no visible remnants of a normal prehospital temperature pattern evident in his temperature graph.

Subject 3

Data collection began ~1.5 hours before his customary midsleep. At this point, his temperature was steadily increasing from 36.9⁰ C, reaching a peak of 38.4⁰ C at his customary awakening time. His temperature remained elevated during all awake periods, reaching a peak during the second half of the awake period. The peak also occurred between 11-15 hours after midsleep on all days. In addition, his temperature decreased around his customary bedtime, reached a trough around his customary midsleep period, and remained decreased during his customary sleep time. In summary, there was clear and evidence of a complete and normal prehospital sleep / wake temperature pattern present in the data.

Subject 4

This data graph was difficult to analyze due to the marked and frequent temperature fluctuations (~3.5⁰ C). However, there did not appear to be any notable decrease in temperature around her customary bedtime on Day 1 or 2, nor were there troughs around her customary midsleep. Her temperature did not remain decreased during her customary sleep time on Day 1 but did remain decreased on Day 2. Her temperature increased around her customary awake time and remained increased only on Day 1. However, it did not peak during the second half on Day 1. Instead, her temperature began a steady decrease

during this time which continued until it reached a plateau around her customary bedtime on Day 2. Therefore, there were two remnants of her prehospital temperature pattern noted during her customary awake period on Day 1, but this was not noted on Day 2. By Day 2, there was only one remnant of her prehospital temperature pattern that was noted during her customary sleep period.

Subject 5

On Day 1, there were only two remnants of Subject 5's prehospital temperature pattern during his customary sleep period and customary awake period. Missing was a constant lower temperature during his customary sleep time and a peak in the temperature during the second half of the awake time. By Day 2, however, there was complete evidence of his prehospital temperature pattern. His temperature decreased around bedtime, remained decreased during his customary sleep time, and reached a trough at customary midsleep. It then began to increase around customary awakening, remained increased during customary awake period, and peaked during the second half of his awake period. There was, therefore, increased evidence of a normal body temperature pattern from Day 1 to Day 2.

Subject 6

There was no evidence of Subject 6's prehospital temperature pattern during his customary sleep time on either Day 1 or Day 2. His temperature did decrease at his customary bedtime on both days but he was given acetaminophen within 1 hour of this time period and therefore it is not possible to say definitively that this pattern was present. In addition, his temperature did not remain decreased but instead began to increase and reached a peak at midsleep. In contrast, his temperature did increase around his customary awakening, was increased during his customary awake time, and peaked in the second half on Days 1 and 2. Thus, there was no evidence of his prehospital temperature pattern

during his customary sleep time, but there was evidence of his prehospital temperature pattern during his customary awake time.

Subject 7

Only one remnant of Subject 7's prehospital temperature pattern was noted during her customary sleep and awake periods on Day 1 and Day 2. Her temperature did decrease around her customary bedtime, but it did not reach a trough at midsleep or remain decreased during her customary sleep time on Day 1. On Day 2, her temperature reached a trough around her customary midsleep, but it did not remain decreased. Instead, her body temperature was fluctuating around her customary bedtime. Her temperature did peak during the second half of her customary awake time on Day 1, and it did increase around her customary awake time on Day 2. However, neither of these remnants were noted on both days and her body temperature did not remain increased on either day. Therefore, it appeared that there was very little evidence of her prehospital temperature pattern evident in her graph.

Subject 8

This temperature graph strongly resembled Subject 4's graph. There were marked fluctuations noted on Day 1, but only one remnant of his prehospital temperature pattern was noted during his customary awake period. There was no evidence of his prehospital temperature pattern during his customary sleep time. On Day 2 this was reversed in that there was one remnant of his prehospital sleep pattern during his customary sleep time but no evidence of his prehospital pattern during his customary awake time.

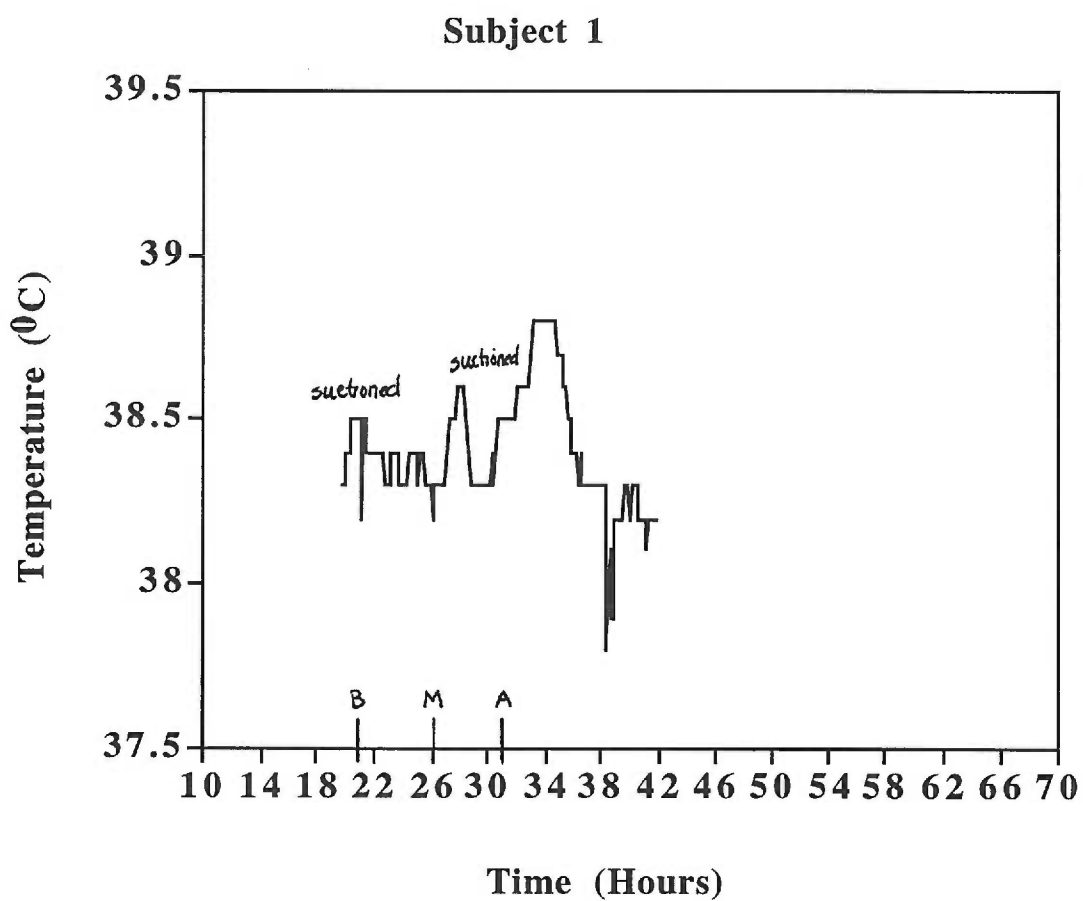


Figure 1. Temperature fluctuations over a 22 hour interval in a 8-year-old female with kyphosis and spina bifida. Admission severity of illness was low and the patient had a good outcome.

Subject 2

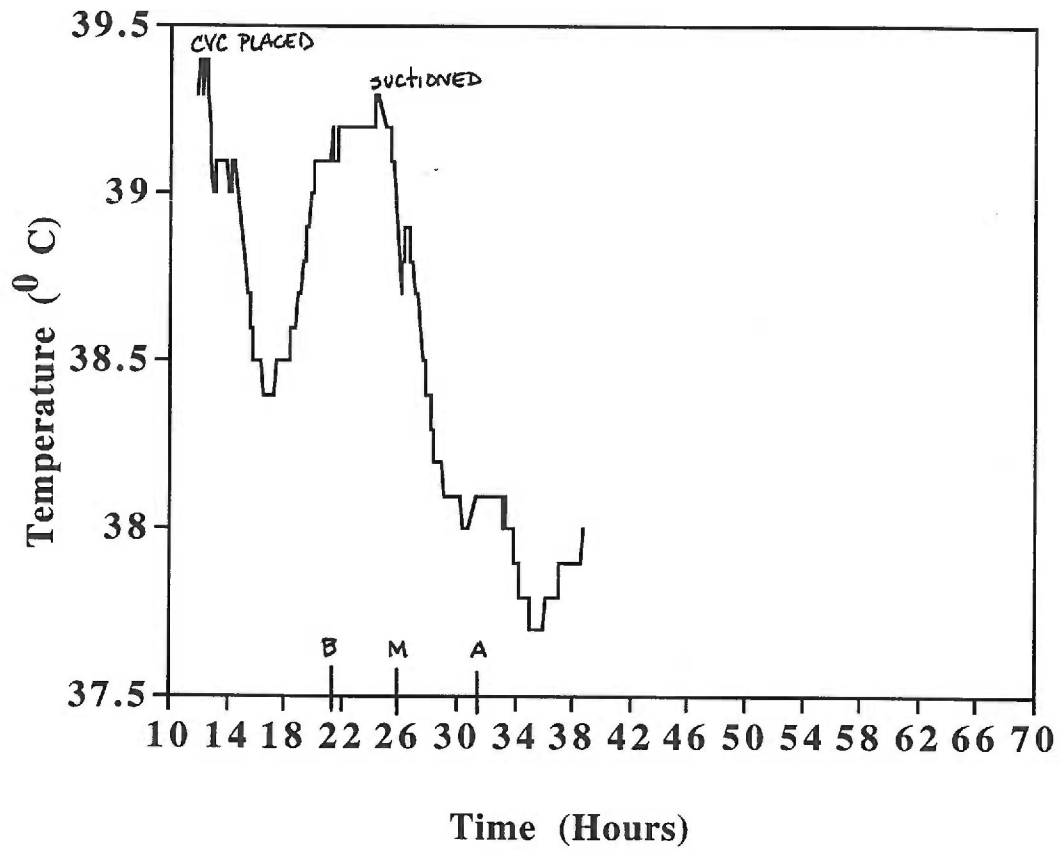


Figure 2. Temperature fluctuations over a 26 hour interval in a 6-year-old male with meningococemia. Admission severity of illness was high and the patient died.

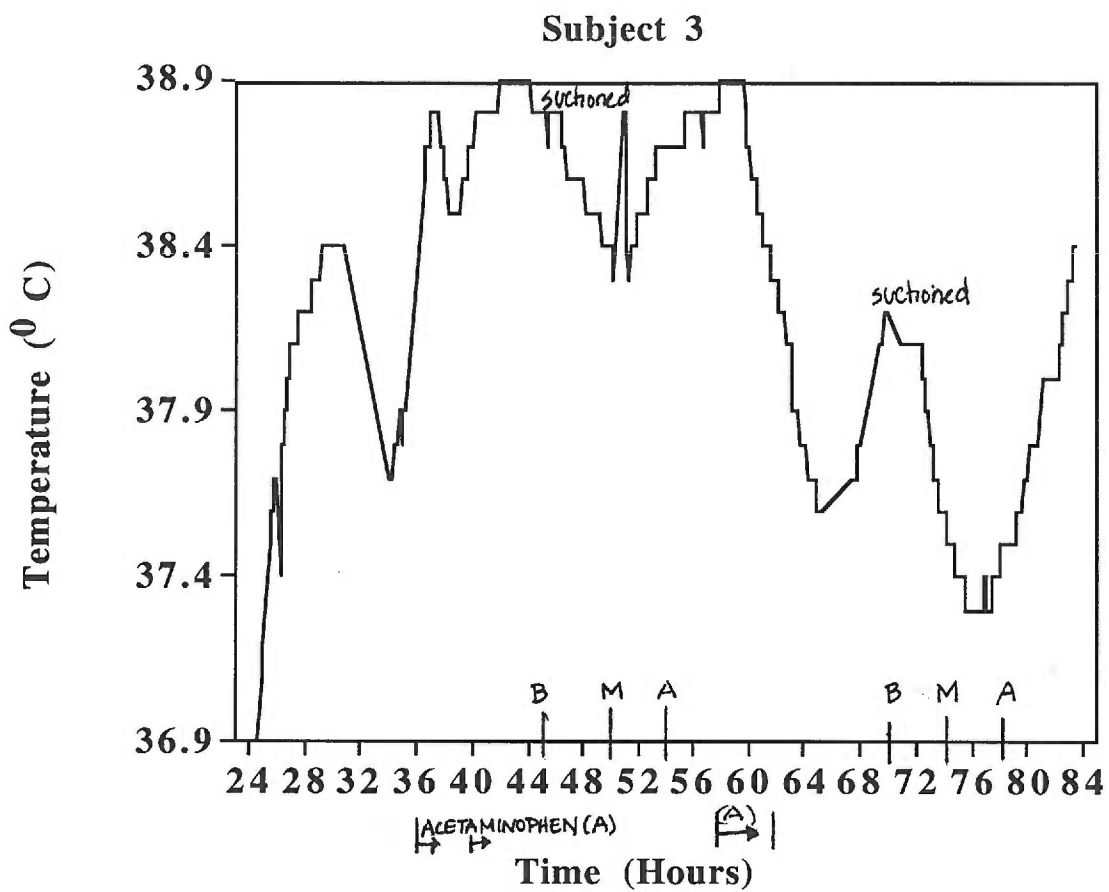


Figure 3. Temperature fluctuations over a 59 hour interval in a 17-year-old male with scoliosis. Admission severity of illness was moderate and the patient had a good outcome.

Subject 4

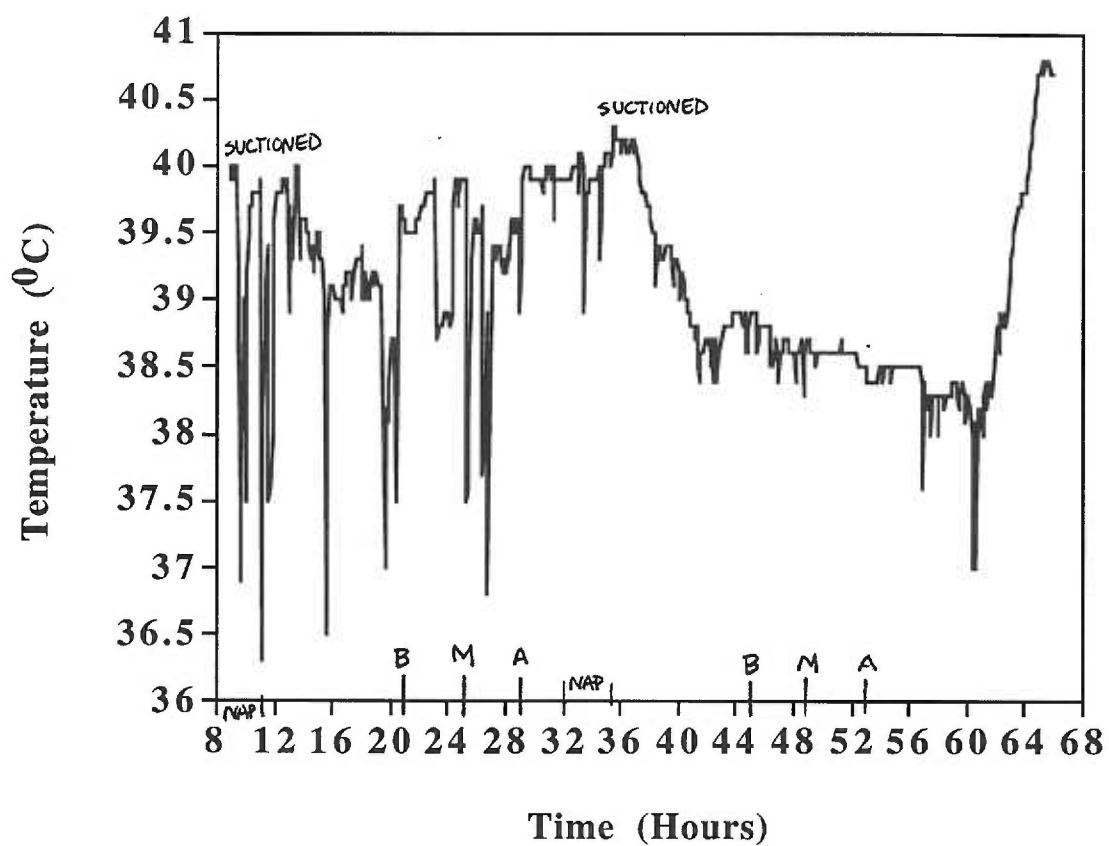


Figure 4. Temperature fluctuations over a 57 hour interval in a 8-year-old female with hepatic failure. Admission severity of illness was moderate and the patient poor outcome.

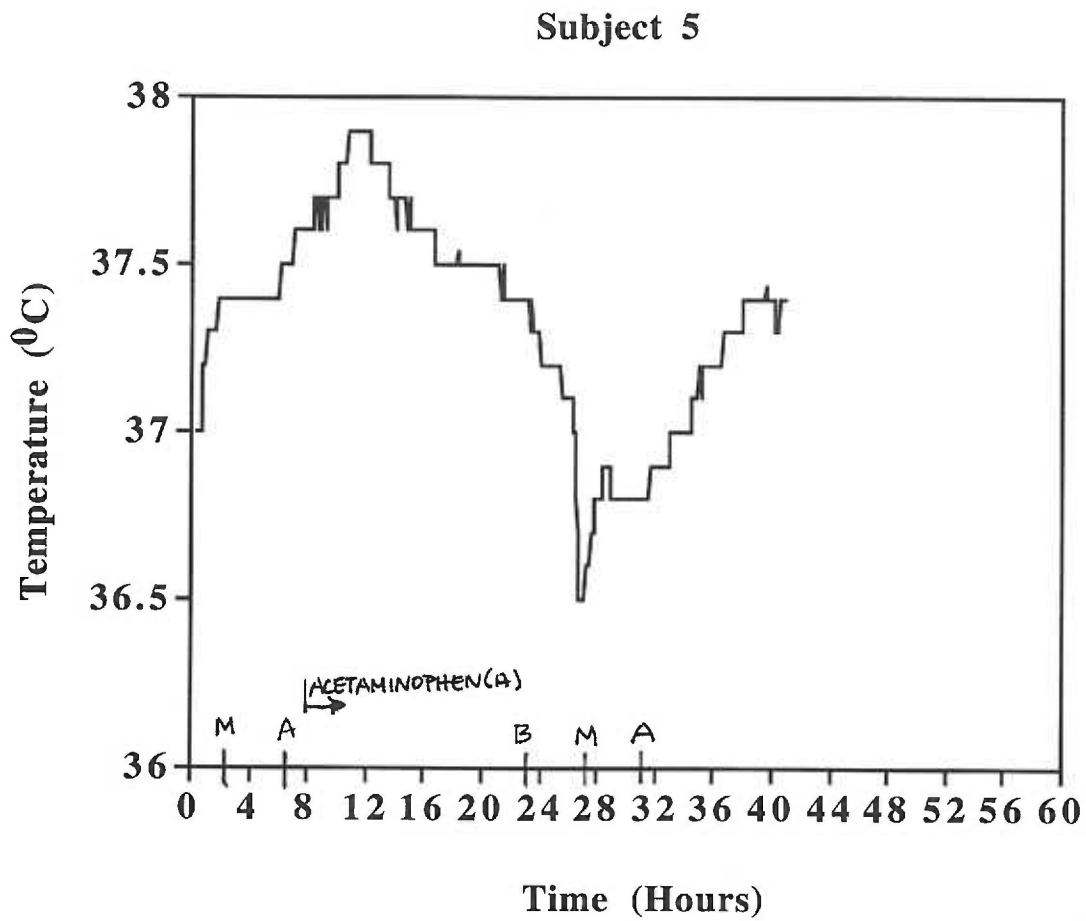


Figure 5. Temperature fluctuations over a 40 hour interval in a 13-year-old male with skull and facial fractures. Admission severity of illness was low and the patient had a good outcome.

Subject 6

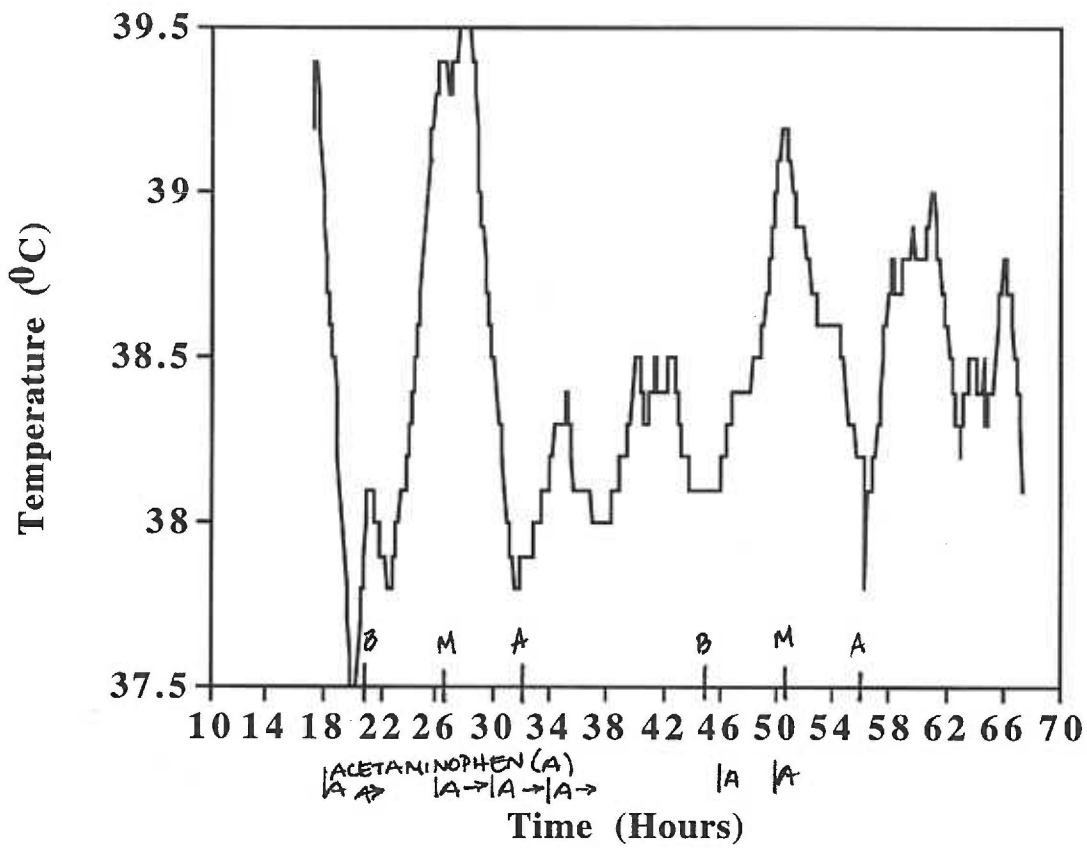


Figure 6. Temperature fluctuations over a 50 hour interval in a 3-year-old male s/p motor vehicle accident with spinal fracture and perforated cecum. Admission severity of illness was low and the patient had a poor overall functional outcome.

Subject 7

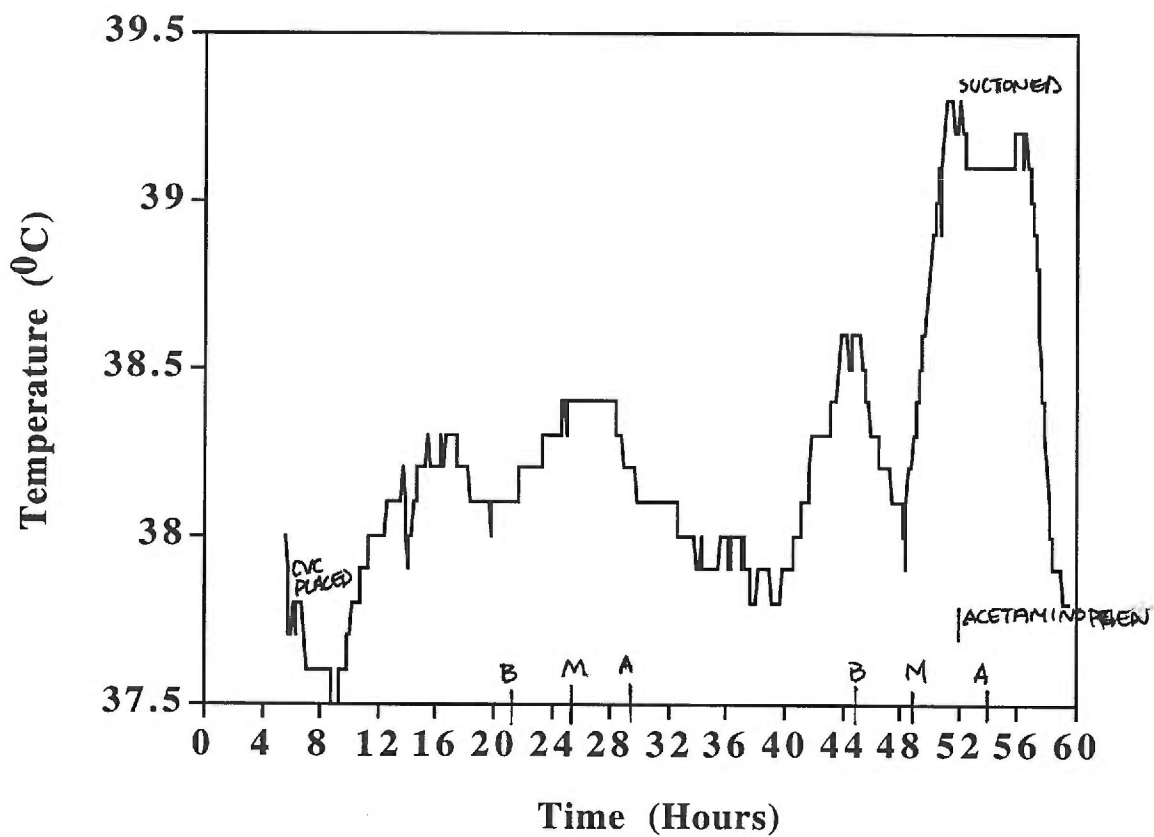


Figure 7. Temperature fluctuations over a 53 hour interval in a 8-year-old female with meningococemia. Admission severity of illness was high and the patient good outcome.

Subject 8

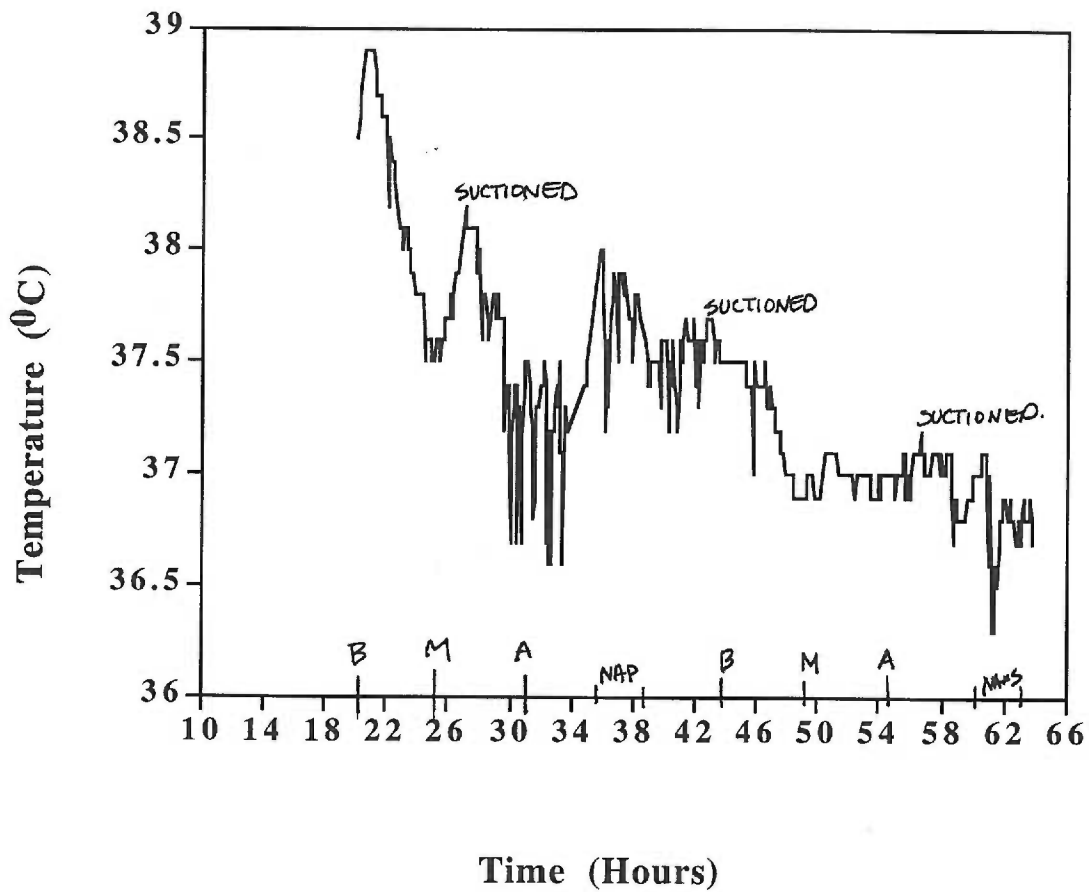


Figure 8. Temperature fluctuations over a 43 hour interval in a 4-year-old male with a head injury after being struck by a car. Admission severity of illness was moderate, and the subject had a poor neurological outcome.

Summary of Individual Analysis

In summary, of the eight patients studied, two subjects had expected patterns during both customary sleep and customary awake times. Three subjects showed evidence of their prehospital temperature pattern during their customary sleep time while five did not. Similarly, three patients showed evidence of their prehospital temperature pattern during their customary awake time. In addition, however, there were two additional patients with evidence of their prehospital temperature pattern during their customary awake on Day 1 that was not present on Day 2.

Graphical Analysis by Groups of Subjects

The eight subjects were grouped according to their diagnosis, outcome, and admission PRISM. Evidence of or deviations from their prehospital temperature patterns were noted within each group.

Graphical Analysis According to Diagnosis

The five diagnostic groups were: Elective Postoperative (N=2), Trauma Postoperative (N=2), Nonsurgical Trauma (N=1), Meningococemia (N=2), and Hepatic Failure (N=1). Within the Elective Postoperative group, both subjects demonstrated a normal temperature pattern during the customary sleep period but only 1 subject had a normal pattern during the customary awake time. Only one subject in the Trauma Postoperative group demonstrated a normal pattern during the customary sleep period while both subjects showed normal patterns during customary awake times. The Nonsurgical Trauma patient did not show normal temperature patterns during his customary sleep time and had normal patterns during his customary awake time only on Day 1. The patient with Hepatic Failure had identical findings. In contrast, neither subject in the meningococemia group had any evidence of normal temperature patterns during either the customary sleep time or awake time. Table 5 provides a summary of these findings.

TABLE 5. Patterns in Body Temperature Rhythms During Customary Sleep and Awake Times for Subject Group Divided by Diagnosis.

	Elective Postoperative	Trauma Postoperative	Trauma	Meningo-cocccemica	Hepatic Failure
Normal temp. pattern during customary sleep time	Sub 1-YES Sub 3-YES 2/2	Sub 5-YES Sub 6 - NO 1/2	Sub 8 - NO 0	Sub 2:NO Sub 7:NO 0/2	Sub 4: NO 0
Normal temp. pattern during customary awake time	Sub 1 - NO Sub 3-YES 1/2	Sub 5 - YES BUT NO PEAK Sub 6 YES BUT DAY 2 NO PEAK 2/2	Sub 8 - DAY 1;YES DAY 2;NO	Sub 2:NO Sub 7:NO 0/2	Sub 4: DAY 1,YES DAY 2,NO
Normal pattern during both customary sleep and awake time	Sub 1 - NO Sub 3 - YES 1/2	Sub 5-YES Sub 6-NO 1/2	Sub 8- NO 0/1	Sub 2- NO Sub 7 -NO 0/2	Sub 4 -NO 0/1

Graphical Analysis According to Admission PRISM

Subjects were grouped according to their Admission PRISM score. Group 1 was low severity of illness (PRISM scores 1-9, N=3); Group 2 was moderate severity of illness (PRISM scores 10 -15 (N=3); Group 3 was high severity of illness (PRISM scores ≥ 15 , (N=2). These groupings were determined based on the % mortality associated within the range of PRISM scores (see Table 6). Scores 1-9 are associated with $\leq 4.4\%$ mortality; scores of 10-15 are associated with 8.0% - 13.9% mortality; and scores > 15 are associated with a 23% mortality (Pollack et al., 1988).

In Group 1 (low severity), two of the three subjects retained their prehospital temperature pattern during their sleep and awake time. In Group 2 (moderate severity), two of the three subjects had evidence of their prehospital temperature pattern during their customary awake time. However, only one of the three subjects retained a prehospital temperature pattern during the customary sleep time. In Group 3 (high severity), none of the subjects showed any evidence of their prehospital sleep pattern during their customary sleep or awake times. In summary, results of this grouped analysis indicated that as the PRISM scores increased (i.e. increasing severity of illness), subjects lost their prehospital temperature pattern during their customary sleep time. And in the most critically ill subjects, there was a total loss of their prehospital temperature patterns during both sleep and awake times. A summary of this analysis can be found in Table 6.

TABLE 6. Patterns in Body Temperature Rhythms During Customary Sleep and Awake Times for Subject Group Divided by PRISM Scores.

	Low Prism (1-6)	Moderate (10-15)	High (>15)
Normal temperature pattern during customary sleep time	Sub 1: YES Sub 5: YES Sub 6: NO 2/3	Sub 3: YES Sub 4: NO Sub 8: NO 1/3	Sub 2: NO Sub 7: NO 0/2
Normal temperature pattern during customary awake time	Sub 1: NO Sub 5: YES Sub 6: YES 2/3	Sub 3: YES Sub 4: YES, DAY 1 NO ON DAY 2 Sub 8: YES, DAY 1 NO ON DAY 2	Sub 2: NO Sub 7: NO 0/2
Normal temperature pattern during both customary sleep and awake time	Sub 1: NO Sub 5: YES Sub 6: NO 1/3	Sub 3: YES Sub 4: NO Sub 8: NO 1/3	Sub 2: NO Sub 7: NO 0/2

Graphical Analysis According to Outcome

Outcome was measured by change in discharge POPC and PCPC scores from their baseline. A good outcome was defined if there was no change in discharge scores, or if the change was only from normal and healthy to mild disability (level 2) in either or both POPC or PCPC. Poor neurological and overall performance outcomes were defined as a change only in POPC or PCPC to level 3 or higher indicating a moderate to severe disability. The divided sample included: Good Outcome, N=4; Poor Neurological Outcome, N=3; and Poor Overall Function, N = 1.

All subjects with good outcomes demonstrated retention of prehospital temperature patterns during either their customary sleep time, or their customary awake time, or both. In contrast, the subject in the poor overall function group (POPC ≥ 3), exhibited normal prehospital temperature patterns during his customary awake time but not during his customary sleep time. Finally, of the subjects in the poor neurological outcome group (PCPC ≥ 3), one retained evidence of her prehospital temperature pattern during her customary sleep time on Day 1 but not on the Day 2. None of the three subjects retained evidence of their prehospital sleep patterns during their customary sleep time. These results indicate that presence of prehospital temperature patterns was associated with good outcome, while their absence was associated with poor outcome. Table 7 provides a summary of these results.

TABLE 7. Patterns of Body Temperature During Customary Sleep and Awake Times for Subjects Divided by Outcome.

	Good Outcome	Poor Neurological Outcome	Poor Overall Functional Outcome
Normal temperature pattern during customary sleep time	Sub 1:yes Sub 3:YES Sub 5:YES Sub 7:NO, but did decrease at bedtime on day 1, and trough at midsleep on day 2. 3/4	Sub 2:NO Sub 4:DAY 1:NO DAY 2: Only 1 remnant noted = temp. was decreased during sleep Sub 8: NO, only noted decrease around bedtime on day 1 and 2.	Sub 6:NO
Normal temperature pattern during customary awake time	Sub 1: NO Sub 3: YES Sub 5: YES, except did not peak during the second half of day 1 or 2. Sub 7: NO, but did peak on 2nd 1/2 of day 1. 2/4	Sub 2: NO Sub 4: D1:YES D2:NO Sub 8: DAY 1:YES DAY 2:NO	Sub 6: YES
Normal temperature pattern during both customary sleep and awake time	Sub 1: NO Sub 3: YES Sub 5: YES Sub 7: NO 2/4	Sub 2: NO Sub 4: NO Sub 8: NO 0/3	Sub 6: NO 0/1

Summary of Graphical Analysis of Groups

Results of the grouped analysis revealed that there were differences in the temperature patterns of subjects within groups that are characterized by their diagnosis, PRISM score, and outcome. Specifically, subjects with high PRISM scores, indicating greater severity of illness, and poor outcome tended to retain fewer components of their prehospital temperature pattern than those with lower severity of illness and good outcome.

Statistical Analysis of Groups

Frequency tables (cross tabulations) were computed in order to measure the association between the categorical variables listed in the group analysis. Pearson Chi-square was used to determine the significance of any associations. Results for the PRISM group showed a Pearson Chi-square of 2.31, $p = 0.32$. The Outcome Group had a Pearson Chi-square of 4.8, $p = 0.09$. The null hypothesis in two-way tables is that there is no association in the table. Neither group had significant p values and therefore the null hypothesis could not be rejected.

Assumptions of the Pearson Chi-square include that there need to be at least 5 subjects per cell. Power analysis indicated that to meet the assumptions of the Pearson Chi-square, the minimum sample size needed for more definitive results would be an N of 30.

Summary of Results

Eight patients (3F / 5M) admitted to the pediatric intensive care unit (PICU) between February, 1997 and April, 1997 were studied. Individual graphical analysis identified the presence of temperature patterns during customary sleep and awake times based on prehospital sleep and wake schedules in some of the patients. When looking at the group as a whole, only two of the eight subjects demonstrated prehospital temperature patterns during either their customary sleep time and their customary awake time. A finer discrimination between subjects was identified when the subjects were placed into groups

based on their severity of illness (PRISM) and outcome. This grouped analysis revealed that subjects with increased PRISM scores and poor outcomes retained fewer components of their prehospital temperature pattern than subjects with lower PRISM scores and good outcomes.

CHAPTER V

DISCUSSION

The primary aim of this study was to describe the patterns of body temperature fluctuations over forty-eight hours in selected patients in a PICU. The secondary aim was to examine identified patterns of body temperature fluctuations in relation to the PRISM score and outcome at PICU discharge. Data was analyzed for both individual subjects and the study group. Temperature patterns were compared to prehospital customary sleep and awake times. Descriptive statistics (calculation of the mean) and visual graphical analysis, fast Fourier transform and periodogram analysis, were used to analyze individual subjects, while visual graphical analysis and Pearson Chi square correlation were used to analyze the group. This chapter discusses the results of these analyses and relates the findings to the relevant studies from the medical literature. The potential significance of this work to clinical nursing practice and further research are discussed.

Analysis of Individual Subjects

The eight subjects studied were critically ill medical and surgical patients admitted to the PICU.

Temperature patterns were compared to prehospital customary sleep and awake times. Three subjects retained evidence of either their prehospital customary sleep or awake times, while only two subjects showed evidence of their prehospital temperature pattern during both customary sleep and awake times. Last, two subjects showed evidence of their prehospital customary awake time on day 1 but not on day 2. Possible explanations for the differences across subjects include the following. First, the two subjects that had evidence of their prehospital temperature patterns during both sleep and awake times were the oldest (13 and 17 years of age). This may reflect a greater stability in their temperature patterns as a result of their age and higher level of maturity. Studies

have shown the presence of a body temperature rhythm in infants as young as 2 days of age (Weinert et al., 1994) however, none of the studies reviewed looked at the influence that puberty or continued growth and development may have on the stability of circadian rhythms. The ages of the two subjects in this study suggest that increased age or development may play a role. The differences between the first and second day that was noted in two subjects may be due to either a change in their environment resulting in a disruption of customary time cues, or a change in their physiological state as a result of a progression in their illness. In a study of the desynchronization of body temperature after subarachnoid hemorrhage, the authors noted that the prominent period was changing relative to the clinical course, especially the level of response (Fujishima & Motohashi, 1991). Finally, some subjects maintained evidence of their prehospital customary sleep patterns but not awake patterns (and vice versa). This may indicate either a greater amount of environmental disturbance during either their customary awake or their customary sleep time, or it may reflect that either their sleep or their awake patterns are more vulnerable to changes or illness.

Other influences on the noted body temperature patterns may include the presence of fever, use of acetaminophen, amount of time the subjects were asleep as a result of their illness or medication, the impact of certain interventions such as endotracheal tube suctioning, and the underlying severity of their illness. Seven of the eight subjects were febrile ($> 38^{\circ}\text{C}$) at least during some part of the data collection. Mean body temperatures for all 8 subjects ranged from 37.3°C to 39.1°C . Some patients received acetaminophen as an antipyretic which would influence body temperature. All subjects were estimated to be asleep during a significant portion of daytime hours which may have affected body temperature patterns. All subjects were mechanically ventilated. Small fluctuations in body temperature were temporally associated with painful or noxious stimuli such as

endotracheal tube suctioning or central venous catheter placement. Finally, changes in the magnitude of temperature fluctuations tended to be associated with severity of illness.

Studies have shown that the presence of fever does not disrupt the temporal pattern of normal body temperature fluctuations. Instead, when the episode of fever is brief, the normal temperature rhythm is rapidly restored following release of endotoxins or other pyrogens that cause fever. In sustained periods of fever, temporal patterns of body temperature are maintained although temperature fluctuations may be oscillating about a higher mean over time (Tweedie, Bell, Clegg, Campbell, Minors, & Waterhouse, 1989).

In the PICU, air from mechanical respirators is humidified and warmed to 37°C. It is not definitively known what effect, if any, warmed inspired gas may have on body temperature or fluctuations in body temperature. Based upon results from an adult study (Hoeksel, 1991), it is likely that the temperature of inspired air from the ventilator has no significant effect on body temperature fluctuations, but the effect on absolute body temperature is unclear. In other words, it is unclear if the use of warmed inspired gas caused an elevation in the mean body temperature over time. In this study population, the effect would likely be minimal as temperature was measured via bladder probe versus an oral site used in the adult studies.

Temperature fluctuations ranging from 0.3°C to 0.5°C were noted with noxious stimuli. These fluctuations followed a predictable pattern in that there was an increase in temperature occurring within 5 minutes of the stimulus and returning to baseline within 20 - 25 minutes. These fluctuations were likely due to the subject's stress response with release of endogenous catecholamines, an increase in catabolism, and increased metabolic rate. This is manifested by an increase in heart rate, cardiac output and vasoconstriction resulting in a brief increase in body temperature.

Review of the environmental data demonstrated that many subjects were reported as being asleep for long periods during the day and night. Since sleep has a known masking effect on body temperature rhythms, it may be that the prolonged sleep periods in this population resulted in diminished or absent temperature patterns. However, the PICU data records were not always complete or accurate. In addition, there was no reliable method used to determine awake or sleep states. Therefore, this information was only noted but may not be reliably factored into the analysis of the data.

Acetaminophen administration was sometimes associated with a decrease in body temperature within 15-30 minutes following administration. Acetaminophen inhibits prostaglandin production in the hypothalamus and therefore blocks set point elevations and maintains the set point at near normal levels. It decreases the set point only in febrile conditions because it is only in those cases that prostaglandins are the intervening signal. Onset of action occurs within 10 to 60 minutes depending on route of administration and the pH of the stomach. Its half-life is 1.25 to 3 hours. While fever on its own does not influence the rhythmic pattern of body temperature, acetaminophen may cause an exogenously mediated decrease in body temperature that is not related to the body's own temperature fluctuations associated with circadian rhythms. Therefore, since acetaminophen administration may artifactually affect analysis of body temperature patterns by lowering body temperature, changes in body temperature that coincided with its administration were not considered during the visual analysis.

Perhaps the most important finding in this study revealed during visual inspection of individual subject's temperature graphs, was that the magnitude of body temperature fluctuation varied markedly between subjects. This may be due in part, to the differences between PICU admission times and start of data collection (e.g. day vs night effect) as noted above. Alternatively, the difference in the magnitude of fluctuations in body temperature between subjects may be due to the underlying severity of illness. Table 3

shows that PRISM scores ranged from 6 to 45. Therefore, some subjects had a low severity of illness and others had a high severity of illness. Studies of heart rate variability in severely ill or injured pediatric patients reported that the total amount of heart rate variability decreased as severity of illness or injury increased (Goldstein, 1997). If the hypothesis that the degree of physiologic signal variability is negatively correlated with severity of illness applies to the magnitude of fluctuations in body temperature, then increased fluctuations in body temperature may be associated with decreased severity of illness. Conversely, decreased fluctuations would be associated with a higher severity of illness. This hypothesis is supported by the work of Alster, Pratt, & Feinsod (1993). They noted in their study of temperature rhythms in comatose patients, that fluctuations in body temperature may be due to a decrease in the subject's ability to respond to the environment as a result of increased severity of illness. In other words, fluctuations in body temperature, especially if they resembled cyclic variations that were fluctuating about a mean, could be interpreted as an internal attempt to correct the rhythm by a feedback system whose receptive ability to external cues is damaged or depressed but not absent. They further concluded that a loss of temperature oscillation for an extended period of time was not a good sign in terms of patient outcome (Alster et al., 1993).

Another potential explanation for the differences in the magnitude of fluctuation in body temperature between subjects may be the result of direct injury to the thermoregulatory center in the brain. When the thermoregulatory center is damaged or destroyed, the body assumes a poikilothermic state (i.e. it assumes the temperature conditions of its environment). If the temperature of the environment is fluctuating with a large amplitude, then the recorded temperature of a subject with a damaged thermoregulatory center would fluctuate with a similar amplitude. Tweedie, Bell, & Clegg's (1989) findings of increased amplitudes in subjects who were premorbid supports this concept.

In summary, the main findings noted from the visual graphical analysis and noted when comparing body temperature patterns to prehospital customary sleep and awake times was that only two subjects retained remnants of their prehospital body temperature patterns during both their reported customary sleep and awake times. In addition, there were differences in the magnitude of body temperature fluctuations. The small number of subjects who maintained their prehospital body temperature patterns and the differences in the magnitude of body temperature fluctuations were probably due to the combined effect of the environment of the PICU and differences in the severity of their underlying illness. This hypothesis is supported further in the group analysis.

Fast Fourier Transform and Periodogram Analysis

There was no statistical evidence of a major periodic component in body temperature fluctuations in any subject. This differs from what was previously reported in the adult literature where studies on patients in a medical ICU detected periods of 6, 12, 16, and ≥ 24 hours (Lanuza et al., 1989; Felver, 1990).

The absence of periodic temperature fluctuations in this study population may have several explanations. First, adults in a medical or surgical ICU may have a different response to critical illness or injury than children in a PICU. In addition, many subjects from the adult studies had diagnoses that were usually cardiac related. This greatly differs from the study population who were between 3 and 17 years of age, none of whom had primary cardiac disease. Critically ill children may have significantly different responses in body temperature patterns compared to adults, thus resulting in a lack of periodicity.

Second, as mentioned earlier, the overt temporal pattern of a physiological variable is the net result of the endogenous rhythm that is entrained and influenced by the environment (Weinert et al., 1994). Body temperature follows a circadian rhythm that has a nadir in the early morning and peaks in the late afternoon in a day active person.

The cycle is influenced by exogenous factors, the most prominent being the light and dark cycle (Maher, Browne, Daly, McCann, & Daly, 1993). When an individual is removed from the environment to which their biologic rhythms are entrained, their biologic rhythms resynchronize to the new environment (Redfern et al., 1994). The environment of the ICU lacks or alters environmental patterns that entrain endogenous rhythms (Felver, 1989). In addition, the environment contains numerous factors which are exogenous influences or transient stimuli that alter the pattern of the individual's sleep / wake schedule (Felver & Hoeksel, 1993). Temperature is one of the more stable rhythms, taking 7-14 days to re-entrain fully in adults (Felver, 1995). It is possible that the PICU environment may have resulted in significant disruption in the subject's body temperature patterns resulting in a complete loss of periodicity. However, as noted earlier, subjects were studied during the first 5 days after PICU admission to reduce this effect. It is possible that body temperature rhythms may re-entrain faster or become disrupted more easily in children than in adults. The lack of a standard entry time into the study may also have played a role in determining the results, but the need for flexibility if the subject's condition required additional nursing or medical assessment or interventions unavoidably delayed the start of data collection in some subjects.

Thirdly, it is known that for patients in an ICU, there may also be alterations in the expression of their biologic rhythms due to their pathology or pathophysiology, or because of changes in their physiologic status as their disease state progresses or resolves (Felver, 1995). While critically ill adults have been reported to maintain some, albeit frequently abnormal, temperature patterns in an ICU, children may have a different pathophysiologic response resulting in diminished or loss of temperature rhythms.

Fourthly, surgery and anesthesia may affect biologic rhythms. Four of the eight subjects in this study were postoperative. In a review of the literature, Lanuza (1995) discussed several studies which looked at circadian rhythms in postoperative adults.

Lanuza identified a disruption in diurnal and circadian rhythms that occurred in most studies and was influenced by the severity of the surgical procedure. In her own study, Lanuza (1995) studied the pattern of body temperature rhythms in 20 adults who underwent cardiac surgery. Temperature was measured orally and recorded every two hours. She found that no subject experienced temperature peaks during the expected time frame of 11-15 hours after midsleep. Instead, subjects either: (1) had peaks which occurred earlier than the expected time (and so were phase-advanced); (2) had peaks that were phase-delayed occurring much later at night; or, (3) had no significant rhythm at all during the 24-hour period possibly as a result of a prolonged stress response. This last pattern is consistent with our results. All postoperative subjects in this study experienced significant injuries or underwent extensive surgical procedures. The resultant prolonged stress response, similar to that observed in Lanuza's study, may explain the lack of temperature periods.

The effect of anesthesia as an independent factor influencing body temperature is difficult to assess since its presence is usually concurrent with a postoperative status. In other words, it is difficult to separate the effect of anesthesia versus the effect of surgery. A study by Sessler, Lee, & McGuire (1991) attempted to differentiate the effect of anesthesia from the effect of surgery by assessing the effect of isoflurane anesthesia administration in 5 healthy male volunteers. In this study, isoflurane anesthesia was administered starting at approximately 1000 hours for 3 hours. Central body temperature was recorded at 3 minute intervals for 2 days prior to receiving the anesthesia, during administration of the anesthesia and then for 2 days after, using an ingested sensor. The authors did not report any significant changes in the central temperature mesor, but temperature amplitude was significantly reduced on the day of anesthesia. The amplitude remained reduced until the next day. Furthermore, they reported no statistically significant change in acrophase on the day following anesthesia when compared to the 2

days preceding the administration. The authors conclude that isoflurane anesthesia had no effect on the pattern of body temperature. Therefore, the alterations in body temperature patterns noted in the postoperative subjects may be due to the effect of the surgery, or it may be that the effect of the stress response in the postoperative subjects in this study overwhelmed any effect of anesthesia.

Finally, the most likely explanation for the lack of periodicity in the data is due to Type II beta error. While some patterns were noted in this small sample, no definitive conclusions may be drawn since the sample size hinders the power of the statistical analysis. The study population was small due to the restricted time frame during which the study took place. In addition, during the first month of data collection, the PICU census had severely decreased.

The final limitation was the amount of data collected for each subject. Fast Fourier Transform and Periodogram analysis (FFT) requires that there be twice the amount of data collected for the period trying to detect. Therefore, in order to detect 24 periods, data sets need to be 48 hours in length. Although all patients were screened to determine if their length of stay in the PICU was predicted to be ≥ 48 hours, this was based on the subjective opinion of the patient's nurse and physician. In several instances, the patients' condition improved and they were discharged before the end of data collection. Several subjects had their indwelling catheter discontinued prior to the end of data collection because their physiological condition no longer required monitoring of their urine output.

Several subjects had ≥ 48 hours of data collected. However, all had missing data points (e.g. data was erroneously not printed, subject was taken off the bedside monitor during intrahospital transport, data deleted due to possibility of rewarming trend after surgery, and indwelling catheter irrigation with room temperature normal saline). In addition, FFT analysis requires that the number of data points be a power of 2. The data

sets of several subjects were between 256 and 512 data points. For these subjects, the data set was truncated to the lower interval when running the FFT. This further decreased the amount of data available for analysis and, in some cases, resulted in < 48 hours of data. The level of discrimination is decreased as the number of data points falls from 512 to 256. Data sets of 256 may only detect periodicities around 24 hours at approximations of either 21 or 31 hours. For data sets of 512, it is possible to determine the presence of 24 hour periods with greater precision. Thus, when looking for circadian rhythms with 24 hour period lengths, using data sets of 512 or greater is preferable. This would require longer data collection than was possible in this study.

Maintaining control of the temperature of the environment in the PICU would facilitate the differentiation of the effect of increased severity of illness on body temperature patterns (degree of fluctuation) from body temperature fluctuations due to an impaired or absent thermoregulatory system. But maintaining thermal control would be difficult to impossible in a PICU. Therefore, it would be necessary to analyze subjects with presumed injury to their thermoregulatory system separately from the other subjects.

All these factors contributed to prohibiting a complete and thorough statistically sound analysis. However, the preliminary results from this study indicate the potential fruitfulness of further research in this area to evaluate temperature patterns more completely in the PICU population.

Analysis of the Study Group as a Whole

Following individual visual graphical analyses, subjects were divided into groups according to diagnosis (elective postoperative, trauma postoperative, trauma, meningococemia, and hepatic failure), PRISM score (low, moderate, and high) and outcome (good, poor neurological, and poor overall function). Deviations from prehospital patterns in body temperature during customary sleep and awake times within each grouping are shown in Tables 4-6.

Results from Table 4 show that there were differences across diagnosis. In addition, comparisons demonstrated that the subjects in the postoperative (both elective and trauma) tended to retain evidence of their prehospitalization temperature pattern while the subjects in the meningococemia group did not. These differences may be due to the ages of the subjects in the postoperative group as noted earlier (2 of the subjects were teenagers), or it may reflect that their underlying illness exerted a greater impact on their physiologic state which resulted in a greater disruption in their temperature patterns.

Table 5 supports this hypothesis. This table shows that subjects with a greater severity of illness tended to retain fewer or no components of their prehospital temperature pattern than those with lower severity of illness. These results imply that a greater severity of illness causes a greater or complete disruption of body temperature patterns. These results are consistent with the adult literature noted in the preceding sections.

Finally, the subjects were grouped according to outcome (Table 6). Again, these results provide further support to the hypothesis that disruptions in body temperature patterns are associated with severity of illness. In this grouping, all subjects with good outcomes demonstrated retention of their prehospital temperature patterns during either their customary sleep or their customary awake time, or both. In contrast, the subject who suffered a poor overall function exhibited normal patterns during his customary awake time but not during his customary sleep time. Finally, of the subjects in the poor neurologic group (which were the more severely affected since a poor neurologic outcome causes an associated poor overall function), 1 did not retain any pattern of his prehospital temperature pattern on either day while the other 2 subjects retained evidence of their prehospital body temperature patterns during their customary sleep time on day 1, but not on day 2. The loss of their body temperature patterns over time was consistent with a change in their discharge outcome scores when compared to their baseline scores,

indicating that as their illness or injury progressed, there was further disruption in their body temperature patterns. The first subject had a significant change in his level of functioning on the first day which is consistent with there being no remnants of his prehospital body temperature patterns on Day 1. In addition, none of the subjects in the poor neurologic group maintained their prehospital body temperature pattern during their customary sleep time. The overall results of this group showed that those with a poor outcome as a result of their underlying illness or injury had greater disruption in their body temperature patterns. This supports the results found in the PRISM group, and is also consistent with the findings in the adult literature.

Pearson Chi square correlations were performed on the three subsets of data to identify the strength of the relationship between the above groups and the presence of prehospital temperature patterns. None of the results showed a statistically significant correlation. However, this may be due to the limited sample size and the low power of the statistic used as a result of the small sample size. Power analysis revealed that a sample size of at least 30 subjects would be needed for more definitive results. Therefore, further research using a larger sample size is needed in order to more accurately quantify this relationship.

Significance to Clinical Nursing Practice

As stated earlier, an important function of nursing is to assess and interpret temperature measurements with the primary objective to detect patterns and trends which might indicate altered outcomes. It is known that physiologic variables may vary according to time of day as a result of changes in their underlying biologic rhythms. These fluctuations are manifested by changes in vital signs and biochemical laboratory values. Therefore, knowledge of patients' own circadian rhythms may be useful in nursing assessment, monitoring, and in planning patient care (Lanuza, 1995). Further, in order to make accurate assessments of the patients' status when evaluating them for signs

of infection, hypermetabolism, endocrine disturbances, drug effect, etc., it is necessary to take into account normal fluctuations in body temperature. In addition, awareness of an increased vulnerability and its consequences (ex. prolonged recovery or altered outcome) alerts the nurse of the need for careful monitoring and assessment for early detection and prevention of complications.

Specific timing of medications and therapeutic interventions to coincide with biological rhythms (Thomas, 1995) are examples of applying a knowledge of biological rhythms in health care. But evaluation of temporal patterns of body temperature is currently not a standard of practice. There is potential, however, in its use as a monitoring tool for assessing detrimental environmental influences or underlying pathophysiologic processes. In other words, if normal patterns of body temperature fluctuations are lost or altered, could this be a sign of a detrimental environmental change (e.g. an alteration in room temperature, lighting patterns, ambient noise levels, or altered sleep/wake cycles, etc.). Alternatively, could changes in body temperature fluctuations be a sign of an underlying pathophysiologic process such as infection, central nervous system disturbance, or metabolic imbalance (Felver, 1995). If alterations in patterns were detected, and their cause identified, it may be possible to institute measures to decrease the impact of the PICU environment, or decrease the impact of their illness.

In order to use this information as a monitoring tool, however, customary prehospitalization bedtimes and awake times need to be assessed on PICU admission and added to the flowsheets so that the information is readily available. In addition, more sophisticated means of detecting alterations in fluctuations need to be developed for use at the patient's bedside. Due to the need for frequent and accurate measurements and recordings of body temperature in order to note changes in temporal patterns, simply recording hourly temperature measurements as is done in current practice would not provide enough information. Instead, techniques which would analyze frequent

measurements or continuous recordings are needed. Non-linear techniques such as power spectral analysis, and detrended fluctuation analysis can analyze frequent measurements and are currently utilized in clinical research studies. However, the tools for using these techniques in real time have not yet been developed.

Future Research

This study provided a basis for the analysis of body temperature patterns in the PICU. However, there were several limitations in this study as noted earlier. Therefore, research is needed in the following areas.

First, this study used a small sample (N=8) with a wide variation in subject age (range = 3 to 17 years). Therefore, this study needs to be repeated with a larger sample size that could be divided into age groups to allow factoring out developmental issues. In addition, research in normal, healthy children is needed to serve as controls.

Second, this study needs to be repeated with longer periods of data collection so that there is an adequate number of data points to detect periods of varying length. This should consider the constantly changing baseline due to changes in febrile state that was noted in this study in order to divide the data set up into segments.

Third, further research in the field of altered patterns as a result of illness or injury or the PICU environment is needed in order to define and evaluate its clinical applicability and specifically, its use as a monitoring tool. This includes research that would identify predictors of the pattern of body temperature and predictors of a change in the pattern. To facilitate this, the research should consider use of more sophisticated techniques with continuous recordings such as the non-linear techniques named above.

Last, if disrupted rhythms are detected, then an additional aspect would be to determine how long it takes for disrupted circadian rhythms to return to normal.

Conclusion

This study provided an initial description of body temperature patterns in pediatric patients in the PICU. There was no evidence of a major periodic component in body temperature fluctuations found in any subject. The graphical analysis of each subject and of groups of subjects, however, indicated that there may be a relationship between alterations in body temperature patterns and severity of illness and outcome. This is an important finding that may have significant clinical relevance. Further study of this area, is therefore warranted.

References

- Alster, J., Pratt, H., & Feinsod, M. (1993). Density spectral array, evoked potentials, and temperature rhythms in the evaluation and prognosis of the comatose patient. Brain Injury, 7(3), 191-208.
- Aschoff, J. (1976). Circadian systems in man and their implications. Hospital Practice, 11, 51-7.
- Baker, C. (1984). Sensory overload and noise in the ICU: Sources of environmental stress. Critical Care Quarterly, 6, 66-80.
- Ballard, K. (1981). Identification of environmental stressors for patients in a surgical intensive care unit. Issues in Mental Health Nursing, 3, 89-108.
- Bell, C., Campbell, I., Minors, D., & Waterhouse, J. (1984). Body temperature rhythms in intensive care. British Journal of Anesthesia, 56, 428.
- Boivin, D., Duffy, J., Kropnauer, R., & Czeisler, C. (1996). Dose-response relationships for resetting of human circadian clock by light. Nature, 379, 540-545.
- Brown, P.J., Dove, R.A., Tuffnell, C.S., & Ford, R.P. (1992) Oscillations of body temperature at night. Archives Dis Child, 67, 1255-1258.
- Campbell, I.T., Minors, D.S., & Waterhouse J.M. (1986) Are circadian rhythms important in intensive care? Intensive Care Nursing, 1, 144-150.
- Carli, F., & Aber, V. (1987). Thermogenesis after major elective surgical procedures. British Journal of Surgery, 74, 1041-1045.
- Clancy, J. & McVicar, A. (1994). Circadian rhythms 1:Physiology. British Journal of Nursing, 3, 657-661.
- Cohen, D., & Sherman, S. (1988). The Nervous System. In R. Berne, & M. Levy (Eds.) Physiology 2nd edition. St. Louis: C.V. Mosby Company.

Czeisler, C., Allan, J., Strogatz, S., Ronda, J., Sanchez, R., Rios, C., Freitag, W., Richardson, G., & Kronauer, R. (1986). Bright light resets the human circadian pacemaker independent of the timing of the sleep-wake cycle. Science, 233, 667-670.

Dobree, L. (1993). How do we keep time? Understanding human circadian rhythms. Professional Nurse, 4, 446-449.

Erickson, R., & Moser-Woo, T. (1994). Accuracy of infrared ear thermometry and traditional temperature methods in young children. Heart & Lung, 23, 181-195.

Falk, S., & Woods, N. (1973). Hospital noise: levels and potential health hazards. The New England Journal of Medicine, 289, 774-781.

Farr, L., Campbell-Grossman, C., & Mack, J. (1988). Circadian disruption and surgical recovery. Nursing Research, 37, 170-175.

Farr, L., Gaspar, T., & Minors, D. (1984). Desynchronization with surgery. In E. Haus & H.F. Kabat (Eds.), Chronobiology (pp544-547). New York: Karger.

Farr, L., Keene, A., Samson, D., & Michael, A. (1984). Alterations in circadian excretion of urinary variables and physiological indicators of stress following surgery. Nursing Research, 33, 140-146.

Farr, L., Keene, A., Samson, D., & Michael-Jacoby, A. (1986) Relationships between disruption of rhythmicity and reentrainment in surgical patients. Chronobiologia, 13, 105-113.

Felton, G. (1987). Human biologic rhythms. In J.J. Fitzpatrick, & R.L. Tacenton (Eds.), Annual review of nursing research, Vol. 5. New York: Springer Publishing Co.

Felver, L., (1990). Temporal patterns of oral temperature in intensive care patients. Communicating Nursing Research, 23, 124.

Felver, L. (1991). Temporal patterns of body temperature and heart rate in patients in intensive care units. Communicating Nursing Research, 24, 228.

Felver, L. (1995). Patient-environment interactions in critical care. Critical Care Nursing Clinics of North America, 7, 327-335.

Felver, L., & Hoeksel, (1993). Uninterrupted time during patients' usual sleeping times in ICU. Communicating Nursing Research, 1, 138.

Felver, L., & Pike, R. (1990). Relationship of heart rate, respiratory rate, and arterial blood pressure rhythms in a mechanically ventilated patient to environmental variables in an intensive care unit. Chronobiology: Its Role in Clinical Medicine, General Biology, and Agriculture, Part A. Wiley-Liss, Inc.

Fiser, D. (1992). Assessing the outcome of pediatric intensive care. The Journal of Pediatrics, 121, 68-74.

Gervais, P., Reinberg, A., Pollak, E., Aluelker, C., Dupont, J., Nicaise, A., & Pollet, J. (1973). Persistence and-or-alternation of various circadian rhythms in toxic comas (temperature, pulse arterial pressure, water, and urinary potassium. European Journal of Toxicology, 6, 94-99.

Glotzbach, S., Edgar, D., & Ariagno, R. (1995). Biological rhythmicity in preterm infants prior to discharge from neonatal intensive care. Pediatrics, 95, 231-237.

Glotzbach, S., Edgar, D., Bopeddiker, M., & Ariagno, R. (1994). Biological rhythmicity in normal infants during the first three months of life. Pediatrics, 94, 482-488.

Goldstein, B., Fiser, D., Kelly, M., Mickelsen, D., Ruttimann, U., & Pollack, M. (in press) Decomplexification in critical illness and injury: the relationship between heart rate variability, severity of illness, and outcome. Critical Care Medicine.

Goswami, R., Krishan, K., Suryaprakash, B., Vaidyanathan, S., Rao, K., Rao, M., Goswami, A., Goll, A., & Sharma, P. (1985). Circadian desynchronization in pulse rate, systolic and diastolic blood pressure, rectal temperature, and urine output in traumatic tetraplegics. Indian Journal of Physiology and Pharmacology, 29, 199-206.

Hellburgge, T. (1974). The development of circadian and ultradian rhythms of premature and full-term infants. In L.E. Scheving (Ed.), Chronobiology. Tokyo: Igaky Shoin.

Hildebandt, G. (1976). Circadian variations of thermoregulatory response in man. In L.E. Scheving (Ed.), Chronobiology (pp.234-240). Tokyo: Igaky Shain.

Hilton, B. (1985). Noise in acute patient care areas. Research in Nursing & Health, 8, 283-291.

Hoeksel, R. (1991). Temporal patterns of bladder, oral, pulmonary artery, and rectal temperature in critically ill adults in a surgical intensive care unit. Unpublished doctoral dissertation, Oregon Health Sciences University.

Lanuza, D., Robinson, C., Marotta, S., & Patel, M. (1989). Body temperature and heart rate rhythms in acutely head-injured patients. Applied Nursing Research, 2, 135-139.

Lanuza, D. (1995). Postoperative circadian rhythms and cortisol stress response to two types of cardiac surgery. American Journal of Critical Care, 4 (3), 212-220.

Leach, C., Ruskin, J., Halberg, F., & Siother, R. (1983). Infradian variability in total (TE) and longest runs (LE) cardiac ectopies, heart rate (H), blood pressure and temperature (T), of a comatose man. Chronobiologia, 10, 138.

Lodmore, M.R., Petersen, S.A., & Wailoo, M.P. (1992). Factors affecting the development of night time temperature rhythms. XXXX

Maher, J., Browne, P., Daly, L., McCann, S., & Daly, P. (1993) A circadian ditribution to febrile episodes in neutropenic patients Support Care Cancer, 1, 98-100.

Mills, J. N., Minors, D.S., & Waterhouse, J.M. (1978). The effect of sleep upon human circadian rhythms. Chronobiologia, 5, 14-27.

Minors, D.S., Nicholson, Spencer, Stone, & Waterhouse J.M. (1986). Irregularity of rest and activity; Studies on circadian rhythmicity in man. Journal of Physiology, 381, 279-295.

- Minors, D.S., & Waterhouse, J.M. (1981). Circadian rhythms and the human. Boston: Wright PSG.
- Minors, D.S., & Waterhouse, J.M. (1983). Does “Anchor Sleep” entrain circadian rhythms? Evidence from constant routine studies. Journal of Physiology, 345 451-467.
- Minors, D.S., & Waterhouse, J.M. (1986). Circadian rhythms and their mechanisms. Experientia,42, 1-13.
- Moore, R. Y., & Eichler, V.B. (1972). Loss of a circadian adrenal corticosterone rhythm following suprachiasmatic lesions in the rat. Brain Research,42,201-206.
- Moore, R. Y., & Lenn, N.J. (1972). A retinohypothalamic projection in the rat. Journal of Comparative Neurology,146, 1-14.
- Moore-Ede, M.C. (1986). Physiology of the circadian timing system: Predictive versus reactive homeostasis. American Journal of Physiology,250, R735-R752.
- Moore-Ede, M.C., Czeisler, C., & Richardson, G. (1983). Circadian timekeeping in health and disease: Part 1. Basic properties of circadian pacemakers. New England Journal of Medicine, 309, 530-536.
- Moore-Ede, M., Sulzman, F., & Fuller, C. (1982). The clocks that time us. Cambridge MA: Harvard University Press.
- Okawa, M., Takahasi, K., & Sasaki, H. (1986). Disturbance of circadian rhythms in severely brain-damaged patients correlated with CT findings./ Neurology, 274-282.
- Pagano, M., & Gauvreau, K. (1993). Principles of Biostatistics. California: Duxbury Press.
- Pollack M., Ruttimann, U., & Getson, P. (1988). Pediatric risk of mortality (PRISM) score. Critical Care Medicine, 16, 1110-1116.
- Redfern, P., Minors, D., & Waterhouse, J. (1994). Circadian rhythms, jet lag, and chronobiotics: An overview. Chronobiol Int, 11, 253-265.

Rigaud, D., Accary, J., Chastre, J., Mignon, M., Laigneau, J., reinber, A., & Bonfils, S. (1988). Persistence of circadian rhythms in gastric acid, gastrin, and pancreatic polypeptide secretions despite loss of cortisol and body temperature rhythms in man under stress. Gastroenterology Clinical Biology, 12, 12-18.

Samples, J., Van Cott, M., Long, C., King, I., & Kersenbrock, A. (1985). Circadian rhythms: Basis for screening for fever. Nursing Research, 34, 377-379.

Schechtman, V., & Harper, R. (1991). Time of night effects on heart rate variation in normal neonates. Journal of Developmental Physiology, 16, 349-353.

Shinozaki, T., Deane, R., & Perkins, F. (1988). Infrared tympanic thermometer: Evaluation of a new clinical thermometer. Critical Care Medicine, 16, 148-150.

Sitka, U., Weinert, D., Berle, K. Rumler, W., & Schuh, J., (1994). Investigations of the rhythmic function of heart rate, blood pressure and temperature in neonates. European Journal of Pediatrics, 153, 117-122.

Sorensen, K., & Luckmann, J. (1986). Basic Nursing, A Psychophysiologic Approach, 2nd Edition. Philadelphia: W.B. Saunders Company.

Spencer, K., & Bale, S. (1990). A logical approach: management of surgical wounds. Professional Nurse, 5, 303-307.

Tenreiro, S., Dowse, H., D'Souza, S., Minors, D., Chiswick, M., Simms, D., & Waterhouse, J. (1991). The development of ultradian and circadian rhythms in premature babies maintained in constant conditions. Early Human development, 27, 33-52.

Thomas, K. (1995). Biorhythms in infants and role of the care environment. Journal of Perinatal and Neonatal Nursing, 9, (2), 61-75.

Topf, M. (1992). Effects of personal control over hospital noise on sleep. Research in Nursing & Health, 15, 19-28.

Tweedie, L., Bell, C., & Clegg, A. (1987). Retrospective study of temperature rhythms of intensive care patients. Critical Care Medicine, 17 (11), 1159-1165.

Weinert, D., Sitka, U., Minors, D., & Waterhouse, J. (1994). The development of circadian rhythmicity in neonates. Early Human Development, *36*, 117-126.

Wilkinson, L., Hill, M., & Vangi, E. (1992). Systat Statistics Manual. Evanston Illinois: Systat Inc.

Wilson, V. (1987). Identification of stressors related to patients' psychological responses to the surgical intensive care unit. Heart & Lung, *16*, 267-273.

Winget, C.M., DeRoshia, C.W., Markely, C.L., & Holley, D.C. (1984). A review of human physiological and performance changes associated with desynchronization of biological rhythms. Aviation, Space, Environmental Medicine, *55*, 1085-1096.

Woods, S. (1991). Temporal patterns of body temperature and heart rate in patients in intensive care units. Communicating Nursing Research, *24*, 227.

Woods, N.F., & Catanzaro, M. (1988). Nursing research: Theory and practice. St. Louis: C.V. Mosby.

APPENDIX A
INFORMED CONSENT FORM

IRB# 4337
Date of Approval: 3/7/97

OREGON HEALTH SCIENCES UNIVERSITY
CONSENT FORM

TITLE: Body Temperature Rhythms in the Pediatric Intensive Care Unit

Principal Investigator: Karen Sonnenthal, RN (Telephone 503-494-4591)
Brahm Goldstein, MD (Telephone 503-494-1544)
Linda Felver, RN, Phd (Telephone 503-494-3723)

PURPOSE

Henceforth, "you" will refer to "you/your child" in this consent form.

You have been invited to participate in this research project because you are a patient in the Pediatric Intensive Care Unit (PICU). The purpose of this study is to describe the patterns of body temperature rhythms in patients of all ages admitted to the PICU. Many physiological variables fluctuate over time. These biological rhythms occur about every twenty-four hours, and are termed circadian rhythms. An example of such a biological rhythm is changes in body temperature. The effect of admission to an intensive care unit on biological rhythms has been documented in adults but not in children. By recording measurements of temperature, we can identify the pattern of the body temperature changes and in doing so, evaluate the changes in the rhythm that occur during illness and hospitalization in children.

PROCEDURES:

If you agree to participate in this study, we will ask you or transcribe from your chart your date of birth, gender, current medical diagnosis, height, weight, operations performed during this hospitalization, and list of current medications. In addition, you will be asked about your regular sleeping habits outside of the hospital. Then your temperature will be measured by one of two ways. If your condition warrants that you have a foley catheter (a catheter that is placed into your bladder to monitor your urine output), then your temperature will be measured every five minutes by a thermometer that is part of the foley catheter. If you do not have a foley catheter, then your temperature will be measured using an "ear-based" thermometer, that is placed gently in the outer portion of the ear canal, every hour while you are awake for the next forty-eight hours. The frequency of this measurement is consistent with the standard of care for all patients admitted to the PICU. If your medical condition requires additional temperature measurements while either awake or asleep, we will record these as well. The temperature readings will be recorded more frequently with the foley catheter since with this method, your temperature is continuously displayed on the bedside monitor and the readings can therefore be done without disturbing you.

RISKS AND DISCOMFORTS

There is no predictable risk from participation in this study.

BENEFITS

There is no known benefit from your participating in this study. However, by serving as a subject, you may contribute to new information which may benefit patients in the future.

COSTS

You will incur no costs by participating in this study. Routine care costs will include all charges for hospitalization, administration of drugs, laboratory tests, imaging studies, and physician fees.

ALTERNATIVE:

If you decide not to participate or to withdraw from the study at any time, your doctors will continue to provide the same treatment and care.

CONFIDENTIALITY

A record of the results of these studies will be kept in a confidential locked file in the investigator's office. The confidentiality of this file is carefully guarded and no information by which you can be identified, such as name or social security number, will be released or published. While the data from the study and / or results may be published, your name will not be used for publication or publicity purposes.

LIABILITY

The Oregon Health Sciences University, as a public institution, is subject to the Oregon Tort Claims Act, and is self insured for liability claims. If you suffer any injury from the research project, compensation would be available to you only if you establish that the injury occurred through the fault of the University, its officers or employees. However, you have not waived any of your legal rights by signing this consent form. If you have further questions, please call the Medical Services Director at (503) 494-8014.

PARTICIPATION

If you would like further information about the study, or if you have any further questions at any time, you may contact Karen Sonnenthal, RN at (503)-494-4591. If you have any questions regarding your rights as a research subject, you may contact the Oregon Health Sciences University Institutional Review Board at (503)-494-7887.

You may refuse to participate, or you may withdraw from this study at any time without affecting your relationship with or treatment at the Oregon Health Sciences University. You may be withdrawn from this study by the principle investigator at any time if she feels it is in your best interest.

Your signature below indicates that you have read the foregoing and agree to participate in this study. A copy of this consent form will be given to you after it is signed.

Subject name: _____ Unit No. _____

Signature: _____ Date _____

Patient/Guardian _____ Print name
_____ Signature _____ Date

Auditor Witness _____ Print name
_____ Signature _____ Date

Principle Investigator: _____ Print name
_____ Signature _____ Date

APPENDIX B
SUBJECT SCREENING TOOL AND
SUBJECT INFORMATION RECORD

Pt. Stamp:

SUBJECT SCREENING TOOL

Screening Date & Time: _____

Circle YES if the patient meets the listed criterion; NO if the patient fails to meet the criterion. Please page Karen Sonnenthal bpr 1249 for any questions.

YES / NO Age between birth and 18 years.
Birthdate: _____ Age: _____

YES / NO Patient admitted to the PICU, or transferred from the ward or another ICU before the 5th Hospital admission day.
Hospital Admission Date: _____
ICU Admission Date: _____

YES / NO Patient likely to remain in ICU for at least the next 48 hours.

YES / NO Patient is not diagnosed with: acute spinal injury with cord transection or quadriplegia, malignant hyperthermia, cold-water drowning, hypo- or hyperthyroidism, cerebral spinal fluid otorrhea, or Down syndrome.

YES / NO Patient's physical condition allows tympanic membrane temperature measurement.

YES / NO Patient is not admitted solely for a procedure (including EEG monitoring).

MEETS SUBJECT SELECTION CRITERIA (ALL CRITERIA CIRCLED YES)

_____ YES

_____ NO

Name of person performing screening: _____

Initials /med rec #

SUBJECT INFORMATION RECORD

Subject ID Number: _____

Today's Date: _____

DOB _____ AGE _____ Gender _____

Height (ft/in) _____ Weight (kg) _____ Race _____

Hospital Admission date & time: _____

ICU Admission date & time: _____

Admission GCS: _____ Admission Prism: _____

Hospital Acuity Score: _____

List Current Medical Diagnosis: _____

PMHx: Healthy: _____

Chronic Illness / Disability: _____

Surgery: Date: _____

Type: _____

Duration (hrs): _____

Anesthesia type: _____

Blood loss: _____

Fluids Admin.: _____

Prehospital sleep / wake schedule:

Awake: _____

Nap: _____

Bedtime: _____

APPENDIX C
ENVIRONMENTAL AND EVENT DATA COLLECTION FORM

PT INITIALS:
SUBJECT NUMBER:

ACTUAL TIME RECORDED	0700	0800	0900	1000	1100	1200	1300	1400	1500	1600	1700
PATIENT'S ROOM NUMBER											
PATIENT TEMPERATURE											
CURTAIN OPEN											
CURTAIN CLOSED/LIGHTS ON (any lights)											
CURTAINS CLOSED/LIGHTS OFF (any lights)											
PT AWAKE/QUIET											
PT AWAKE/ACTIVE											
SLEEPING											
SEDATED											
SEDATED WITH MUSCLE RELAX											
VISITORS IN ROOM											
TV / RADIO ON											
COOLING/WARMING BLANKET ON											
OTHER COMMENTS RE ENV.											
DAILY PRISM											
MEDICATIONS											

INSTRUCTIONS: 1. Record hourly tympanic membrane temperature measurements noting the actual time (e.g. 0713, or 0753) the measurement was taken. Please do not wake the patient to record their temperature unless their standard care dictates hourly temperatures. The exception to this is if the patient is sleeping due to sedation. If this is the case, please record hourly temperatures.
 2. Write yes or no (Y or N) for each listed condition after every recorded temperature.
 3. Please note the patient's room number, especially if the patient is moved to a different room. If the patient stays in the same room, go ahead and use arrows across to indicate no change.
 4. Please note if the patient is off the unit or any other reason which prohibits data collection.
 5. I will fill in the daily medications from the med sheets.

APPENDIX D
WORKSHEETS USED IN GRAPHICAL ANALYSIS

T003

QUESTIONS

1	2	3	4	5
QUESTION	YES	Column 3	ACET INV.	NOTES
1 DECREASE IN TEMP - BEDTIME		✓	NO	Potentially self-heating cause of rise in
2				
3				
4 TROUGH - MIDSLEEP	✓			
5				
6				
7 RISE - AWAKENING	✓			
8				
9				
10 DECREASED DURING CUST. SLEEP TIME	✓			
11				
12				
13 INCREASED DURING CUST. AWAKE TIME		✓		not due directly to awaken. but then starts to ↓. However, pt. is post-operative
14				
15				
16 PEAK IN 2ND HALF OF AWAKE TIME		✓		↑ until 3-4° pre-awaken then ↓.
17				
18				
19 DOES PEAK OCCUR WITHIN RANGE OF 20 HRS AFTER MIDSLEEP		✓		Peaks 1-8° pre Midsleep

Day 2

DD 2 data avail. Only 22.9 hrs collected.

by visual inspection

Response to sustenim - yes

night to trough - yes

more to peak - yes

Over sedation - yes.

Platow - No

fluctuation - No.

circadian rhythm evident by FFT
FFT = periodic component.

Do see remnants of circ. patt. during sustenim
sleep time.

1	2	3	4	5
QUESTION	YES	Column 3	ACET INV.	NOTES
1 DECREASE IN TEMP - BEDTIME		✓	yes	
2				
3				
4 TROUGH - MIDSLEEP		✓	YES	increasing CONTINUOUSLY, trough $\frac{1}{2}$ A
5				
6				
7 RISE - AWAKENING		✓		cont. decrease
8				
9				
10 DECREASED DURING CUST. SLEEP TIME	Ⓟ	≠		But not normal since DEPRESSING ENTIRELY / 8 sec exp + p M.
11				
12				
13 INCREASED DURING CUST. AWAKE TIME		✓	Yes	char. p arrival which was during cust. awake time
14				
15				
16 PEAK IN 2ND HALF OF AWAKE TIME	✓	✓		starts to ↓ during 2nd 1/2 (which is p 12)
17				
18				
19 DOES PEAK OCCUR WITHIN RANGE OF		✓	yes	in arrival temp at its peak but time frame only to this of mid-sleep.
20 HRS AFTER MIDSLEEP				temporal order.

Too4

Temp plateau = ~~N~~
 Response to suc. = \checkmark
 higher ∂ temp = \checkmark
 lower ∂ peak = \checkmark
 Occur ∂ stat. = \checkmark
 Cost of fluct. = No.

Bad outcome

FFT = \emptyset periodic comp.
 \emptyset statistically significant
 \emptyset see remnants.

A N I ... other N's, O's, etc.

1	2	3	4	5
QUESTION	YES	Column 3	ACET INV.	NOTES
1 DECREASE IN TEMP - BEDTIME	✓		NO	
2				
3				
4 TROUGH - MIDSLEEP	✓		NO	
5				
6				
7 RISE - AWAKENING	✓			
8				
9				
10 DECREASED DURING CUST. SLEEP TIME	✓			
11				
12				
13 INCREASED DURING CUST. AWAKE TIME	✓	4-2		↓ D1 \bar{P} tylenol given
14				
15				
16 PEAK IN 2ND HALF OF AWAKE TIME	✓			Peak noted \bar{P} admit / not \bar{P} $\Delta 1$ / ? $\Delta 2$ - $\Delta 2$ - $\Delta 2$
17				
18				
19 DOES PEAK OCCUR WITHIN RANGE OF				\bar{P} admit $\sim 16^\circ$ Day 1 = 7-8 Day 2 = 10 \bar{P}
20 HRS AFTER MIDSLEEP				

7005
~~7004~~
total
HR =
~~59.08~~
59.08

Day 1

Day 2

Plataneu? No
Response noted
to ty

higher Δ trough?
Lower Δ peak?
Occur Δ $\Delta 1$?
 $\Delta D_1 - D_2$? Δ in mean
fluorescence? No

1 ✓ Δ
2 ✓
3 ✓
4 ✓
5 Δ data
6
7

ΔD_2 - Δ shut h \downarrow until \bar{P} B.

QU

QUESTIONS

DAY 1

1	2	3	4	5
QUESTION	YES	Column 3	ACET INV.	NOTES
1 DECREASE IN TEMP - BEDTIME 1		✓	NO	initial temp ↓ D1 but then ↑ Plateaued D2
2 TROUGH - MIDSLEEP 2		✓	NO	D1 spiked but → trough / D2 = plateau
3 RISE - AWAKENING 3	✓			
4 DECREASED DURING CUST. SLEEP TIME 4		✓	NO	fluctuating some days
5 INCREASED DURING CUST. AWAKE TIME 5	✓			
6 PEAK IN 2ND HALFOF AWAKE TIME 6		✓		temp. before 1/2 then fell during 2nd 1/2 D1
7 DOES PEAK OCCUR WITHIN RANGE OF 7 HRS AFTER MIDSLEEP		✓		Peaks ~ 10° p midsleep

7006

DAY 2

Resp $\Delta D1 - D2 - 4$
 high Temp. Plateau 4
 low Response to suction
 low high & trough
 low low & peak
 low Plateau
 low Diurnal variation
 low peaks ~ 15-16 hrs p

Plateau
 Plateau but at lower temp during 2nd half in that there was a trough.
 relatively flat / small + fluctuation noted ~ 9° p when sharply ↑ = Abnormal ↑.
 but remember that breath part during most awake period was also.

DAY 1

QUESTIONS	1	2	3	4	5
	QUESTION	YES	Column 3	ACET INV.	NOTES
1	DECREASE IN TEMP - BEDTIME	✓		NO	
2					
3					
4	TROUGH - MIDSLEEP	✓		NO	
5					
6					
7	RISE - AWAKENING	✓		YES - acet given ORD	
8					
9					
10	DECREASED DURING CUST. SLEEP TIME		✓		Q enough data to truly plot of
11					
12					
13	INCREASED DURING CUST. AWAKE TIME	✓			
14					
15					
16	PEAK IN 2ND HALF OF AWAKE TIME		✓		But acet. given X3 during awake starting 1 1/2 hrs of awake time
17					
18					
19	DOES PEAK OCCUR WITHIN RANGE OF		✓		Peaks ~ 7° of med.
20	HRS AFTER MIDSLEEP				

- 1) D₁ - D₂ - ^{Abnormal} DAY 2-1 ✓
- 2) Temp plateau - N ✓
- 3) Resp. h. suction - n / extub. 10 hrs p admit. ✓
- 4) Intubation? - N. ✓
- 5) Intubation? - N. ✓
- 6) Intubation? - N. ✓
- 7) Intubation? - N. ✓

unable to tell since data collection stopped. 10° acet. but it was steadily across awake period

DAY 1

TOO9

QUESTIONS	1	2	3	4	5
	QUESTION	YES	Column 3	ACET INV.	NOTES
1	DECREASE IN TEMP - BEDTIME	* ✓		yes	
2					
3					
4	TROUGH - MIDSLEEP	✓		yes	Peaks @ M. Note lack of temp even @ added influence from dust. Same @ 2
5					
6					
7	RISE - AWAKENING	✓		yes	Think about effect.
8					
9					
10	DECREASED DURING CUST. SLEEP TIME		✓	yes	see note D 2
11					
12					
13	INCREASED DURING CUST. AWAKE TIME	✓			but @ anti-ph. as nite.
14					
15					
16	PEAK IN 2ND HALFOF AWAKE TIME	✓			
17					
18					
19	DOES PEAK OCCUR WITHIN RANGE OF	✓			peaks ~ 13 hrs @ M.
20	HRS AFTER MIDSLEEP				

DAY 20 * ✓
 (2) ✓
 3 ✓
 4 ✓
 5 ✓
 6. ✓
 7. ✓

yes.
 yes
 NO

started to ↓ gain to B but was given by.
 2° gain to ↓. ∴ can't say peak. present
 see note D 1, Room temp was elev. D
 peak. But PZ - room temp was
 @ time last of ant. had effect. some saw in trend DT
 (not done given to gain to A.)
 Both days, temp ↓ @ B then ↑ to peak @ M
 + back ↓ @ just before A.
 Peaked 1st 1/2.
 Peaks ~ 9 1/2 hrs @ M.

QUESTIONS SEPARATED? DAY 1 / DAY 2? Day 1

1	2	3	4	NOTES
QUESTION	YES	NO	ACET INV.	
1 DECREASE IN TEMP - BEDTIME	✓			What it was down
2				at bedtime then at 2 M + down @ awake
3				
4 TROUGH - MIDSLEEP		✓		peaked at midsleep
5				
6				
7 RISE - AWAKENING		✓		↓ a + cont ↓ P A.
8				
9				
10 DECREASED DURING CUST. SLEEP TIME		✓		①
11				
12				
13 INCREASED DURING CUST. AWAKE TIME		✓		↓ During 1st 1/2 hrs awake time / slowly ↑ last 5 hrs awake
14				
15				
16 PEAK IN 2ND HALFOF AWAKE TIME	✓			Yes just before bedtime
17				
18				
19 DOES PEAK OCCUR WITHIN RANGE OF				
20 HRS AFTER MIDSLEEP		✓		Peaks 17-18 P M.

Platane? No Day 2 => Dues

Was there a response to suctioning? Yes + more than - high w @ temp NO

- 1 hr @ peak NO

- occur @ plateau YES

$\Delta O_2 - O_2$ Yes D midsleep

H... .. Not at all peak @ 2 M + down

#1) temp was further dropping rapidly @ 5 decrease occurred / started ~ 20 P B.

#2) transpiration ~ 1 1/2 hrs @ then snoring ↑ peak actually @ P M then at ~ 2 down low. unmarked. Bumps... for 2-3 hrs only ↓ 1st 3 1/2 hrs then started to snore ↑ was transiently transpiration ↓ @ awakening

#3) maybe

#4) & clear - all - down concerned supply @ P A.

#5) "

QUESTIONS

1	2	3	4	5
QUESTION	YES	Column 3	ACET INV.	NOTES
1 DECREASE IN TEMP - BEDTIME		✓		
2				
3				
4 TROUGH - MIDSLEEP	✓			
5				
6				
7 RISE - AWAKENING				
8				
9				
10 DECREASED DURING CUST. SLEEP TIME				
11				
12				
13 INCREASED DURING CUST. AWAKE TIME	✓			
14				
15				
16 PEAK IN 2ND HALFOF AWAKE TIME		✓		
17				
18				
19 DOES PEAK OCCUR WITHIN RANGE OF				
20 HRS AFTER MIDSLEEP				

To()

Difficult to tell where the baseline is.

Day 1

Day 2

- 1
- 2
- 3
- 4
- 5
- 6
- 7

Peak: ~9-10^op
 started ~11^o a B
 Did not see the peak
 ↓ to lower baseline
 only ~12^o as per noted
 did not seem temp down, awake period
 or previous day was lower than
 stayed ~ same as
 ↓
 a trough occurred ~11^o p Mid