

COMPARISON OF SUBJECTIVE REPORTS TO HEART RATE  
DURING SLEEP IN OLDER INSTITUTIONALIZED WOMEN

BY

DEANNE J. O'ROURKE

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**Comparison of Subjective Reports to Heart Rate During Sleep  
in Older Institutionalized Women**

**BY**

**Deanne J. O'Rourke**

**A Thesis/Practicum submitted to the Faculty of Graduate Studies of The University  
of Manitoba in partial fulfillment of the requirements of the degree  
of  
Master of Nursing**

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## Abstract

The purpose of this descriptive research project was to compare subjective reports of sleep quality and quantity during older institutionalized women's overnight sleep period to heart rate values and profiles obtained during the same time frame. This was undertaken to determine if relationships existed between these types of data in an effort to explore the use of heart rate monitoring as a means to objectively measure sleep patterns. A convenience sample of 23 older women living in a personal care setting was used in this study and each resident participated for one night of monitoring. The study employed the use of sleep diaries and sleep quality measurement scales as subjective indicators of the quantity and quality of sleep respectively, and portable heart rate monitors to record objective data during sleep. Statistical analysis using bivariate and step-wise multiple regression models suggested significant relationships between sleep diary data and the subjective scales. There was little statistical evidence to support significant relationships between the sleep diary data and heart rate values and there were no relationships evident between the subjective scales and the heart rate values. Visual comparison between the sleep diary data and heart rate profiles offered some positive findings for supporting the use of heart rate measurement to determine sleep patterns. Overall, the study findings do not support the use of heart rate monitoring as an independent means to indicate sleep quality and quantity and further investigation into this area of research is suggested.

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## Table of Contents

	<u>Page</u>
1. Chapter One: Statement of the Problem	9
2. Chapter Two: Literature Review	12
2.1 Normal human sleep	12
2.2 Normal age-related changes in sleep	16
2.3 Age-related sleep disturbances	19
2.4 Mechanisms of sleep control	23
2.5 Sleep measurement issues	27
2.5.1 Objective measures of sleep	28
2.5.2 Heart rate recording as an objective measure of sleep	31
2.5.3 Subjective measures of sleep	37
2.6 Conceptual framework	39
3. Chapter Three: Research Methodology	40
3.1 Operational definition of variables	40
3.2 Research questions and hypotheses	41
3.2.1 Research questions	41
3.2.2 Research hypotheses	42
3.3 Assumptions pertaining to direction of relationships	42
3.4 Instrumentation	44
3.4.1 Subjective sleep measurement	44
3.4.2 Objective sleep measurement	47
3.5 Sampling design	48
3.6 Procedure	50
3.7 Access to the research setting	51
3.8 Ethical considerations	52
3.9 Data analysis	53
3.9.1 Research hypothesis #1	54
3.9.2 Research hypothesis #2	55
3.9.3 Research hypothesis #3	58
3.9.4 Research hypothesis #4	58
4. Chapter Four: Study Findings and Results	59
4.1 Description of the sample	59
4.1.1 Participant involvement in the study	59
4.1.2 Demographics of the participants	60
4.1.3 Description of the study variables	61
4.2 Internal consistency of the VSH scale	62
4.3 Hypothesis #1	63
4.4 Hypothesis #2	64

	<u>Page</u>
4.5 Hypothesis #3	65
4.6 Hypothesis #4	65
5.0 Chapter Five: Discussion	67
5.1 Comparison of the subjective measures	67
5.2 The use of HR as an indicator of sleep quality and quantity	69
5.3 Sleep patterns of institutionalized elderly	74
5.4 Limitations of the study	75
5.5 Implications for future research	76
5.6 Clinical implications	77
6.0 References	78
7.0 Appendix A: Interactions of Brain Structures in a Homeostatic Model of Sleep Control	90
8.0 Appendix B: Ranking of Functional Dominance of Brain Structures in a Homeostatic Model of sleep Control	91
9.0 Appendix C: Stanford Sleepiness Scale	92
10.0 Appendix D: VSH Sleep Scale	93
11.0 Appendix E: Sleep Diary	95
12.0 Appendix F: Demographic Information Form	97
13.0 Appendix G: Consent Form	98
14.0 Appendix H: Frequencies of Medical Diagnoses Appearing in the Health Record	100
15.0 Appendix I: Frequencies of Regular and PRN Medications Recorded in the Health Record	101
16.0 Appendix J: Record of Instrument Artifacts removed for the HR Data	102
17.0 Appendix K: Heart Rate Profiles for each Study Participant (ID#1-23)	105

## Index of Tables

	<u>Page</u>
Table 1: Use of Sleep Medications during the Study Night	128
Table 2: Summary of the Study's Nighttime Variables	129
Table 3: Summary of the VSH Sub-scores	130
Table 4: Correlation Matrix for Sleep Diary, Sleep Scales and HR Variables	131
Table 5: Multiple Regression Model Results for VSH Score versus Total Sleep Time, Wakenings after Sleep Onset	132
Table 6: Multiple Regression Model Results for VSH Score versus Total Sleep Time, Wakenings after Sleep Onset, Interaction Term	133
Table 7: Multiple Regression Model Results for SSS Score versus Total Sleep Time, Wakenings after Sleep Onset, Interaction Term	134
Table 8: Multiple Regression Model Results for Total Sleep Time versus Corrected Mean1, SD1, Interaction Term	135
Table 9: Multiple Regression Model Results for Total Sleep Time versus Corrected Mean2, SD2, Interaction Term	136
Table 10: Multiple Regression Model Results for Wakening after Sleep Onset versus Corrected Mean1, SD1	137
Table 11: Multiple Regression Model Results for Wakenings after Sleep Onset versus Corrected Mean2, SD2	138
Table 12: Multiple Regression Model Results for VSH versus Corrected Mean1, SD1, Interaction Term	139
Table 13: Multiple Regression Model Results for VSH versus Corrected Mean2, SD2, Interaction Term	140
Table 14: Multiple Regression Model Results for SSS versus Corrected Mean1, SD1, Interaction Term	141
Table 15: Multiple Regression Model Results for SSS versus Corrected Mean2, SD2, Interaction Term	142



	<u>Page</u>
Table 16: Summary of Comparison between Sleep Diaries and HR Profiles for Sleep Onset, Sleep Termination and Wakenings after Sleep Onset	143
Table 17: Comparison of Mean HR Values from the Sleep Diary and HR Profiles to the Conceptual Framework Predicting Decreases in HR from Baseline→Sleep Latency→Sleep Onset	146

Index of Figures	<u>Page</u>
Figure 1: Conceptual model of the percent decrease in HR values from evening baseline to sleep onset.	147
Figure 2: Hypothetical sleep scenarios as indicated by the four-quadrant graph.	148
Figure 3: Scatter plot Total Sleep Time by VSH score.	149
Figure 4: Scatter plot Total Sleep Time by SSS score	150
Figure 5: Scatter plot Wakenings after Sleep Onset by VSH score	151
Figure 6: Scatter plot Wakenings after Sleep Onset by SSS score	152
Figure 7: Scatter plot Wakenings after Sleep Onset by SD1	153
Figure 8: Scatter plot Wakenings after Sleep Onset by SD2	154
Figure 9: Four-quadrant graph: Corrected Mean1 by SD1	155
Figure 10: Four-quadrant graph: Corrected Mean2 by SD2	156

## Comparison of Subjective Reports to Heart Rate

### During Sleep in Older Institutionalized Women

#### Chapter One: Statement of the Problem

Sleep behavior is a phenomenon that is universal to all humans and occupies a significant proportion of our time, as we spend approximately one third of our lives asleep. Sleep is believed to be a basic human need to promote physical and mental health and assist in the healing process (Adam & Oswald, 1983; 1984; Hodgson, 1991; Webb, 1988). As the aging process proceeds, a number of physiological changes occur that can impair the ability to obtain adequate sleep. This results in decreased quality and quantity of sleep in older adults (Ancoli-Israel, 1997; Dement, Miles & Carskadon, 1982). For elderly persons living within an institutionalized setting, environmental factors may also compound the problem of sleep disruption (Gentili, Weiner, Kuchibhatla & Edinger, 1997).

Many studies have been conducted to describe the physiological changes and resultant effects on sleep patterns in older adults. A crucial consideration in the study of sleep in the elderly person is the method of measurement used to gain a true reflection of actual sleep quality and quantity. The subjective and objective measures of sleep that have been employed in the literature are often not appropriate or reliable for use in the institutionalized elderly population and may actually reflect inadequate representations of sleep patterns. For example, the electroencephalogram (EEG), which is a widely utilized sleep measurement tool in clinical and research domains, is problematic when used with an elderly population (Jacobs, Ancoli-Israel, Parker & Kripke, 1989; Regestein & Morris, 1987). The standardized scoring methods that have been developed to define sleep stages

are based upon a young, healthy adult population but when applied to older individuals, they may result in an underestimation of sleep due to the physical changes that accompany normal aging (Webb, 1989). Secondly, EEG scoring techniques do not account for brief arousals less than 20 to 30 seconds that commonly disrupt the sleep of elderly individuals. Therefore, methodologies employing this type of sleep measurement within an elderly population may not be representing an accurate reflection of sleep patterns (Dement et al., 1982; Pressman & Fry, 1988; Schnelle, Cruise, Alessi, Al-Samarrai & Ouslander, 1998).

The relationship between heart rate (HR) fluctuations and depth of sleep has been well documented in the literature (Baust & Bohnert, 1969; Bonnet & Arand, 1997; Flemons, Horne, Guilleminault & Gillis, 1994; Harper, Frysinger, Zhang, Trelease & Terreberry, 1988). Studies that have examined the effects of heart rate during sleep have also employed concurrent polysomnographic measures to determine HR changes during the varied stages of sleep. There is only one known published study that has utilized HR data alone to determine the depth of sleep by stage classification (Welch & Richardson, 1973). These researchers concluded that it was possible to derive a fairly accurate representation of sleep patterns from HR data within study participants who exhibited stable sleep patterns. However, no known study has taken the next step and utilized HR monitoring independent of any other physiological measure to collect objective data and provide a comparison with subjective information reported by the individual person pertaining to both the quality and quantity of sleep.

The rationale for why this research question has not been addressed can only be postulated. The majority of literature pertaining to sleep behavior tends to focus on

younger, healthy individuals and most attention has been directed towards the measurement and description of sleep by use of EEG and polysomnography. Studies relating to sleep patterns in elderly have primarily utilized sleep surveys and questionnaires to describe the existence of sleep disturbance in this population. Perhaps the potential to utilize HR monitoring as a measure of sleep quality and quantity in the elderly has been overshadowed by the recent attention directed towards the use of wrist actigraphs to measure activity during sleep as an indicator of sleep-wake patterns.

The purpose of this study was to compare subjective reports of the quality and quantity of sleep during older institutionalized individuals' overnight sleep period to HR values and profiles obtained during the same timeframe. This data was examined to determine if any relationships and similarities existed between these types of measurements. This study employed the use of sleep diaries and sleep quality measurement scales as subjective indicators of the quantity and quality of sleep respectively, and portable heart rate monitors to record objective HR data during sleep. The research questions that guided this study were;

1. Is there a significant relationship between sleep diary records and subjective reports of sleep quality?
2. Is there a significant relationship between sleep diary records and overall measures of HR during the sleep period?
3. Is there a significant relationship between subjective reports of sleep quality and overall measures of HR during the sleep period?
4. Is there a relationship between sleep diary records and patterns of HR fluctuations during sleep?

## Chapter Two: Literature Review

Despite the general consensus among nurses and sleep researchers that promotion of optimal sleep patterns and prevention of sleep disruption in elderly and institutionalized individuals should hold top priority for research initiatives, much of the current body of literature has focused on the physiological structure, function and control of sleep (Dement et al., 1982; Spenceley, 1993). Most bodies of literature, including nursing, contain relatively few studies concerning the individual sleep experience and promotion of sleep in compromised populations like the institutionalized elderly. One possible reason for this void may be the methodological difficulties of studying sleep behaviors in this setting such as the use of physiological sleep monitoring equipment and the existence of appropriate tools to measure sleep quality and quantity in the elderly (Jacobs et al., 1989; Regestein & Morris, 1987).

This chapter provides a framework for the potential of utilizing HR monitoring as a means of sleep measurement by reviewing the current literature pertaining to normal sleep physiology, age-related changes in sleep and mechanisms that control HR during sleep and sleep measurement issues. A conceptual model based on this scientific literature is proposed to guide data analysis.

### Normal Human Sleep

A simple behavioral definition of sleep, according to Carskadon and Dement (1994), is “a reversible behavioral state of perceptual disengagement from, and unresponsiveness to the environment” (p. 16). This definition considers sleep as a discrete, separate state from wakefulness. Earlier sleep research emphasized this dichotomous paradigm that considered sleep a passive process and an inactive state of the

brain. With the first use of electroencephalograms (EEGs) to study sleep behaviors beginning in the late 1920's, it was discovered that the brain was not in a passive state of activity but instead, displayed unique waveforms in comparison to wakefulness (Dement, 1994). Therefore, it may be more accurate to define sleep as a phase of a continuous cycle or circadian rhythm that is characterized by EEG changes and other physiological measures (Hodgson, 1991).

Although the research concerning sleep definitions and structure outlines a fairly predictable pattern of sleep cycling, the term 'normal' is used to refer to sleep patterns of young, healthy adults. There is a great degree of variability in sleep behaviors even amongst this population. It is common to expect greater variations in sleep patterns in older individuals and people with sleep disorders or chronic illness (Bliwise, 1994; Carskadon & Dement, 1994; Dement et al., 1982).

There are distinct types of sleep stages or phases, which cycle throughout a period of sleep. This finding was first documented in experiments in the 1930's that utilized EEG recordings to study sleep (Borbely, 1994). Since these early experiments, scientific measurement in the area of sleep architecture has been expanded with the addition of other physiological monitoring including the electro-oculogram (EOG), the electromyogram (EMG), the electrocardiogram (ECG) and respiratory measures used concurrently with the EEG (Sanford, 1983). This monitoring procedure is known as polysomnography and is considered the gold standard of objective sleep measurement.

With the addition of these monitoring techniques, researchers were able to determine that sleep consisted of two distinct types of activity that were as different from each other as they were from wakefulness. Non-rapid eye movement (non-REM) sleep is

the first type and is further divided into four stages (1 to 4) which are defined by EEG activity (Carskadon & Dement, 1994). The EEG pattern in non-REM sleep is defined as synchronous which is represented by low frequency, high amplitude activity (Turpin, 1986). Of the 4 stages of non-REM sleep, stage 1 displays EEG measures closest to waking and is described as light, transitional sleep. Stage 2 non-REM is characterized by a slower EEG wave frequency than stage 1 with 'sleep spindle' and 'K-complex' waveforms that are unique to this stage. Stages 3 and 4 are associated with very large, slow frequency delta waves that dominate the EEG. These two stages are often collectively referred to as slow wave sleep (SWS), as the only method to distinguish the two from an EEG recording is by the percentage of delta waves that are present. As non-REM sleep progresses through the stages, the EOG reflects gradual slowing of eye movements and the EMG demonstrates a decline in muscle tension. Non-REM sleep has also been referred to as S-sleep (Sanford, 1983; Turpin, 1986).

The second type is rapid eye movement (REM) sleep. In contrast to the slowing of polysomnographic measures in non-REM sleep, erratic EEG activation, complete loss of muscle tone (except the oculomotor and diaphragmatic muscles), bursts of rapid eye movements, accelerations of cardiorespiratory functions and dreaming characterize REM sleep (Culebras, 1992b). REM waveform activity is generally referred to as desynchronous and closely resembles that of the waking state. This type of sleep is also called D-sleep (Sanford, 1983; Turpin, 1986).

The normal progression of sleep through these stages follows an ideal pattern and repeats a number of times throughout a sleep period. An individual enters sleep through stage 1 non-REM that lasts between 1 and 7 minutes and then progresses through stages



2, 3 and 4. In the first sleep cycle, the time spent in stage 3 is only a few minutes and the time in stage 4 is approximately 20 to 40 minutes. There is then a reverse ascent through the lighter stages of non-REM sleep where a few minutes are spent in stage 3 followed by 5-10 minutes in stage 2. This precedes entry into the first REM period of the night which is quite brief, lasting only between 1 to 5 minutes. This cycle repeats itself 4 to 6 times throughout the night by entering and exiting REM sleep via stage 2 non-REM (Carskadon & Dement, 1994).

As a rule, REM periods lengthen across the night while stages 3 and 4 occupy less time and may disappear by the later cycles replaced by stage 2 sleep. Young adults typically receive the majority of their REM sleep in the last third of the sleep period and most of the SWS in the first third of the night. The average total length of the sleep cycle is between 90 to 120 minutes with the first cycle being slightly shorter and the latter cycles slightly longer. Non-REM sleep comprises 75 to 80 percent of total sleep while the remaining 20 to 25 percent is REM sleep (Carskadon & Dement, 1994). Some researchers have expanded on this model of sleep architecture to further describe sleep as core or optional. As the first part of the sleep period contains the majority of the deeper SWS (stages 3 and 4), this is determined as the essential core of sleep. The remainder of the night which contains lighter stages of sleep (REM and stage 2), is considered optional sleep. The hypothesis implies that core sleep is necessary for optimal daytime function while optional sleep is flexible and increases or decreases can be tolerated without any undue effect on the body's functioning (Horne, 1983; Wauquier & van Sweden, 1992).

A few research studies have focused on the physiology of sleep structure during napping that occurs throughout the day. There appears to be a difference in the type of

sleep that is evident in a nap and is dependent upon the time of day. Naps that are taken in the morning seem to perpetuate the patterning of the later cycles of sleep as they tend to be characterized by Stage 1 and REM sleep (Knowles, Coulter, Wahnnon, Reitz & MacLean, 1990; Reynolds et al., 1991). Conversely, afternoon naps contain mostly slow wave and non-REM sleep which reflect an early start to the usual nightly sleep pattern (Aber & Webb, 1986; Knowles et al., 1990).

#### Normal Age-Related Changes in Sleep

A common myth is that as one becomes older, the body naturally requires less sleep. In fact, it is not the need for sleep but the ability to sleep that declines with increasing age (Ancoli-Israel, 1997). Many large cross-sectional surveys of elderly persons living within the community have supported this claim. A study of 600 community-dwelling elderly in Finland found that 12.5% subjectively rated the quality of their sleep as poor (Raiha, Seppala, Impivaara, Hyypa, Knuts & Sourander, 1994). In a longitudinal study by Livingston, Blizard and Mann (1993) that interviewed a population of elderly individuals twice in two years, subjective sleep disturbances were found to be quite common with 33% and 43% of the total sample reporting positive findings respectively. Some surveys have shown up to 75% of elderly respondents to be dissatisfied with either the quality or quantity of their sleep (Pressman & Fry, 1988).

There are number of physiological age-related changes that are reflected in the sleep architecture. In general terms, the amplitude of the EEG waveforms in older adults is significantly reduced compared to a young adult while the frequency of the waves remains similar (Dement et al., 1982; Feinsilver & Hertz, 1993). This is largely seen in the delta waves that define SWS in stages 3 and 4 non-REM. The large 'sleep spindles'

that characterize stage 2 sleep appear less frequently and are often poorly formed when compared to younger counterparts (Prinz & Vitiello, 1993). It is thought that the aging process causes some loss of neurons, synaptic connections and a reduction of cerebral blood flow. These changes to the brain may cause an electrophysiological state that results in this reduction of EEG waveform amplitude (Pressman & Fry, 1988).

Using standard EEG scoring techniques, the elderly tend to spend increased time in stage 1 sleep (8 to 15% of total sleep time) with higher occurrences of shifts into this stage. This finding in itself is suggestive of the high prevalence of sleep disturbances in the elderly (Bliwise, 1994; Dement et al., 1982). Stage 2 sleep appears to remain the same but there is an absolute and relative reduction of the time spent in stages 3 and 4 (Dement et al, 1982; Hoch, Reynolds & Houck, 1988). By age 60, SWS may no longer be present in EEG sleep recordings (Carskadon & Dement, 1994). As these findings seem quite severe, Webb (1989) advises caution as the usual EEG scoring techniques may not accurately reflect the true sleep status of an elderly person due to the natural decrease in wave amplitude that accompanies aging. When EEG samples are scored using the frequency criterion and disregarding the amplitude, little or no reduction in stages 3 and 4 sleep are seen.

The total amount of REM sleep in the elderly tends to remain stable until extreme old age where there is some decline. There is evidence that the distribution of REM sleep changes throughout the night with the first REM period appearing earlier and lasting longer. There is also a shift of the REM sleep to the earlier portion of the night, which is a reversal from young adults (Bliwise, 1994; Dement et al., 1982, Pressman & Fry, 1988).

This shifting of REM sleep may partially be explained by the changes in the circadian rhythm that are attributed to aging. One difference that occurs in circadian timing is the advanced sleep phase syndrome. It is suggested that as we age, our circadian clock advances approximately one hour per decade after the age of 60 (Ancoli-Israel, 1997; Swift & Shapiro, 1993). Wauquier and van Sweden (1992) believe that the appearance of higher amounts of REM sleep in the first sleep cycle and early in the night are a result of this circadian shift. They suggest that REM sleep comprises more of the core portion of sleep rather than the optional as it may serve an important role of cognitive functioning in aging.

Other researchers suggest that there is a decline in the amplitude of the circadian rhythm as a result of an age-related decrease in size of the suprachiasmatic nucleus (SCN) (Atkinson, Witte, Nold, Sasse & Lemmer, 1994; Bliwise, 1994). The SCN is situated in the hypothalamus and contains the circadian pacemaker that controls the internal timing of the rhythm by environment light cues. The SCN receives information regarding light changes by means of pathways to the retina. It then interacts with the pineal gland to stimulate the secretion of melatonin that acts to stabilize the circadian function. Haimov and colleagues (1994) have demonstrated that peak levels of melatonin were reduced by 49% in elderly persons when compared to younger adults. Their data suggests a relationship between decreased circadian functioning and sleep disturbances in the elderly. Some have described the change as moving from a biphasic sleep-wake pattern of a younger adult to a polyphasic cycle similar to infants (Prinz & Vitiello, 1993).

### Age-related Sleep Disturbances

Age-related sleep disturbances can be distinguished from sleep disorders and are considered to be persistent, mild and irreversible alterations in sleep that usually do not produce severe daytime sleepiness (Beck-Little & Weinrich, 1998). This section reviews the specific elderly sleep disturbances that are reported in the literature with a brief description of some factors that influence sleep in this population.

The age related changes in sleep architecture discussed above combine with other social, pharmacological and environment factors that produce common behavioral sleep disturbances. Social factors may include a decreased level of activity due to physical limitations or retirement status. Webb and Aber (1984) did find that the removal of work schedules affected the sleep-wake pattern by an increase of the amount of total daily sleep by 1 to 1 ½ hours but did not show an increase in the tendency to nap. Pharmacological factors may play a large role in affecting sleep behaviors in the elderly. Hypnotic/sedative sleep medication use increases with age and is very common in institutional settings (Bliwise, 1994; Seppala, Rajala & Sourander, 1993; Wooten, 1992). While sleep aid medications act to decrease the time required to fall asleep (Bachman, 1992), they can produce adverse effects that are detrimental to healthy sleep. They suppress SWS (stages 3 and 4) and REM sleep, cause residual daytime somnolence and resultant rebound insomnia when discontinued (Maczaj, 1993; Prinz, Vitiello, Raskind & Thorpy, 1990; Wooten, 1992). In a study that examined sleep disturbances in elderly, the authors reported that complaints of habitual difficulties with maintaining sleep at night and frequent early morning awakenings were more common amongst sleeping pill users, indicating that medications intended to aid sleep may have the reverse effect (Gislason,

Reynisdottir, Kristbjarnarson & Benediktsdottir, 1993). Environmental factors that may also contribute to sleep disturbances in the institutionalized elderly in hospitals and long term care are noise and light levels at night and staff care routines (Gentili et al., 1997).

There are a number of sleep parameters that are used in the literature to describe age-related changes in sleep. Perhaps the most significant reported sleep disturbance that occurs with aging is the number of nighttime awakenings after sleep onset (WASO) (Boselli, Parrino, Smerieri, & Terzano, 1998; Buysse, Brownman, Monk, Reynolds, Fasiczka & Kupfer, 1992; Gislason et al., 1993). Hayter (1983) reported that the frequency of WASO increased significantly after the age of 75. These awakenings can be very distressing as they interrupt sleep frequently during a night and can last for an extended period of time (Prinz & Vitiello, 1993). One factor that is proposed to contribute to the difficulty maintaining sleep is a lowering of the auditory awakening threshold in older individuals compared to younger adults (Bliwise, 1994; Prinz & Vitiello, 1993). As shown above, elderly adults also tend to spend more time in lighter stages of sleep where arousals are more likely to occur.

Although the need for sleep does not decrease with age, research indicates that total sleep time (TST) does decline slightly. When TST is considered only over the nighttime sleeping period, it is markedly decreased in comparison with younger adults (Feinsilver & Hertz, 1993, Pressman & Fry, 1988). However, when TST is calculated over a 24-hour period that includes napping, it is very similar or slightly higher for older adults (Hayter, 1983; Pressman & Fry, 1988). It is evident in studies involving community dwelling and institutionalized elderly that older individuals spend significantly more time in bed (TIB) to achieve their needed level of sleep (Ancoli-Israel,

Parker, Sinaee, Fell & Kripke, 1989; Hayter, 1983). This can also be described as a general decrease in sleep efficiency (SE) that is seen with increasing age. SE is the ratio of time asleep to spent in bed (Feinsilver & Hertz, 1993).

Sleep latency (SL) is another common sleep parameter that is discussed in relation to aging and refers to the time from 'lights out' to the onset of sleep (Pressman & Fry, 1988). Some authors suggest that SL only increases slightly with age (Bliwise, 1994; Feinsilver & Hertz, 1993; Pressman & Fry, 1988). Gislason et al. (1993) noted that 9.6% of their 200 elderly respondents reported habitual increased SL, while 14.4% reported difficulty initiating sleep occasionally. Fifty four percent of elderly respondents in another large survey study indicated that they experienced some SL with 23.6% experiencing this frequently (Frisoni, de Leo, Rozzini, Bernadrini, Dello Buono & Trabucchi, 1992). This increase is reported to be more evident in the seventh and eighth decade (Frisoni, et al., 1992; Gislason, et al., 1993; Hayter, 1983).

Due to the advanced phase circadian shift discussed above, there is a reported alteration in bedtime and time of wakening in the morning. Buysse et al. (1992) compared the 24-hour sleep-wake patterns of elderly and younger subjects and found that older individuals went to bed approximately 40 minutes earlier than younger adults and woke up an average of 76 minutes earlier. Early morning awakening (EMA) was also reported by Gislason et al. (1993) with a prevalence of 16.7% experiencing habitual problems with an average waking time of one hour earlier than their elderly counterparts who did not report this difficulty.

A common belief relating to sleep changes with aging is the increased occurrence of daytime naps. The relationship between night sleep and napping behavior could

influence each other in a number of ways. A decreased homeostatic ability to promote adequate sleep at night coupled with a decline in the circadian rhythm amplitude could very well lead to an increased tendency to sleep less at night and nap during the day (Buysse et al., 1992). A decreased quality and quantity of night sleep should result in increased sleepiness the next day and frequent naps. Studies have indicated that napping behaviors increase with age and are greater in older adults when compared to young adults (Hayter, 1983; Reynolds Jennings, Hoch, Monk, Berman, Hall, Matzzie, Buysse & Kupfer, 1991; Roth, Roehrs, Carskadon & Dement, 1994). The relationship between daytime napping and excessive sleepiness during the day is less clear. Point prevalence of excessive daytime sleepiness of elderly persons in a longitudinal survey conducted by Hays, Blazer and Foley (1996) was reported to be quite significant at a rate of 25.2%. In their study, a subject was considered to be experiencing excessive daytime sleepiness if they reported feeling so sleepy during most of the day or evening that they had to take a nap. However, Reynolds and colleagues (1991) compared daytime sleepiness between young adults and healthy 'old-old' elderly using Multiple Sleep Latency Tests (MSLT) and their results suggested that excessive sleepiness might not be a concern in the elderly. They found that young adults were objectively sleepier at points throughout the day than the older individuals. They suggest that the decrease of SWS and increased awakenings at night may not place elderly at risk for excessive sleepiness and that maintenance of a sleep efficiency of about 80% or higher and total sleep times of 6 hours seems to preserve daytime alertness in this group.



### Mechanisms of Sleep Control

Recent research proposes that certain physiological and rhythmic mechanisms interact to control the complex states of sleep and wakefulness. The mechanisms have been identified as neurological processes, homeostatic systems and circadian rhythms. As HR fluctuations during sleep are directly linked to neuronal and homeostatic functioning, theories related to these two processes are discussed in depth.

Through research that has been conducted during the past seven decades, a number of neuronal structures and neurochemicals have been linked to the complex control of sleep-wake states. In the neurological realm, sleep and wakefulness are antagonists that compete for dominant brain activity. Neuronal brain structures that control the sleep-wake cycle are found in the brainstem, the hypothalamus and the basal forebrain with relay nuclei located in the thalamus and cerebral cortex. Sleep-wake impulses are conducted through these deep brain structures by means of a common pathway termed the reticular activating system (RAS).

Wakefulness is maintained through tonic activity in the RAS and environmental inputs of sensory stimuli such as pain and noise. Reticular system activation ascends from the brainstem to the cortex via two routes. The dorsal route travels from the medulla in the brainstem through the midbrain to terminate in the thalamic relay center. From here, the signal is transmitted to the cerebral cortex in a widespread manner. The ventral path also travels through the midbrain but passes into the hypothalamus and then onto the basal forebrain. The impulse is then transmitted to the cerebral cortex through relays in this area. Impairment of either the hypothalamus or basal forebrain results in the inability to maintain wakefulness, indicating the existence of an important waking center

in these areas. Although many neurochemicals may play a role in wakefulness, sympathetic neurons of the RAS containing catecholamine and acetylcholine neurotransmitters play the largest role in maintaining the waking state (Culebras, 1992a; Jones, 1994).

Sleep is induced as certain types of sensory input are lessened and sleep-promoting neurons along the RAS concentrated in the lower brainstem, anterior hypothalamus, preoptic area and basal forebrain become active. Serotonin-containing neurons along the RAS play a crucial role in sleep promotion as they induce cortical slow wave activity by dampening the activity of the RAS waking neurons and blocking afferent information to the cortex. These neurons are thought to have extensive overlap with parasympathetic autonomic regulatory systems (Culebras, 1992a; Jones, 1994).

Although many questions about REM sleep remain unanswered, much has been learned in the past 30 years about the neural mechanisms that generate REM sleep. One hypothesis that has been proposed is that each REM phenomenon is thought to be controlled by a defined center in the brainstem (Culebras, 1992a). To date, researchers have not found evidence of an 'executive' group of neurons. It is known that there is a system of neurons residing in the lateral pontine region of the brainstem that are responsible for the initiation and maintenance of REM sleep. Neural mechanisms that control other characteristics of REM sleep such as muscle atonia and rapid eye movements, are not well understood and add to the mysteriousness of this sleep state (Siegel, 1994).

An important theoretical consideration to explore is the link between sleep-wake states and the autonomic nervous system (ANS). The ANS operates by the principle of

homeostasis, which has been used to define many physiological mechanisms from temperature regulation to fluid and electrolyte balance. Homeostasis is the physiological balance that is maintained by continuous adjustments in autonomic effectors to meet any changes that the organism experiences in the environment. When considering the autonomic changes that occur in the regulation of the sleep-wake process, the principles of homeostasis define this activity as well (Parmeggiani, 1988).

When examining the specific neurological mechanisms that control the sleep-wake process, the homeostatic influence of the autonomic system becomes clearer. The autonomic innervation of organs and body systems is divided into sympathetic or parasympathetic functions. Activation of sympathetic neural fibers and neurotransmitters result in a stimulated, active body state or process, while the parasympathetic system provides a reverse effect that slows or inhibits physiological functions. The constant adjustments between these two systems work to provide an overall state of homeostasis. As discussed above, waking states are accompanied by activation of neural fibers and centers that have a sympathetic effect and sleep is controlled by neurons that hold a parasympathetic purpose. In a broad sense, homeostatic mechanisms act to counterbalance deviations from an average reference point of sleep. As shown in sleep deprivation studies, these mechanisms react to replace or augment SWS when decreased or absent. When sleep is in excess, they respond by reducing the amount of sleep (Borbely, 1994).

Much research has also been directed towards the autonomic changes that occur between the different stages of sleep, which indicates a difference in parasympathetic and sympathetic activity in non-REM and REM states. Earlier research that examined

hemodynamic changes during sleep suggested that the reduction of mean arterial blood pressure, heart rate and cardiac output during sleep was a result of decreased sympathetic activity. Any phasic increases in these physiologic measures during sleep were initially believed to be caused by inhibition of the parasympathetic system (Baust & Bohnert, 1969; Khatri & Freis, 1967). Recent studies employing pharmaceutical and spectral analysis techniques to identify sympathetic and parasympathetic activity have suggested a slightly different view. These studies demonstrated that there is progressively greater parasympathetic activity as one moves through stages 1 to 4 non-REM sleep and less parasympathetic influence in the erratic state of REM sleep. Other than its decrease from wakefulness to stage 1 sleep, sympathetic activity remains relatively constant (Bonnet & Arand, 1997; Zemaityte, Varoneckas & Sokolov, 1984). This latter research supports the concept of serotonin neurons suppressing the activity of the neurons responsible for maintaining wakefulness in the RAS, as described above.

The complex relationship between the neuronal structures of the brain, the activity of the autonomic nervous system and the concept of homeostasis in the sleep-wake process can be summarized in a conceptual model proposed by Parmeggiani (1988; 1994). In this model, the behavioral states of wake/non-REM sleep/REM sleep are an expression of the functional dominance of three structurally distinct areas of the brain: the telencephalon (forebrain and cerebral cortex), the diencephalon (midbrain including the hypothalamus and thalamus) and the rhombencephalon (brainstem). Three ranks of functional dominance are attached in the order of telencephalon (T) > diencephalon (D) > rhombencephalon (R). The transition between sleep and wakefulness and REM and non-REM sleep is characterized by a shift in functional dominance between these structures

(Appendix A). This model also stresses the critical role of the diencephalic structures' effect, specifically the hypothalamus, on the brainstem mechanisms that underlie the dichotomy between non-REM and REM sleep. During wakefulness, homeostatic control of the hypothalamus on the brainstem is affected by telencephalon dominance ( $T > D > R$ ). During non-REM sleep, the stability of the autonomic functions (i.e. prevalence of the parasympathetic effect) is at a maximum indicating the loss of telencephalic dominance ( $D > R > T$ ). The impaired homeostatic regulation in REM sleep is believed to be a result of a loss of dominance in the hypothalamus (diencephalon) which gives way to rhombencephalon influence ( $R > T > D$ ). This action releases brainstem mechanisms that cause a great deal of instability in homeostatic functioning. These shifts do not occur randomly but in a specific pattern (Appendix B). Out of six possible combinations, only three are expressed in the sleep-wake cycle. This limiting criterion is attributed to the stability of the D-R hierarchy in the regulation of physiological functioning. The only D-R split that is observed is during REM sleep where homeostatic instability is present. This further supports the concept that this hierarchical relationship is needed to maintain physiological balance.

#### Sleep Measurement Issues

According to Webb (1989), the three dimensions of sleep to be measured in research are sleep patterns, sleep structure and evaluative responses pertaining to sleep. Measurement of sleep patterns account for the amount and timing of sleep within a 24-hour period and include total sleep time, length and placement of sleep, sleep latencies and continuity of sleep. Sleep structure concerns the physiological measurements of sleep stages. Personal statements that reflect the quality of sleep and feelings of

sleepiness define the evaluation of sleep. There are numerous techniques that have been used in research studies to measure these dimensions of sleep and can be classified as either objective or subjective. These methods will be discussed in detail below with information pertaining to their use and limitations.

#### Objective measures of sleep.

Objective measures of the physiological changes associated with sleep contribute data in regards to the pattern and structure of sleep (Webb, 1989). It is generally regarded by sleep researchers that polysomnographic techniques, especially the EEG, provide the most valued and accurate descriptions of sleep behavior and view this as the gold standard in sleep measurement. Through these measures, the various stages of non-REM and REM sleep can be determined, as well as the shifts into and time spent in each stage, sleep latencies, total sleep time, wakefulness after sleep onset and termination of sleep to within 20 to 30 seconds (Closs, 1988).

Although quantitatively precise, this type of sleep measurement presents some difficulties, especially in regards to its use in the elderly population. Perhaps a fundamental issue regarding the EEG's ability to validly describe sleep is the basic scoring rules that have been repeatedly used by sleep researchers. To date, standardized scoring methods of the EEGs of older persons have not been developed. Secondly, although the scoring rules are widely documented and employed, there continues to be dissention and debate as to the polysomnographic definition of sleep onset. These differences in scoring may affect the reliability of this technique and also create difficulty comparing published results. A third issue concerns the actual scoring of the sleep stages. EEG tracings of brain activity during sleep are examined in 20 to 30 seconds

epochs or intervals. Each epoch is scored either by a trained technician or computer program as stage 1, 2, 3 or 4 non-REM, REM or wakefulness based upon the majority of the sleep stage occupying that time. For example, if 49% of the EEG demonstrated REM sleep and 51% stage 2, the epoch would be scored as stage 2. Other events, such as brief arousals occurring for less than 10 to 15 seconds (depending upon the EEG recording speed) are disregarded and sleep studies usually report large changes in sleep stages that overlook possibly significant details. It is noted that brief 2 to 15 second arousals and numerous shifts from deep sleep to stage 1 sleep frequently interrupt the sleep of older adults. Therefore, studies utilizing this type of measurement technique do not reflect a true picture of how these transient arousals are affecting the sleep of the elderly (Dement et al., 1982; Pressman & Fry, 1988; Schnelle et al., 1998).

Polysomnographic testing can also be very involved and employs the use of a number of electrodes, transducers, probes and wire leads to collect the physiological data. Individuals who are too ill or confused may not be able to tolerate the rigors of this monitoring (Jacobs et al., 1989; Regestein & Morris, 1987). Many sleep studies of this nature also occur in a sleep laboratory setting in order to facilitate access to this equipment. Sleep labs can pose a number of problems to researchers and the validity of their data. The use of a sleep lab is an inefficient method to study the sleep behaviors of many subjects resulting in studies of small sample sizes. Secondly, it involves removing a person from their usual environment and timing of the sleep-wake cycle that can provoke anxiety or result in data that is non-representative to actual sleep behavior. Although the more recent development and use of portable polysomnography equipment has allowed for this type of data to be gathered in a person's own environment, the

expense of this equipment may still be a barrier to its frequent use (Closs, 1988; Turpin, 1986).

A second type of objective measurement is observation, where trained observers systematically rate whether a person is asleep or awake at regular intervals. These intervals usually range from one hour (Regestein & Morris, 1987) to 30 minutes (Fisher & McIsaac, 1995; O'Rourke & Burke, 2000). This technique is frequently used when the investigators have ready access to the participants, such as in a hospital or long term care setting or the monitoring is completed by a spouse or family member at home (Turpin, 1986). Again, concerns are presented with this type of monitoring. It may be difficult for the observer to determine if an individual is truly asleep or only resting without rousing them. Also, intermittent observations do not allow for accurate generalizations of the entire time period, as the individual may wake up between observations. Both of these issues may result in an overestimation of sleep time (Closs, 1988; Jacobs et al., 1989; Regestein & Morris, 1987).

A final method of objective sleep data collection is by means of measuring activity during sleep. By placing a wristwatch actigraph on a person's arm and a recording unit on a belt around their waist or by the bedside, this technique monitors wrist activity and provides a dichotomous distinction between sleep and wake dependent upon the level of activity. This method has been found to be comparable to EEG recordings in determining sleep-wake states in healthy young and old adults and has been used in a number of recent studies (Ancoli-Israel et al., 1989; Cruise, Schnelle, Alessi, Simmons & Ouslander, 1998; Jacobs et al., 1989; Schnelle, Alessi, Ouslander & Simmons, 1993; Schnelle, Cruise et al., 1998; Schnelle, Ouslander, Simmons, Alessi &



Gravel, 1993a; Schnelle, Ouslander, Simmons, Alessi & Gravel, 1993b). One issue that has been raised in regards to wrist activity monitoring is use of this method in infirm, elderly persons. It has been suggested that ill, elderly people can often be inactive for sustained lengths of time during wakefulness resulting in possible overestimations of sleep time (Jacobs et al., 1989).

#### Heart rate recording as an objective measure of sleep.

A relationship between sleep and HR has been documented in the literature since the 1920's (MacWilliam, 1923). Independent measures of HR show a distinctive and reliable correlation to EEG sleep-related changes and could be used for sleep state scoring (Brooks, Hoffman, Suckling, Kleyntjens, Koenig, Coleman & Treumann, 1956; Harper et al., 1988; Walsh & Richardson, 1973). There has been much research devoted to the comparison between HR fluctuations and EEG changes between physiological alterations in sleep stages. However, there is only one known published study that utilized the HR data of 8 young, healthy males independently to determine the depth of sleep by stage classification (Welch & Richardson, 1973). This section will present a review of the current literature concerning HR monitoring in sleep and explore the possibilities of its use for clinical and research purposes.

Smith (1988) has suggested that the relative ease of measuring the predictable pattern of cardiovascular responses offers the potential to use this approach in the study of the neural basis of behavior. Research into the autonomic influences during sleep demonstrates a progressive outflow of parasympathetic activity through the stages of non-REM sleep with the highest in stage 4 and less influence during the erratic state of REM sleep. Heart rate patterning during sleep also follows this rise and fall of

parasympathetic activity as the HR progressively decreases throughout the stages of non-REM sleep with lowest values in stage 4 and exhibits wide fluctuations during REM sleep (Baust & Bohnert, 1969; Bonnet & Arand, 1997; Cajochen, Pischke, Aeschbach, & Borbely, 1994; Flemons et al., 1994; Harper et al., 1988; Zemaityte et al., 1984). The specific changes in HR patterning during sleep will be examined to determine if they could provide accuracy in describing the quality and quantity of sleep in elderly persons.

An important consideration in the study of sleep is the transition between sleep-wake states and the actual time measurement of sleep initiation. This is a complicated event to measure, as there is even discrepancy in the EEG definition of sleep onset. Generally speaking, there is a tonic decrease in HR between wakefulness and non-REM sleep (Baust & Bohnert, 1969; George & Kryger, 1985). The transition that occurs between the evening period preceding bedtime, the resting state prior to sleep onset and the movement into early stages of non-REM sleep have been studied in detail. A study that examined 24-hour heart rate and blood pressure variations in adult men found a 13% decline in HR values as opposed to normal waking HR during the four hours prior to bedtime (Degaute, van de Borne, Linkowski & Van Cauter, 1991). They also found that after bedtime, a further drop of 12% occurred and felt that their results indicated a general trend of declining HR between wakefulness and sleep and not a definite time of sleep onset. However, other studies have demonstrated a reduction in HR between resting and sleep onset. Khartri & Freis (1967) reported an average HR of 66.2 beats per minute (bpm) during rest with a decrease of 5.5% when stage 1 sleep was entered. A second study that examined the HR patterns of patients with cardiovascular disease found a significant difference in mean HR between wakefulness, stage 1 and stage 2 sleep

(Richards, Curry, Lyons & Todd, 1996). The decrease between awake and stage 1 was approximately 2% with a further 1.5% reduction between stage 1 and 2. A study by Gander, Connell & Graeber (1986) that examined the sleep effects of circadian rhythms on individuals during normal daily activities and strict 24-hour bedrest, reported that HR patterns still demonstrated a nighttime decrease compared to wakefulness even when the subjects spent the entire 24-hour day in bed. This finding has also been confirmed in the study of HR differences between quiet rest and sleep in baboons (Smith, 1988).

According to this knowledge of sleep transition and HR changes, it may not be possible to pinpoint sleep onset to a precise time using this measurement. However, if this technique was used in conjunction with a self-report measure such as a sleep diary, it may be possible to provide a reasonably accurate estimation of the transition to sleep. This may be useful in clinical settings where precise measurements are not required to gain an understanding of a person's sleep patterns.

Another consideration in HR monitoring during sleep cycle changes is the ability of this technique to distinguish between periods of slow wave sleep (SWS) and REM episodes. This distinction would provide insight into the depth or quality of sleep by determining the amount of time spent in deeper SWS and lighter stages such as REM and Stage I by means of HR values. In general terms, deeper sleep states tend to exhibit decreased heart rates and show less beat-to-beat variation than REM sleep (Aldredge & Welch, 1973). As mentioned above, HR continues its decrease between stages 1 and 2 non-REM sleep as the parasympathetic responses become progressively stronger. Stage 2 sleep exhibits a higher average HR than stages 3 or 4 (Aldredge & Welch, 1973) but there is little or no difference between these two deep stages of SWS (Zemaityte et al.,

1984). Brooks et al. (1956) reported a 10% increase in HR when the depth of sleep shifted down by one stage (i.e., stage 4 to 3, 3 to 2), a 13.7% increase when a two stage lessening of sleep occurred and a 21.5% increase with a three stage change from deep sleep to a lighter stage (i.e., stage 4 to 1).

A study by Welsh and Richardson (1973) was conducted to compare the accuracy of sleep stage classification calculated independently from HR data, to EEG sleep recordings. Although the authors did not specify the age of their study population, they described their sample as 8 young, healthy males. Using an algorithm to determine the HR-classified sleep states, the authors analyzed 16 nights of HR and EEG data from 8 subjects. Their results indicated that Stage I and REM sleep were difficult to differentiate from each other by HR values alone, as well as Stages III and IV. When these two sets of sleep stages were combined, the HR-classified sleep stages were accurate 60 to 77% of the time in 9 of the 16 nights of data. Seven nights of the 16 were shown to have correlations above 0.5 ranging as high as 0.77. They concluded that it is possible to derive a fairly accurate representation of sleep patterns from HR data among subjects who do not exhibit large intra-cycle and intra-night fluctuations in HR.

High HR levels with great variability and instability in REM sleep as compared to non-REM sleep have been widely documented (Aldredge & Welch, 1973; Cajochen et al., 1994; Khatri & Freis, 1967; Smith, 1988; Snyder, Hobson, Morrison & Goldfrank, 1964; Zemaityte et al., 1984). It has been documented that there is a 6% increase in HR between stage 2 non-REM and REM stages (Snyder et al., 1964). REM sleep is usually characterized by a burst of eye movements accompanied by an increase in HR, which is followed by bradycardia for a few seconds (Baust & Bohnert, 1969). Some authors have

disputed the extent of this REM HR variability across the night sleep period. Snyder et al. reported that HR variability decreased with successive REM periods during the night while others (Bonnet & Arand, 1997; Degaute et al., 1991) presented data that indicated there is no change in the amount of HR variation during REM episodes. Due to its erratic nature, HR in REM sleep can equal or surpass HR values in the waking state (George & Kryger, 1985).

In considering the above scientific literature, the changes in HR during the different stages of the sleep wake-cycle can be illustrated by the following;

Wake /REM> Resting > Stage 1 > Stage 2 > Stage 3/4

Based on this model, individual recordings of HR during sleep could be analyzed to arrive at an objective measurement of the quality of sleep. Although George & Kryger (1985) have proposed that averaging HR values during REM sleep can be misleading due to the wide fluctuations, it may be valuable to compare periods of sleep time with increased or decreased HR variations to determine time spent in deep or light stages of sleep. This may also be accomplished by comparing peaks and valleys of HR scores to a baseline resting heart rate.

The ability for this measurement to detect mornings awakenings and arousals during the period of sleep would also be an important consideration. Degaute et al. (1991) found that heart rate began to rise prior to the natural end of a sleep period and awakening was associated with a very abrupt and large rise in HR that lasted over 30 minutes. Therefore, it appears to be easier to detect the termination of sleep as opposed to sleep onset. It has also been documented that transient arousals during sleep are associated with increased HR values to the awake baseline level (Khatri & Freis, 1976).

Short periods of wakefulness are related to an increase in sympathetic activity which may last longer than the waking event. This is evidenced by the appearance of heart rate acceleration by at least 10 beats prior to EEGs changes indicative of arousal (Bonnet & Arand, 1997). Movements during sleep that are associated with awakenings are accompanied by brief and significant increases in HR that may often rise as much as 50% of the current sleep rate and quickly return to normal (Brooks et al., 1956; Snyder et al., 1964). The frequency of these brief HR peaks could be identified on an individual sleep-HR recording and compared to an awake baseline value, a subjective report of awakenings or self-reported feelings of sleepiness/restfulness to obtain information regarding arousals during sleep. This technique may also be useful in identifying transient arousals that are often neglected in EEGs and self-reports as some HR profiling equipment can be set to record a data point every 5 seconds.

A final consideration of HR monitoring as a tool to collect objective information about sleep behavior is the variation of patterns due to age-related changes that occur with HR regulation. Elderly individuals demonstrate slightly lower HR values at night with less variability compared to younger adults (Atkinson et al., 1994; Jonsson, Lipsitz, Kelley & Koestner, 1990; Lombardi, Malliani & Pagani, 1997; Smirne, Ferini-Strambi, Montanan & Canal, 1987). This reduced cardiovascular activity has been attributed to a number of age-related physiological changes; a decreased amplitude of the circadian rhythm (Atkinson et al., 1994), stronger parasympathetic activity during sleep with a preservation of the normal level of sympathetic response (Lombardi et al., 1997; Smirne et al., 1987) and decreased vagal modulation of the HR (Lombardi et al., 1997; Richards et al., 1996). With these differences in mind, it may be easier to determine some sleep-

related events such as arousals or deviations from baseline when examining the HR profiles of elderly individuals.

If this method were to be tested for its ability to measure sleep quantity and quality, caution would be required when interpreting results. As previously mentioned, there is a great deal of individual variation in sleep patterns. This is also true of HR patterning during sleep. In fact, when different subjects are compared to each other, there often is no relationship between their average heart rate and depth of sleep. Pooling HR data from a group of individuals which is then averaged for an entire night's sleep or for a sleep stage does not indicate the HR behavior for an individual. It is suggested that HR variations during each sleep cycle be considered on an individual basis (Aldredge & Welch, 1973). By considering the above limitations and suggestions, it may be valuable to design a study that compares objective HR measures of sleep to subjective reports to see if an accurate reflection of sleep behavior can be obtained from this method.

#### Subjective measures of sleep.

Subjective evaluations involve a retrospective report about the quantity and quality of sleep experienced by the individual. This can provide information to describe the actual sleep pattern by inquiring about times of sleep onset and wakening, total time spent sleeping, including napping and amount and length of awakenings during sleep. They are also used to determine individuals' perceptions, values and beliefs about sleep (Webb, 1989). Information gained by the subjective perspective is based upon the assumption that sleep and wakefulness are intertwined and a good night's sleep is associated with feeling well rested the next day. Although subjective measures are considered the least intrusive or complicated means of obtaining information about sleep,

it is felt by some authors that they do not always correlate with objective measures of sleep measurement (Closs, 1988; Turpin, 1986).

The simplest and most efficient type of subjective measurement is the use of rating scales. Visual analogue scales (VAS) have been noted to be sensitive to changes in beliefs and attitudes about sleep and are useful for repeated assessments. Although these scales are not useful with all populations, as some people are not able to grasp the principles needed to complete a VAS, a number of researchers studying sleep have used them successfully (Closs, 1988).

More specific rating scales have been developed to measure perceived moods and feelings related to sleep. One of these tools is the Stanford Sleepiness Scale (Hoddes, Zarcone, Smythe, Phillips and Dement, 1973), which was developed to reflect subjective feelings of sleepiness and has been widely used in sleep research.

Many sleep researchers have also used sleep questionnaires to elicit information pertaining to sleep patterns. Data can be obtained either through self-administered questionnaires or interviews. Sleep questionnaires ask detailed questions to determine perceived times of sleep onset and wakening, amount of time until falling asleep, number and length of awakenings during the night and total sleeping time (Closs, 1988; Webb, 1989).

The sleep diary or sleep log is also used widely to obtain this self-reported information. Sleep diaries are day-by-day reports of sleeping and waking activities that are documented by the individual. After each night of sleep, subjects and patients are asked to accurately record the actual timing and occurrence of sleeping events. Items may include the total number of hours slept, the time of retiring to bed and waking, time



until sleep onset, early morning awakenings, amount and duration of daytime napping and other symptoms such as nighttime arousals, sleepwalking, nightmares and sleep attacks. Diaries may be kept for as short a period as overnight or over several weeks (Bachman, 1992; Haythornthwaite, Hegel & Kerns, 1991; Rogers, Caruso & Aldrich, 1993).

### Conceptual Framework

Upon review of the literature, it is observed that no established framework exists that describes the relationship between sleep quality and quantity and HR fluctuations during sleep. Therefore, the following framework was formulated on the basis of the conceptual link of HR changes during sleep to the ANS/homeostasis model and the current body of literature that has developed within this field to date.

From the literature described above, higher and lower HR values during sleep appear to be a reflection of sleep quality (Cajochen et al., 1994). Therefore, in general terms, higher overall heart rate values during sleep would indicate a poorer sleep quality and quantity and lower values would reflect enhanced sleep states. In terms of determining specific sleep parameters, the following indicators could be applied. Heart rate values decline by 13% from usual daytime to the evening period (4 hours prior to bedtime). The HR further decreases by 12% from the evening values once an individual is settled into bed and attempting to fall asleep (sleep latency). Once sleep onset occurs, the HR falls another 2-5.5% in Stage I non-REM sleep (Figure 1). As sleep progresses throughout the deeper stages of sleep, the HR continues to slow. When periods of REM sleep or nighttime arousals are encountered, the HR becomes extremely variable and can

meet or exceed normal waking values. The termination of sleep in the morning is indicated by a rise in HR to normal waking levels.

As this study is only concerned with the general differentiation between states of sleep and wake during a sleep period as compared to the determination of precise sleep stages, the fluctuations in HR between Stages II, III, IV and REM sleep are not included in this model. However, this conceptual framework allows for the comparison of overall sleep quality and depth and offers a means to estimate sleep parameters such as sleep latency (time from going to bed to sleep onset), sleep onset and sleep termination.

### Chapter Three: Research Methodology

The following section addresses the particulars of the research process. A description of the study variables is provided, followed by the research questions, hypotheses and assumptions pertaining to the direction of relationships between the study variables. Background information is provided regarding the measurement tools utilized in the study. An explanation of the sampling design and study procedure is offered, as well as ethical considerations. Finally, the data analysis plan is also discussed.

#### Operational Definitions of Variables

A number of key variables and terms were significant in the study and require operational definitions for clarity;

1. Subjective sleep data: The collective term for the self-report data obtained from the sleep diary and the participants' report of sleep quality pertaining to the previous night as indicated by the Stanford Sleepiness Scale (SSS) (Hoddes et al., 1973) and the Verran and Synder-Halpern (VSH) Sleep Scale (Synder-Halpern & Verran, 1987).

2. Objective sleep data: The collective term used to describe indicators of sleep quality and quantity by HR measurements.
3. Wakenings after sleep onset (WASO): The number and duration of wakenings during the night after initial sleep onset, as obtained from the sleep diary record.
4. Total sleep time (TST): The total time spent sleeping during a nighttime period from going to bed to rising the next morning minus WASO, as reported in the participants' sleep diary.
5. Sleep latency (SL): The time from going to bed to falling asleep, as determined from the participants' sleep diary recordings.
6. Sleep onset (SO): The participants' report of the time they actually fell asleep at the beginning of the sleep period, as obtained from the sleep diary record.
7. Sleep termination (ST): The time of last awakening in the morning prior to getting up for the day, as reported in the participants' sleep diary.

### Research Questions and Hypothesis

Based upon the above literature findings, related physiological theories and the conceptual framework derived from this information, the following research questions and hypotheses guided this study;

#### Research questions.

1. Is there a significant relationship between sleep diary records and subjective reports (SSS and VSH scales) of sleep quality?
2. Is there a significant relationship between sleep diary records and overall measures of HR during the sleep period?

3. Is there a significant relationship between subjective reports of sleep quality (SSS and VSH scales) and overall measures of HR during the sleep period?
4. Is there a relationship between sleep diary records and patterns of HR fluctuations during sleep?

Research hypotheses.

1. Sleep diary values (WASO and TST) will be related to subjective reports of sleep quality (SSS and VSH scores).
2. Sleep diary values (WASO and TST) will be related to overall measures of HR during sleep.
3. Subjective reports of sleep quality (SSS and VSH scores) will be related to overall measures of nightly HR values.
4. Sleep diary records will be related to HR patterns observed during that night's monitoring period.

A descriptive study design, employing bivariate and multiple regression statistical procedures, was utilized to examine these research questions. A descriptive design lends itself well to this relatively undocumented topic as it offers an approach to investigate the feasibility of HR profiling during sleep in this population and describe any relationships that may exist between the objective and subjective sleep measurements utilized in this study.

Assumptions pertaining to Direction of Relationships

In regards to the above research questions and hypotheses, a number of relationship assumptions require explanation for clarity. In consideration of the direction of the relationships between the sleep diary values and objective sleep rating scales, the

following would be expected. Total sleep time should have a direct relationship with the VSH score, as higher amounts of sleep should be observed with higher reports of sleep quality. However, TST should demonstrate an inverse relationship with the SSS, as higher scores reflect increased feelings of sleepiness and should be related to less amounts of reported sleep. It is predicted that WASO would have an inverse relationship with the VSH score, as more awakenings during the night should be reflected in poorer reports of sleep. Conversely, WASO should demonstrate a direct relationship with the SSS scores, as higher numbers of awakenings should coincide with higher ratings of sleepiness during the day. When considering the relationship between the two scales, it is thought that the VSH scores would have an inverse relationship with the SSS, as higher reports of sleep quality should be related to lower reports of sleepiness. In regards to the two sleep diary values, TST should also demonstrate an inverse relationship with WASO, suggesting that higher amounts of total sleep correspond to fewer awakenings during the night.

In consideration of the relationships between the sleep diary records and the HR values, the following relationships are expected. TST should reflect a direct relationship with the corrected HR mean values. This relationship direction is postulated because the larger the corrected HR mean, the larger the difference from the evening baseline HR, suggesting the individual spent more time in a sleep state. It is then expected that TST would show an inverse relationship to heart rate variability (SD), as less variability should indicate longer periods of sleep time. In consideration of the WASO variable, it is predicted that there would be an inverse relationship with the corrected HR mean suggesting that increased awakenings should reduce the difference in HR from the mean

baseline. Lastly, it is expected a direct relationship would be observed between WASO and the heart rate variability, as an increased number of WASO should result in larger HR fluctuations from the norm.

The final group of assumptions addresses the relationship between the subjective sleep scales and the HR values. It is thought that the VSH score should have a direct relationship with the corrected HR mean values, suggesting that the higher differences in HR from the mean would indicate increased sleep quality. In turn, VSH scores should demonstrate an inverse relationship with the measure of HR variability, inferring higher scores would be related to fewer shifts between waking and sleeping states. SSS scores would be expected to have an inverse relationship with the corrected HR means, suggesting that higher ratings of sleepiness would be reflected in HR values that did not move much below an evening baseline (inferring less sleep). And finally, it is predicted that SSS scores would show a direct relationship with HR variability, as higher reports of sleepiness should correspond with frequent shifts between sleep and wakefulness.

### Instrumentation

#### Subjective sleep measurement.

The measurement tool used to collect subjective reports of sleep quantity was the sleep diary. Sleep diaries or sleep logs are detailed self-reports of sleeping and waking activities and are a sleep measurement technique that is widely utilized in the clinical and research settings. The format of sleep diaries varies greatly as they can be used to document sleep-wake patterns as short as a single overnight period or on a 24- hour basis for many days or weeks. Participants are required to recall sleep events of the previous night and record observations in a written format on a standardized sleep record.

Some authors argue that self-report measures are not sensitive enough to detect individual variations in sleep and do not correlate precisely with polysomnography (Closs, 1988; Webb, 1989). However, there is evidence that suggests individuals can estimate sleep onset latencies, sleeping and waking times and time awake after sleep onset with some degree of accuracy (Friedman, Bliwise, Yesavage & Salom, 1991; Hoch et al., 1987 as reported in Webb, 1989; Rogers et al., 1993). In Rogers et al.'s study of narcoleptic and normal subjects, they reported an overall agreement of 0.87 correlation between polysomnography and sleep diaries. Sensitivity and specificity was also high at 92.3% and 95.6% respectively. They also noted that the type of study participant affected the accuracy of the sleep diaries with narcoleptic individuals making significantly more errors than the controls. Friedman et al. (1991) suggest that this difference may be in the subjective interpretation of sleep stages, as people with sleep disorders may interpret stage 1 sleep as wake time while good sleepers do not. Subjects in the study were found to have the most disagreements with the polysomnographic recordings during sleep transition times (i.e., sleep onset and termination). They suggest that encouraging study participants to complete the record immediately after awakening in the morning can enhance the accuracy of sleep diary entries.

Although reliability testing on the use of sleep diaries reported in the known literature centers on middle-aged subjects (Haythornthewaite et al., 1991; Rogers et al, 1993), this has not been addressed in a purely elderly population. However, a number of studies have successfully employed the use of sleep diaries or logs in the study of elderly sleep patterns (Buysse et al, 1992; Floyd, 1995; Hayter, 1985, 1996). Therefore, due to the support in the literature for the use and accuracy of sleep diaries, this measurement

tool was chosen as the benchmark in which to compare subjective reports of sleep quality and objective HR data.

Two tools were utilized to obtain subjective measures of sleep quality. The first instrument was the Stanford Sleepiness Scale (SSS) (Hoddes et al., 1973). Sleepiness is the main aspect of mood that is a reflection upon sleep ability and status. The SSS is a single item scale with 7 levels of descriptors ranging from 1 (feeling alert, wide awake) to 7 (excessively sleepy) that was developed to measure subjective reports of sleepiness (Appendix C). The SSS is a flexible and well-known tool, as it has been used with a number of populations and can be administered in a self-report or interview approach. Studies involving young adults have demonstrated that the SSS is sensitive to total and partial sleep deprivation, subsequent recovery and polysomnographic recordings (Closs, 1988). On subjects who underwent one night total sleep deprivation, Hoddes et al (1973) found the mean SSS rating correlated ( $r=.68$ ) with performance on Wilkinson addition and vigilance tests. Herscovitch and Broughton (1981) also investigated the sensitivity of the SSS in young adults to the effects of cumulative partial sleep deprivation. They found that the SSS values rose significantly after 5 days of partial sleep deprivation relative to baseline and recovery values. As with the sleep diaries, the SSS has not been validated with an elderly sample. However, studies have employed the use of the SSS in this population (Carskadon, Seidel, Greenblatt & Dement, 1982; Newmann & Broughton, 1991)

The second measurement tool used in this study to obtain a subjective reflection of sleep quality was the VSH Sleep Scale (Snyder-Halpern & Verran, 1987). This cumulative scale involves the use of a series of 8 visual analogue scales to address



significant characteristics associated with sleep quality; sleep fragmentation, length, delay and depth (Appendix D). Study participants are asked to answer each question by placing a vertical mark on a 10 cm line between the two verbal extremes. A measurement is taken from the 0 end point to the indicator mark and a score is obtained for the question. It is possible to obtain a total scale score between 0 to 80, which is reflective of subjective sleep quality. Scores on items relating to awakenings, movement during sleep and sleep latency must be reversed and then all of the items summed. A higher score indicates a better quality of sleep. When tested on normal subjects, the VSH scale had a reliability coefficient of .82 theta. Convergence construct validity was tested against two other subjective sleep quality tools and all but one scale item demonstrated correlations above 0.5. The scale was also noted to detect age related changes in sleep disturbance in their elderly cohort (Snyder-Halpern & Verran, 1987).

Visual analogue scales are a simple and effective method of measuring subjective sleep quality and have been used in various sleep studies (Closs, 1988; Babkoff, Caspy, Mikulincer, 1991). Although no known studies have tested their use in measuring sleep in an elderly population, these scales have been accepted as a valid and reliable method for the measurement of clinical phenomena (Wewers & Lowe, 1990).

#### Objective sleep measurement.

Heart rate profiles during sleep were obtained by means of a Polar Vantage NV portable heart rate monitor and Polar Precision Performance Software for Windows version 2.0 (Polar Electro Oy, Kempele, Finland). The monitoring system consists of a narrow plastic band secured around the chest which contains two HR electrodes and a transmitter, and a wrist watch that stores the data points transmitted from the chest unit.

The chest transmitter detects the electrical impulses of the cardiac cycle and measures the distance between the R waves (R to R interval) to determine an average heart rate in beats per minute (bpm). This HR monitoring system has the ability to provide HR averaging over 5, 15 and 60 second intervals. The 5 second interval was utilized for this study in an attempt to detect HR fluctuations that may be evident in brief arousals common in this population (Dement et al., 1982; Pressman & Fry, 1988; Schnelle, Cruise, et al, 1998). Total memory capacity for recordings at 5 second intervals allows for 11 hours and 15 minutes of continuous HR monitoring.

Information that is collected and stored in the wrist watch receiver is then downloaded to the Polar Precision computer software program through an interface device. Once this information is input into the program, a number of graphical and listing outputs can be displayed.

Although no known studies have utilized portable HR monitors to obtain sleep HR profiles in elderly individuals, this technique has been utilized to examine daily activities and risk for recurrent falls in institutionalized elderly (Jonsson et al., 1990). The 38 elders in their study were 86 years old, on average, and wore the ambulatory HR monitor for 6 hours from 0700 to 1300. The authors did not report any difficulties in the use of this measurement technique in their elderly study population.

### Sampling Design

The sample for this study was drawn from two long-term care and rehabilitation centers in Winnipeg, Manitoba (Riverview Health Centre and Deer Lodge Centre). These facilities provides a range of in-patient services to the elderly population that includes general personal care, interim personal care, special needs personal care,

personal care respite, rehabilitation (geriatric, respiratory, neurology, orthopedic), chronic care (general, respiratory, neurology), palliative care and behavior management.

The target population included all residents residing in a general or interim personal care bed. A convenience sample was used to recruit 23 study participants. Each resident was requested to participate for one night of sleep monitoring. Using a one-tailed test, a sample of 23 provides 0.75 power to detect a correlation coefficient effect size ( $r$ ) of 0.40 (moderate to large effect) at a significance level of 0.10 (Cohen, 1969).

The Managers/Coordinators on the personal care units were approached after facility access was gained and requested to formulate a list of residents on their respective units that met the following inclusion criteria;

1. The resident was female and 60 years of age and older;
2. The resident was able to read and/or communicate in English;
3. The resident was cognitively intact as indicated by a score of 24 or higher on the Folstein Mini-Mental Status Exam (MMSE) (Folstein, Folstein, & McHugh, 1975; Tombaugh & McIntyre, 1992);
4. The resident had not been diagnosed with a sleep disorder (i.e., sleep apnea, narcolepsy), and;
5. The resident did not have an implanted electronic cardiac device, such as a pacemaker or internal defibrillator.

Only females were included in the sample to eliminate possible gender bias in a relatively small sample size and facilitate participant recruitment in the PCH setting.

Residents were required to communicate in English and be cognitively intact to allow use of the sleep diaries.

The Managers/Coordinators were also requested to compile a list of residents that met the inclusion criteria and thought to be cognitively intact but did not have a MMSE score

on their chart. In these cases, the researcher administered a MMSE, after gaining consent, to determine eligibility in the study pertaining to this criterion.

Once this information was determined, the Managers/Coordinators were requested to approach potential participants to mention the study and inquire as to their interest in receiving further information. If they indicated interest in the study, the researcher then approached them at a later date to further explain details about their participation. At this time, the residents were shown a copy of the sleep diary record and asked to read the information printed on the sheet and to provide a sample entry to determine their ability to utilize and comprehend the form. Consent to participate in the study was then obtained as the final inclusion criteria.

### Procedure

During the researcher's interview with the participants to gain consent and describe the study, residents amenable to participation were asked to state their usual bedtime. On a mutually agreeable evening, the researcher returned to the unit prior to the stated bedtime and applied the HR monitoring system to the resident. The time of monitor application was noted on the individual's sleep diary record. At this time, the sleep diary record and required entries were again reviewed with the resident (Appendix E). Demographic information from the resident's chart was also collected once consent was gained (Appendix F).

To obtain an evening baseline HR recording, the participants were asked to sit quietly on the side of the bed or in a chair for 5 minutes sometime prior to bedtime. Before falling asleep for the night, the participants were requested to record the exact time in hours and minutes of when they physically went to bed. During the nighttime

period, the sleep diary was kept on a clipboard and left on or near the bed. Participants were encouraged to write down the time of nighttime awakenings during the night to assist with recall the next morning. Upon awakening in the morning the participants were requested to record the time of sleep onset the previous night, the time and length of periods of wakefulness after sleep onset, final awakening in the morning and the time they rose from bed for the day. If the resident did not have a digital clock in their room, a large-numbered digital clock was placed at the bedside to ensure the participants had access to the correct time. The HR monitor watch was set to the exact time of the bedside clocks.

The researcher returned in the morning after the monitored sleep period to remove the apparatus. The resident's skin condition around the site of the chest strap was examined for signs of breakdown. At this time, the sleep diary record was reviewed with the resident for accuracy and completeness. If any sections were incomplete, the participants were asked to recall the times of the missing information. The VSH sleep scale of subjective sleep quality was also administered at this time and the researcher inquired as to any difficulties with the HR monitor, any unusual occurrences during the night, sources of sleep disturbance and if they had taken any sleep aid medications prior to bedtime or during that night. To obtain a measurement of sleepiness during the daytime period, the SSS was left with the participants to self-administer at noontime on that same day.

#### Access to the Research Setting

In order to gain research access to these long-term care facilities, the researcher was required to submit a completed request for research access form, copies of the

research proposal and evidence of ethical approval to both facilities' research access coordinators. Research access to the facilities was approved prior to the initiation of any data collection in the respective sites.

### Ethical Considerations

Any study involving human subjects must ensure that the rights of the research participants are safeguarded (Tri-Council Policy Statement, 1998). As this study involved a sample of older institutionalized adults, a number of ethical issues require consideration.

A third party initially approached the potential participants to inquire as to their interest in the study. If they indicated interest in the project, the researcher then approached them to provide further information and obtain informed consent. Informed consent requires that research participants be informed of the purpose, risks and benefits of participation in a study prior to its commencement. In this study, the researcher provided a detailed verbal description of the research project and also physically demonstrated the application of the HR monitor. The research participants were also requested to read and sign a written consent form that affirmed their agreement to participate in the study (Appendix G). Upon signing, they were provided with a copy of this consent form.

During the explanation of involvement in the study, participants were informed that their participation was strictly voluntary and that they may withdraw at any time, if they so desire. This information was also repeated in the content of the consent form.

The confidentiality of all subjects and their respective objective and subjective data was maintained throughout the study. Adherence to the requirements of the Personal

Health Information Act (PHIA) was also maintained throughout the study in regards to the participants' health information that was collected. An identification number identified participants and appeared on all data collection forms and HR profiles in place of names. The researcher was the sole person completing data collection and had access to the data along with her thesis adviser and statistical consultant.

Lastly, a researcher must ensure that participants are protected from physical, mental and emotional stress and harm that may ensue as a result of the research study. In this study, a potential for physical discomfort and skin breakdown existed in relation to wearing the chest HR monitor for a period of time overnight. To address these concerns, the researcher offered to apply the apparatus on the participants prior to their involvement in the study. At this time, they had the opportunity to discuss any comfort issues and withdraw from the study if they felt unable to tolerate wearing the monitor. Participants were also made aware that they may remove the chest device at any time during the night if they were in distress and withdraw from the study at any time. At the end of each monitoring period, the participants' skin around the chest monitor application site was inspected for any signs of skin breakdown. If any were noted, the nursing staff would have been notified and consulted for treatment of the skin breakdown. The interview conducted at the end of each monitoring period was kept brief (approximately 10-15 minutes) to minimize any inconvenience concerning the resident's personal time and daily activities.

### Data Analysis

To provide an overall view of the data, descriptive statistics were used to outline the demographic characteristics of the sample, study variables and HR data. Cronbach's

reliability coefficient alpha was calculated to determine internal consistency of the VSH scale. Bivariate and step-wise multiple regression models were used to examine for relationships between variables. The HR profiles and corresponding sleep diaries from each night of recording were examined both collectively through statistical testing and also on an individual basis to reduce confounding of the data from individual variation in sleep patterns (Aldredge & Welch, 1973). The Polar Precision software package (version 2.0), Microsoft Excel (1997 version) and the Statistical Package for the Social Sciences (SPSS version 9.0) were utilized to obtain graphical and statistical data for this project. As noted above, the level of significance for statistical testing was set at .10.

Hypothesis #1: Sleep diary values (WASO and TST) will be related to subjective reports of sleep quality (SSS and VSH scores).

Research hypothesis #1 was tested by a progression of statistical procedures. To observe for the presence of linear relationships between data in the sleep diaries and measures of subjective sleep quality, TST (in minutes) and WASO were calculated from the sleep diary and examined in relation to both the SSS and VSH scale scores (total and sub-scores) using Spearman's rho and Pearson Product moment correlation ( $r$ ) respectively. In addition, scatter plots were used to assess the existence of linear relationships between these measures. Lastly, two separate step-wise regression models were applied to observe for the relationship of TST, WASO and their interaction term with both SSS and VSH scores. The interaction term was obtained by multiplying TST with WASO and included in the regression model to determine if the impact of TST on the sleep scale scores was influenced by the presence of WASO.



Hypothesis #2: Sleep diary values (WASO and TST) will be related to overall measures of HR during sleep.

To compare data reported in the sleep diaries and the corresponding HR data, as suggested by hypothesis #2, several statistical procedures were used. The first step in the analysis of this hypothesis was to derive a meaningful numeric representation of the HR information during the participants' sleep period. With the assistance of Microsoft Excel software, 5 values were calculated for each sleep period:

1. Overall mean of the HR during the nighttime period
2. Mean difference in HR during the night compared to baseline – sleep diary (corrected mean1)
3. Variability of HR during the night – sleep diary (SD1)
4. Mean difference in HR during the night compared to baseline – HR profile (corrected mean2)
5. Variability of HR during the night – HR profile (SD2)

First, the overall mean of the HR during the nighttime period was determined by averaging all HR measurements between the time the participants stated they fell asleep and woke up for the day in the sleep diaries. It was felt that the overall mean HR would not provide an accurate picture when inter-subject differences are examined, as some individuals may normally have higher or lower average HRs than others. Therefore, this value was used for descriptive purposes and a second variable, corrected mean1, was then calculated for statistical testing. This value expressed the absolute difference between a baseline, or resting HR, during the evening and each HR measurement during the sleep period. The mean HR of the 5-minute resting period prior to sleep was calculated to

represent the resting HR. Each HR measurement during the night, from SO to ST as stated in the sleep diary, was subtracted from the resting HR to obtain the absolute difference between the two values. These differences were then averaged to obtain an overall measure of HR change during the night compared to the evening baseline, or corrected mean1. A corrected mean that was low or close to zero would imply a person had very little sleep during the night because their HR did not show much reduction from their waking baseline HR. Conversely, a high corrected mean would indicate HRs much lower than baseline throughout the night, suggesting the individual spent more time in the sleep state.

It was then noted that a third value was required to reflect the fluctuations or variability of the HR throughout the nighttime period. The standard deviation (SD1) of the corrected HR values above was calculated to represent the variability in HR. Theoretically, this value should predict a steady or varied state of either sleep or wakefulness. For example, a high SD would indicate great variability in sleep states and possibly many shifts between sleep and wakefulness. A low SD value would then suggest a more constant state either in sleep or wakefulness. By examining the mean and standard deviation in combination, it was hypothesized that these values would provide an overall, objective representation of sleep-wake activity during a nighttime period.

The fourth and fifth values, corrected mean2 and SD2, were calculated as described above. However, instead of using the sleep diary data as parameters for SO and ST, the HR profiles were examined visually and these times determined by the researcher based on the expected declines and rises in HR, as supported by the literature in this field.

Once these HR values were computed, analysis of hypothesis #2 proceeded in a similar fashion to the previous hypothesis using Pearson's  $r$  correlations, scatter plots and step-wise regression models. Four separate step-wise regression models were employed to examine the relationship of the two sets of means and standards deviations, plus their interaction term (mean multiplied by standard deviation) on TST and WASO.

Four-quadrant graphs were also used to plot the two sets of corrected HR means with the respective measures of variability (SD) together, as described above. By plotting the mean HR values on the x axis and SD values on the y axis and then dividing the graph into 4 quadrants by two intersecting lines that represent the means of each data set, it was hypothesized that any patterns or relationships that existed between the variables might be observed (Figure 2). The four resultant quadrants can be numbered from I to IV beginning in the upper right quadrant and moving counter-clockwise. This theoretically offered 4 possible sleep scenarios when attempting to predict how well a person slept. Data points falling within quadrant I would represent HR with a higher mean difference from baseline and a high measure of HR variability, suggesting a mediocre sleep where a person was awake on and off throughout the night. Points falling within quadrant II would have a low mean difference in HR from baseline yet a higher measure of variability. Again, it is hypothesized that this would indicate a mediocre sleep, but due to the lower variability in HR, may indicate a person was awake for longer periods during the night as compared to people in quadrant I. Heart rate data with a low mean difference and low measure of variability would fall within quadrant III. This should indicate that a person had remained awake for most of the night as the HR had not changed much since baseline and had remained relatively stable throughout the night.

Therefore, it is thought that this quadrant would represent the worse sleep scenario.

Finally, large mean differences in HR from baseline coupled with a low measure of heart rate variability, was hypothesized to represent long periods of time spent sleeping with little or no interruptions. Thus, people falling within quadrant IV were expected to experience the best sleep in relation to the total sample.

Hypothesis #3: Subjective reports of sleep quality (SSS and VSH scores) will be related to overall measures of nightly HR values.

Analysis of hypothesis #3 was undertaken to observe for relationships between the sleep quality scales and the overall measure of HR during the nighttime period. Again, Pearson's  $r$  and Spearman's  $\rho$  correlations were used, as well as scatter plots and four separate step-wise regression models to determine the relationship of the two sets of HR values (corrected mean, SD and their interaction term) on SSS and VSH scores.

Hypothesis #4: Sleep diary records will be related to HR patterns observed during that night's monitoring period.

To test research hypothesis #4, SO, WASO and ST values from the sleep diaries were superimposed on the corresponding HR profiles to allow visual examination of the two sets of data for congruence. A set of scoring rules was developed from parameters outlined in the current literature and used to determine the times of SO, WASO and ST. Sleep onset was defined as a progressive, sustained decline in HR moving below the baseline or a consistent change in the quality or variability of the HR. The SO value was recorded as the time where this change was truly evident and consistent. Sleep termination was determined by an abrupt rise in HR at the end of the sleep period and the

value recorded just prior to the crest of the HR peak. Wakenings after sleep onset were defined by a sudden and sustained rise in HR followed by subsequent decline that was quite apparent from the surrounding HR data. Rises in HR that were unexplained by the sleep diary were noted if they demonstrated similar patterns to documented WASOs in the same profile. A WASO was considered to be a match between the sleep diary and HR profile if there was some overlap in the time parameters or close proximity of time frame (within 30 minutes) and there was similarity in length. The difference in minutes for SO and ST between the sleep diary entries and HR profiles plus the number of matches and inconsistencies of WASOs were recorded and documented descriptively.

And lastly, the percentage of decrease in HR values between the periods of evening baseline, SL and SO, as documented on the sleep diary and noted on the HR profile, were calculated and then compared to the conceptual model of HR changes during sleep as proposed in Figure 1.

#### Chapter Four: Study Findings and Results

The following section reports the findings of the study and results of the data analysis, as described above. Presented below is the demographic profile of the sample, description of the study variables and HR data and results of the internal consistency of the VSH scale. Statistical results from the correlation coefficients, scatter plots, four-quadrant graphs and step-wise linear regression analyses are discussed. The inductive comparison of sleep diary data and HR profiles are also addressed.

##### Description of the Sample

###### Participant involvement in the study.

Of the residents from the two data collection sites (Riverview Health Centre and Deer Lodge Centre), 31 older adults met the study criteria. Eight residents who were approached by the researcher refused to participate in the study. Reasons for refusal were: not interested in this particular study ( $n=5$ ), not feeling well ( $n=2$ ) and participation in the study would not assist them in returning to their home in the community ( $n=1$ ). Twenty-three residents agreed to participate, representing a 74% response rate.

Two nights of monitoring were repeated for two separate participants, as a full night of recording did not occur on the first attempt. The reasons for these occurrences were interference caused from a resident's personal computer with the monitor's transmitter and accidental depression of the start/stop button during the night.

#### Demographics of the participants.

The older women in the sample ( $N=23$ ) ranged in age from 62 to 99 with a mean age of 79.6 years ( $SD=8.5$ ). MMSE scores ranged from 24 to 30 with a mean score of 27 ( $SD=2$ ). Length of stay (LOS) on the units varied from 91 days to 1,322 days with 560.5 days ( $SD=406.7$ ) or approximately 1 ½ years as the average.

The number of formal medical diagnoses on the health record for each participant ranged from 2 to 10 with a mean of 5.2 ( $SD=1.9$ ). The detailed information on frequencies of medical diagnoses that appeared on the health record is presented in Appendix H. The three most common diagnoses found in the sample were: cerebral vascular accidents/transient ischemic attacks (47.8%), osteoarthritis (39.1%) and congestive heart failure (34.8%).

The number of regularly scheduled and PRN medications in each participants' regime varied from 3 to 16 different drugs with a mean of 9.1 (SD =3.3). When individual medications were grouped, 20 categories of drugs were noted as well as a large number of miscellaneous drugs (Appendix I). When the entire sample was considered, the most common medications included in the participants' regimes were: bowel aids/laxatives (69.5%), diuretics (65.2%), non-narcotic analgesics (52.2%) and antidepressants (52.2%). The mostly commonly ordered medications tended to coincide with the sample's most frequently occurring medical diagnoses.

Of the 23 participants, 8 or 34.8% had taken a sleep-aid medication during their night of participation in the study (Table 1). Of these 8 people, 3 (37.5%) reported taking Tylenol plain for a sleep aid, 2 (25%) an anxiolytic, 2 (25%) an antidepressant and one participant (12.5%) took Tylenol plain, an antidepressant, an anxiolytic and an anti-psychotic prior to bedtime.

#### Description of the study variables.

A summation of the study's sleep variables is presented in Table 2. Total sleep time (TST) from the participants' sleep diaries was calculated by including the number of minutes between reported SO and ST minus the amount of time spent awake during the night or the length of each WASO. TST ranged from 135 minutes or 2.25 hours, to 541 minutes or 9 hours. Mean for the sample was 384.3 minutes (SD =121.6) or nearly 6 and a half-hours of sleep during the night. The number of reported awakenings after sleep onset (WASO) obtained from the sleep diaries ranged from 0 to 4 with a mean of 1.8 (SD =1.2).

Stanford Sleepiness Scale scores ranged the entire breadth of the scale from 1 to 7 with a mean score of 3 ( $SD = 2$ ), suggesting the sample generally reported feeling slightly more alert than sleepy. VSH scale scores ranged from 27.5 to 67.4 out of a possible 80, with a mean of 47.2 ( $SD = 12.1$ ). As the mean is slightly higher than the center point of the scale, this suggests that the participants were not either strongly satisfied or dissatisfied with the quality of their sleep. Means and SD of the sub-scores for the VSH scale are presented in detail (Table 3).

Average HR during sleep ranged from 52.5 bpm to 95.7 bpm, with a mean of 70.5 ( $SD = 12.4$ ). Resting HR obtained from the 5-minute evening baseline measurement ranged from 56.7 bpm to 114.4 bpm, with a mean of 78.9 ( $SD = 15$ ). The average corrected HR from resting (corrected mean1) ranged from  $-0.75$  bpm to 28.1 bpm, showing a mean of 8.4 ( $SD = 6.8$ ). HR variability ( $SD1$ ) varied from 1.7 to 12.3 with a mean of 5.1 ( $SD = 2.7$ ). The second set of corrected HR values (corrected mean2 and  $SD2$ ) demonstrated similar values with ranges of  $-0.76$  to 27.9 bpm and 1.65 to 12.3 respectively and means of 8.5 bpm ( $SD = 6.7$ ) and 5.0 ( $SD = 2.7$ ). At times during the nighttime period, the HR monitor was not able to detect the cardiac impulse correctly and recorded either abnormally low or high HR values. These values were removed from the data set prior to the calculation of the above variables (Appendix J).

#### Internal Consistency of the VSH Scale

The VSH scale demonstrated an acceptable level of internal consistency, as Cronbach's reliability coefficient alpha was .78. This result is similar to the reliability testing found by Snyder-Halpern & Verran (1987) who reported internal consistency of the scale to be .82.



### Hypothesis #1

Sleep diary values (WASO and TST) will be related to subjective reports of sleep quality (SSS and VSH scores).

Bivariate analysis of dependent and independent variables is given in Table 4 and revealed significant findings. As predicted, TST showed a significant positive relationship with the total VSH score ( $r = .65$ ,  $p = .001$ ), which indicated that the higher the total sleep participants obtained, the higher they rated their sleep quality. TST also demonstrated a negative correlation with the SSS, suggesting the higher amounts of sleep were associated with lower ratings of sleepiness, however, this relationship fell short of the desired level of significance ( $r = -.33$ ,  $p = .12$ ). WASO did not significantly correlate with either the VSH or SSS. TST and WASO demonstrated a significant positive relationship ( $r = .43$ ,  $p = .04$ ), which was the opposite direction expected, suggesting that longer sleep times were associated with increased number of awakenings during the sleep period. Also, as predicted, SSS and VSH scale scores correlated negatively ( $r = -.46$ ,  $p = .03$ ), which suggests that lower reports of sleepiness correspond to higher reports of sleep quality, and vice versa.

Scatter plots of the above variables show similar results. The graph of TST plotted with VSH scores indicates a positive relationship between the variables (Figure 3). When TST is plotted with the SSS, a suggestion of a negative relationship is present considering the three points at the top left area of the graph (Figure 4). However, a number of outliers cloud the interpretation. Similar to the bivariate analysis, when WASO are plotted against the VSH and SSS, no relationship is evident (Figures 5 and 6).

Analysis of the two separate step-wise regression models using both VSH and SSS scores as dependent variables and TST, WASO and the interaction term as independent variables, revealed some significant results. Linear regression analysis indicated that the VSH score should be estimated by a function involving linear terms with TST ( $t=5.086$ ,  $p=.000$ ) and WASO ( $t=-2.539$ ,  $p=.02$ ) (Table 5). These two variables accounted for just over half of the variation on VSH scale scores ( $R^2=.565$ ,  $p=.000$ ). The addition of the interaction term to the model was not statistically significant and did not significantly increase explanatory power (Table 6).

Linear regression analysis with SSS score as the dependent variable and TST, WASO and the interaction term as dependent variables also yielded significant findings (Table 7). Using all three dependent variables in the model produced the strongest explanatory ability ( $R^2=.441$ ,  $p=.01$ ) with significance obtained for TST ( $t=-3.863$ ,  $p=.001$ ), WASO ( $t=-1.956$ ,  $p=.065$ ) and the interaction term ( $t=2.369$ ,  $p=.029$ ).

### Hypothesis #2

Sleep diary values (WASO and TST) will be related to overall measures of HR during sleep.

Using bivariate analysis, this hypothesis was partially supported by a significant positive relationship between WASO and both measures of HR variability over night (SD1 and SD2), with Pearson's  $r$  of .38 ( $p=.08$ ) and .39 ( $p=.07$ ). This would suggest that HR variability increased with a higher frequency of awakenings during the night. No other significant relationships were evident (Table 4). Scatter plots of the same variables suggested a positive relationship between WASO and SD1 and SD2 (Figures 7 and 8), however, patterns were not distinguishable from the other graphs.

When the corrected mean1 and corrected mean2 were plotted on a 4-quadrant graph with the respective standard deviations, again there was no evidence of a consistent pattern. In fact, most of the points tended to cluster around the intersection of the two axes, suggesting no relationship between the variables (Figures 9 and 10).

There was no evidence of a regression relationship between TST and corrected mean1, SD1 and their interaction term, nor TST and corrected mean2, SD2 and their interaction term (Tables 8 and 9). The strongest regression models for WASO only included the corrected mean and SD variables, however, these relationships did not achieve statistical significance (Tables 10 and 11).

#### Hypothesis #3

Subjective reports of sleep quality (SSS and VSH scores) will be related to overall measures of nightly HR values.

There were no significant correlations found between these variables (Table 4) or any consistent patterns noted in the scatter plots. Multiple step-wise regression analyses failed to identify any significant regression relationships between the HR values and the SSS and VSH scores (Tables 12 through 15).

#### Hypothesis #4

Sleep diary records will be related to HR patterns observed during that night's monitoring period.

The individual profiles with corresponding data points determined from the sleep diary and HR information is presented in Appendix K. To aid comparison between the two sets of data, times from the sleep diaries were charted on the lower portion of the profiles, while the reference points determined by the researcher using the above

guidelines were documented on the upper section. A summary of the matches and inconsistencies between the two sets of data yielded some interesting results (Table 16). In regards to SO and ST, positive values indicated an overestimation from the participants' perspective in the time it took to fall asleep and the time they awoke in the morning. A negative value represented an underestimation of sleep latency and an earlier time of awakening compared to the profile. On average, study participants overestimated their sleep latency period by 6.6 minutes ( $SD = 19.7$ ), with a range between  $-17$  to  $55$  minutes. Three participants (13%) had an exact match ( $\pm 1$  minute). Estimations of ST varied between  $-17$  to  $42$  minutes, with a mean of  $5.4$  minutes ( $SD = 13.9$ ). Six of 19 subjects (31.5%) had exact matches with the HR profile ( $\pm 1$  minute). Four of the participants were not included in this calculation as ST in these cases occurred after the memory capacity of the HR receiver had been exceeded by time of waking in the morning.

Thirty-five of the 41 (85.4%) recorded WASOs in the sleep diary matched the profile by the above mentioned criteria. Nineteen unexplained rises in HR after sleep onset were evident in the profiles, therefore, 35 events out of 60 (58.3%) were accounted for by the sleep diary records.

When the percentage of HR decline was calculated between evening baseline, SL and SO (Table 17), it was noted that the conceptual model of HR changes as depicted in Figure 1 was not useful in predicting these values in the study population. Only one profile (ID#13) corresponded to the model's parameters for all values. Sleep onset values fit somewhat better into the model with 9 of 23 (39.1%) of the sleep diary records and 11 of 23 (47.8%) of the HR profile data falling within the range of a 2-5.5% decrease ( $\pm$

1%). On average, the sample's SL HR decline from evening baseline was 6.0% and 4.9% for sleep diary records and HR profile estimates respectively. A further mean decrease of 4.59% for sleep diary values and 5.41% for HR profile data was seen between SL and SO.

In summary, the statistical analysis suggested some significant relationships between TST in the sleep diaries and subjective scales of sleep quality. There was slight evidence of a statistical relationship between the HR values and WASO in the sleep diaries but none existed between the HR values and subjective sleep scales. Visual comparison of the sleep diary values and HR profiles demonstrated some evidence of congruence between these two sets of data.

### Chapter Five: Discussion

This chapter integrates the study results with relevant literature in this field and offers explanations for the findings. Comparison of the use of the subjective measures is addressed, as well as the potential to use HR monitoring as an indication of sleep quality and quantity. Some parallels are made between the study's findings and sleep characteristics of the institutionalized elderly population. And finally, limitations of the study and implications for future research and the clinical setting are discussed.

#### Comparison of the Subjective Measures

There was some evidence that suggested consistency between the subjective reports of sleep quantity in the sleep diary and the subjective ratings of sleep quality in this sample, which provides support for the use of the sleep diary as the baseline measurement in this study (Friedman et al., 1991; Hoch et al., 1987; Rogers et al., 1993). From the perspective of the sleep diary, TST demonstrated the strongest agreement with

the VSH scores. This finding suggested that the higher total sleep participants reported, the higher they tended to rate the quality of their sleep and vice versa.

Although there was a suggestion of an inverse relationship between TST and the SSS scores, this did not reach a level of statistical significance. This may be explained by the timing of the administration of the SSS later in the day when ratings of sleepiness are dependent upon other factors, such as morning naps (Hoddes et al., 1973), or the coarseness of the SSS scale compared to the VSH scale. However, the VSH and SSS scores did demonstrate a significant inverse relationship, suggesting that there was a relationship between the participants' ratings of sleep ability and status to feelings of sleepiness later that day (Closs, 1988; Hoddes et al., 1973).

WASO failed to show any significant relationship with either scale through bivariate analysis. However, when it was included in a regression model with TST, there was a significant combined explanatory effect of these variables on both the VSH and SSS scores. This would suggest that WASO alone is not a predictor of the subjective quality of sleep and there is an interaction between awakenings during sleep and TST. A possible related factor might be the length of the WASO, which affected the TST value but was not calculated as an individual variable. This notion is supported in the literature by Prinz and Vitiello (1993), who suggest that WASO can result in sleep disruption by either frequently interrupting sleep or manifest in extended periods of wakefulness during the night. Perhaps the addition of a third sleep diary variable, total length of WASO, may have been useful to establish further agreement between the sleep diary data and sleep quality ratings.

Further to this, when the HR profiles were examined individually, it was noted that 3 participants (ID# 4, 10 and 13) reported the number of WASO as zero or one. However, each person also had a very low TST (range of 135 to 141 minutes) as they had only slept a short period and then lay awake in bed for the remainder of the night. In these cases, the assumption that reports of fewer WASO should correspond to higher ratings of sleep quality (Boselli et al., 1998; Buysse et al., 1992; Gislason et al., 1993), would not be applicable. Through bivariate analysis, TST and WASO did demonstrate a significant positive relationship, which further supports this suggestion that lower amounts of total sleep were related to a lesser number of WASO and vice versa.

#### The Use of HR as an Indicator of Sleep Quality and Quantity

From indications in the literature, it was hypothesized that monitoring fluctuations of HR during the sleep period would provide a useful methodology to obtain an adequate objective representation of sleep quality and quantity for both clinical practice and research (Aldredge & Welch, 1973; Baust & Bohnert, 1969; Brooks et al., 1956; Cajochen et al., 1994; Degaute et al., 1991; Geroge & Kryger, 1985; Khartri & Freis, 1967; Welch & Richardson, 1973; Zemaityte et al., 1984). However, results of the statistical analyses in this study were inconclusive to support the use of HR alone for these purposes. Through bivariate analysis, there was a suggestion of a direct relationship between WASO and variability of HR during the night, indicating that increased numbers of awakenings were related to higher variations in HR and vice versa. However, this relationship was not supported in the multiple regression analysis. There was no evidence of a relationship between TST in the sleep diaries or the subjective sleep quality scales and the HR measures.

There are a number of possible explanations for an absence of the hypothesized findings. First, HR data was recorded in 5 second intervals during the entire night to determine the existence of transient arousals (Dement, et al., 1982; Pressman & Fry, 1988; Schnelle et al., 1998), which in turn resulted in an impressive volume of data. A key aspect of the data analysis process was to determine overall HR values to represent this large data set. Perhaps the corrected means and measures of HR variability used were too coarse of measurements to represent this large volume of data and were not adequately reflecting the sleep experience of the participants. It may have been more representative to sub-divide the HR data into periods of sleep and wakening as determined by the sleep diaries and conduct analyses with these such values (George & Kryger, 1985).

A second suggestion as to why significant findings were not evident may be related to the large variability in sleep patterns between individual participants. As suggested by Aldredge and Welch (1973), when participants' are compared to each other, often there is no relationship between average HR and depth of sleep due to this wide variation. Therefore, inter-subject analysis of HR data presents some difficulties and they suggest that HR variations during each sleep cycle be considered on an intra-subject or individual basis, rather than collectively.

Finally, in addition to the large individual variation in sleep patterns, it is also common to expect even greater differences in sleep patterns among older adults (Bliwise, 1994; Carskadon & Dement, 1994; Dement et al., 1982). Thus, the age of the study's sample may also impact the variability in HR values and subsequent difficulties obtaining significant overall findings.



Visual comparison between the sleep diary records and the HR profiles offered some positive findings for supporting the use of HR measurement to determine sleep patterns. Participants in this study were able to estimate their time of sleep onset and sleep termination as well as WASO with a fair degree of accuracy. Sleep onset and ST were both overestimated by an average of 6.6 minutes and 5.4 minutes respectively. Eighty-five percent of the recorded WASO in the sleep diary matched the changes on the HR profile. This supports the claim made by Friedman et al. (1991) and Rogers et al. (1993) who suggests that individuals without sleep disorders can reasonably estimate sleep onset latencies, waking times and WASO through the use of sleep diaries.

Also of interest was the participants' ability to report ST more accurately than SO, as evidenced by a higher number of exact matches (31.5% to 13%) and less variation of the mean (SD of 13.9 minutes to 19.7), which again is supported in existing literature (Degaute et al., 1991). Another trend noted in many of the HR profiles was a tendency to record a slight delay in the initiation of WASO events. This may be due to either a short delay prior to the participants remembering to document the time when awakening during the night or a physiological acceleration in HR prior to conscious arousal (Bonnet & Arand, 1997).

The study conducted by Welsh and Richardson (1973) suggested that it was possible to attain a reasonably accurate representation of a person's sleep patterns from HR data within subjects who did not demonstrate large variability in the sleep cycle. Partial support for this notion was evident in this study when the HR profiles were examined individually. The HR profiles of 8 participants in particular (ID# 1-3, 10, 11, 17, 19, 21) demonstrated fairly consistent patterns of HR fluctuations during the sleep

period, resulting in a clearer interpretation and increased consistency with the sleep diary data.

However, a number of factors appeared to influence the interpretation and relationship between the sleep diary data and HR profiles. First, a number of events or rises in HR ( $n=19$ ) were evident in the profiles but unexplained by the sleep diary entries. There are four possible explanations for these events.

1. An awakening occurred and the participant did not record the event in the sleep diary.
2. The rise in HR may reflect a transient arousal and the participant was not aware of a conscious awakening (Khatri & Freis, 1976).
3. The rise in HR may be attributed to REM stage sleep (George & Kryger, 1985).
4. The rise in HR may have occurred due to spontaneous movement during sleep (Brooks et al., 1956; Snyder et al., 1964).

Without another means of concurrent monitoring, such as observation, it is not possible to arrive at a conclusion as to why these sustained rises in HR occurred at these times.

Second, due to the nature of the study population, a few of the participants (ID# 14, 15, 20) were quite immobile and either wheelchair or bed-bound during both the day and night. As these individuals tended to exhibit less movement and mobility in general, their HR demonstrated less variation from evening baseline and throughout the night. Interpretation of these profiles proved difficult as evidence of SO, WASO and ST values was not as distinct due to less variation in the HR values.

Also, three of the HR profiles (ID# 4, 6, 12) did not show the expected patterning of HR fluctuations during sleep and were somewhat more difficult to interpret. For

example, profiles #6 and 12 seemed to demonstrate patterns reverse of expected (Aldredge & Welch, 1973; George & Kryger, 1985; Khatri & Freis, 1976), such that HR variability increased during reported periods of sleeping and decreased during wakefulness. There is no known physiological explanation for this clinical phenomenon and may be attributed to age-related changes in sleep physiology or individual differences in sleep architecture.

Finally, the timing of when the sleep diary was completed seemed to influence the accuracy of the record. Participants who completed the diaries when the researcher arrived in the morning rather than immediately after awakening, had more difficulty estimating the sleep parameters. Friedman et al. (1991) also suggests that the accuracy of sleep diaries is enhanced by participants completing the record as soon as they wake in the morning. In this case, participants were able to report a correct number of awakenings during the night, but often could not accurately describe the time or length of the WASO (i.e., ID# 5, 8, 9, 14, 22). HR profiles of participants who completed the diary throughout the night were often quite accurate (i.e., 1-3, 6, 12, 17, 19, 21).

The conceptual framework of projected declines in HR from evening baseline to sleep latency and sleep onset was not useful in predicting these events in this population. Again, this is likely related to the age of the sample, as the conceptual model was based on studies involving younger adults (Degaute et al., 1991; Khartri & Freis, 1967). Based on the results of this study, average HR decline for the sample from evening baseline to sleep latency was between approximately 5-6.0% with a further reduction of 4.5 to 5.5% at sleep onset.

In regards to the use of portable HR monitors in this population, there were very few difficulties. As mentioned above, 2 nights of monitoring were repeated due to circumstances outside of the researcher's control. There was no occurrence of skin breakdown caused by wearing the chest strap during the night. All participants reported that the monitor did not interfere with their sleep or cause discomfort. Residents with respiratory difficulties reported that the monitors did not restrict their breathing or chest expansion. On two occasions (ID#9 and 17), the chest strap appeared to lose contact with the skin surface resulting in abnormal recording of HR values, which required subsequent removal of these portions of data prior to analysis. This appeared to occur when an insufficient amount of conduction jelly was applied to the monitor strap.

#### Sleep Patterns of Institutionalized Elderly

Some of the factors pertaining to sleep patterns and sleep disruption in the institutionalized elderly noted in the literature are compared to this study's findings. The quality of sleep in personal care environments is impacted by many factors including noise, light, staff incontinence care routines, nocturia and pain (Cruise et al., 1998; Gentili et al., 1997; Schnelle, Cruise et al., 1998; Schnelle et al., 1993a; Schnelle et al., 1993b). On average, the participants in this study rated the overall quality of their sleep as mediocre as evidenced by the total VSH score of 47.2/80 and the VSH8 sub-score of 5.5/10. The most frequent stated cause of sleep disruption in the sample was nocturia. As most of the participants in this study dwelled in single, private rooms, staff noise or routines and roommate noise was not an identified source of sleep disruption in this group.

WASO are felt to be one of the most significant reported sleep disturbances in aging (Boselli et al., 1998; Buysse, et al., 1992; Gislason et al., 1993). This claim was supported by this study with 20, or 87% of the total sample reporting at least one WASO during the study night. Two of the three participants who reported no WASO had very little TST that night, indicating they also experienced significant sleep disruption.

#### Limitations of the Study

Due to the descriptive and exploratory nature of this project, a number of limitations regarding sampling and the study design are highlighted. In regards to sampling, the recruitment of participants was accomplished through the use of a convenience sample. Therefore, the results of this study are limited in terms of generalizability to other populations. As the study sample only included older women residents in a personal care environment, gender differences in relation to utilizing HR monitors during sleep have not been addressed and further research is needed to include a male cohort. Also, the sample only included cognitively intact residents and the use of HR monitoring during sleep with residents who are cognitively impaired was not explored and the effects on this population is unknown. The relatively small sample size allowed for a level of significance of .10 increasing the probability of committing a Type I error in the statistical analysis.

In regards to measurement techniques, the following limitations are also noted. As mentioned above, portions of data on two HR profiles were removed prior to data analysis due to inadequate contact between the monitor and the skin. Although, there were no corresponding sleep diary entries during these omitted portions, the possibility exists that there were significant events that could have occurred during these times that

would not be noted in the study. This may result in under-reporting of unexplained events found in the HR profiles.

As the use of HR monitors during sleep was relatively unexplored prior to this study, the sleep diaries were used as the benchmark for comparison to the subjective sleep quality scales and the HR data due to the support in the literature for their accuracy in estimation of sleep parameters (Freidman et al., 1991; Hoch et al., 1987 as reported in Webb, 1989; Rogers et al., 1993). The reliability of the sleep diaries is largely dependent upon the ability of the participants to complete them accurately and in a timely manner. As discussed above, some of the participants in the study were not accurate in their estimations of sleep parameters, which in turn affects the ability of the HR values to represent information in the sleep diaries.

#### Implications for Future Research

Both the literature review in preparation for this study and subsequent findings support the need for further research in this area. The literature in this field indicates that there is a pattern of HR fluctuations that occur during the sleep cycle. However, these study results were inconclusive in supporting the use of HR monitoring as an independent measure of objective sleep quality and quantity. It may be beneficial to approach this research question with the addition of another means of objective sleep measurement, such as continuous observation by either a research assistant or videotaping. This would allow for further investigation of unexplained rises in HR to determine their exact causes.

A repeated-measures design with a larger sample size could also be applied in this research context to allow for intra-subject comparison while decreasing the effects of

inter-subjective variability. This may allow for a clearer understanding of the changes in HR throughout the night and how they relate to quality and quantity of sleep.

Finally, the phenomenon of daytime napping was also not addressed by the current study and research into this area is sparse. It would be interesting to explore the changes in HR during daytime sleeping and waking activities to determine if this type of monitoring would be useful on a 24-hour basis. This is a crucial aspect to consider when studying the sleep patterns of older individuals, as a portion of their sleep is obtained during the daytime (Hayter, 1983; Reynolds et al., 1991; Roth et al., 1994).

#### Clinical Implications

It was hoped that the results of the study would support the use of HR monitoring in a clinical setting to determine levels of sleep disruption and effects of nursing interventions to promote sleep. Unfortunately, the results of this study do not support the use of HR monitoring alone to indicate sleep quality and quantity. However, the study does show agreement with the literature in regards to utilizing sleep diaries (Friedman et al, 1991; Hoch et al., 1987 as reported in Webb, 1989; Rogers et al., 1993) and supports their clinical use for these purposes in this study population.

## References

- Aber, R. & Webb, W. (1986). Effects of a limited nap on night sleep in older subjects. Psychology and Aging, 1(4), 300-302
- Adam, K. & Oswald, I. (1983). Protein synthesis, body renewal and the sleep-wake cycle. Clinical Science, 65, 561-567.
- Adam, K. & Oswald, I. (1984). Sleep helps healing. British Medical Journal, 289, 1400-1401.
- Aldredge, J. L. & Welch, A. J. (1973). Variations of heart rate during sleep as a function of the sleep cycle. Electroencephalography and Clinical Neurophysiology, 35, 193-198.
- Ancoli-Israel, S. (1997). Sleep problems in older adults: Putting myths to bed. Geriatrics, 52(1), 20-30.
- Ancoli-Israel, S., Parker, L., Sinae, R., Fell, R. L. & Kripke, D. F. (1989). Sleep fragmentation in patients from a nursing home. Journal of Gerontology, 44(1), M18-M21.
- Atkinson, G., Witte, K., Nold, G., Sasse, U. & Lemmer, B. (1994). Effects of age on circadian blood pressure and heart rate rhythms in patients with primary hypertension. Chronobiology International, 11(1), 35-44.
- Babkoff, H., Caspy, T. & Mikulincer, M. (1991). Subjective sleepiness ratings: The effects of sleep deprivation, circadian rhythmicity and cognitive performance. Sleep, 14(6), 534-539.
- Bachman, D. L. (1992). Sleep disorders with aging: Evaluation and treatment. Geriatrics, 47(9), 53-61.



- Baust, W. & Bohnert, B. (1969). The regulation of heart rate during sleep. Experimental Brain Research, 7, 169-180.
- Beck-Little, R. & Weinrich, S. P. (1998). Assessment and management of sleep disorders in the elderly. Journal of Gerontological Nursing, 24(4), 21-29.
- Bliwise, D. L. (1994). Normal aging. In Kryger, M. H., Roth, T. & Dement, W. C. (Eds.), Principles and practice of sleep medicine (2<sup>nd</sup> ed.) (pp. 26-37). Philadelphia: W. B. Saunders.
- Bonnet, M. H. & Arand, D. L. (1997). Heart rate variability: Sleep stage, time of night and arousal influences. Electroencephalography and Clinical Neurophysiology, 102, 390-396.
- Borbely, A. A. (1994). Sleep homeostasis and models of sleep regulation. In Kryger, M. H., Roth, T. & Dement, W. C. (Eds.), Principles and practice of sleep medicine (2<sup>nd</sup> ed.) (pp. 309-319). Philadelphia: W. B. Saunders.
- Boselli, M., Parrino, L., Smerieri, A. & Terzano, M. G. (1998). Effect of age on EEG arousals in normal sleep. Sleep, 21(4) (abstract).
- Brooks, C. McC., Hoffman, B. F., Suckling, E. E., Kleyntjens, F., Koenig, E. H., Coleman, K. S. & Treumann, H. J. (1956). Sleep and variations in certain functional activities accompanying cyclic changes in depth of sleep. Journal of Applied Physiology, 9, 97-104.
- Buyse, D. J., Browman, K. E., Monk, T. H., Reynolds, C. F., Fasiczka, A. L. & Kupfer, D. J. (1992). Napping and 24-hour sleep/wake patterns in healthy elderly and young adults. Journal of the American Geriatrics Society, 40 (8), 779-786.

Cajochen, C. Pischke, J., Awschbach, D. & Borbely, A. A. (1994). Heart rate dynamics during human sleep. Physiology and Behavior, 55(4), 769-774.

Carskadon, M. A. & Dement, W. C. (1994). Normal human sleep: An overview. In Kryger, M. H., Roth, T. & Dement, W. C. (Eds.), Principles and practice of sleep medicine (2<sup>nd</sup> ed.) (pp. 16-25). Philadelphia: W. B. Saunders.

Carskadon, M. A., Seidel, W. F., Greenblatt, D. J. & Dement, W. C. (1982). Daytime carryover of Triazolam and Flurazepam in elderly insomniacs. Sleep, 5(4), 361-371.

Closs, S. J. (1988). Assessment of sleep in hospital patients: A review of methods. Journal of Advanced Nursing, 13, 501-510.

Cohen, J. (1969). Statistical Power Analysis for the Behavioral Sciences. New York: Academic Press.

Cruise, P. A., Schnelle, J. F., Alessi, C. A., Simmons, S. F. & Ouslander, J. G. (1998). The nighttime environment and incontinence care practices in nursing homes. Journal of the American Geriatrics Society, 46(2), 181-186.

Culebras, A. (1992a). Neuroanatomic and neurologic correlates of sleep disturbances. Neurology, 42(6), 19-27.

Culebras, A. (1992b). Update on disorders of sleep and the sleep-wake cycle. Psychiatric Clinics of North America, 15(2), 467-489.

Degaute, J.-P., van de Borne, P., Linkowski, P. & Van Cauter, E. (1991). Quantitative analysis of the 24-hour blood pressure and heart rate patterns in young men. Hypertension, 18(2), 199-210.

Dement, W. C. (1994). History of sleep physiology and medicine. In Kryger, M. H., Roth, T. & Dement, W. C. (Eds.), Principles and practice of sleep medicine (2<sup>nd</sup> ed.) (pp. 3-15). Philadelphia: W. B. Saunders.

Dement, W. C., Miles, L. E. & Carskadon, M. A. (1980). White paper on sleep and aging. Journal of the American Geriatrics Society, 30(1), 25-50.

Feinsilver, S. H. & Hertz, G. (1993). Sleep in the elderly patient. Clinics in Chest Medicine, 14(3), 405-411.

Fisher, J. & McIsaac, P. (1995). Let sleeping residents lie. Canadian Nursing Home, 6(2), 19-22.

Flemons, W. W., Horne, S. G., Guilleminault, C. & Gillis, A. M. (1994). Cardiac function during sleep. In Kryger, M. H., Roth, T. & Dement, W. C. (Eds.), Principles and practice of sleep medicine (2<sup>nd</sup> ed.) (pp. 824-833). Philadelphia: W. B. Saunders.

Floyd, J. A. (1995). Another look at napping in older adults. Geriatric Nursing, 16(3), 136-138.

Floyd, J. A. (1996). Napping and the elderly is subject for replicating research. Michigan Nurse, 69(6), 12.

Folstein, M. F., Folstein, S. E. & McHugh, P. R. (1975). >Mini-mental state=: A practical method for grading the cognitive state of patients for the clinician. Journal of Psychiatric Research, 12, 189-198.

Friedman, L., Bliwise, D. L., Yesavage, J. A. & Salom, S. R. (1991). A preliminary study comparing sleep restriction and relaxation treatments for insomnia in older adults. Journal of Gerontology, 46(1), P1-8.

- Frisoni, G. B., de Leo, D., Rozzini, R., Bernadrdini, M., Dello Buono, M. & Trabucchi, M. (1992). Psychic correlates of sleep symptoms in the elderly. International Journal of Geriatric Psychiatry, 7, 891-898.
- Gander, P. H., Connell, L. J. & Graeber, R. C. (1986). Masking of the circadian rhythms of heart rate and core temperature by the rest-activity cycle in man. Journal of Biological Rhythms, 1(2), 119-135.
- Gentili, A., Weiner, D. K., Kuchibhatla, M. & Edinger, J. D. (1997). Factors that disturb sleep in nursing home residents. Aging: Clinical and Experimental Research, 9(3), 207-213.
- George, C. F. & Kryger, M. H. (1985). Sleep and heart rate control. Clinics in Chest Medicine, 6(4), 595-601.
- Gislason, T., Reynisdottir, H., Kristbjarnarson, H & Benediktsdottir, B. (1993). Sleep habits and sleep disturbances among the elderly: An epidemiological survey. Journal of Internal Medicine, 234, 31-39.
- Haimov, I., Laudon, M. Zisapel, N., Souroujon, M., Nof, D., Shlitner, A., Herer, P. & Tzischinsky, O. (1994). Sleep disorders and melatonin rhythms in elderly people. British Journal of Medicine, 309, 167.
- Harper, R. M., Frysinger, R. C., Zhang, J., Trelease, R. B. & Terreberry, R. R. (1988). In Lydic, R. & Biebuyck, J. F. (Eds), Clinical physiology of sleep (pp. 67-77). Bethesda, MD: American Physiological Society.
- Hays, J. C., Blazer, D. G. & Foley, D. J. (1996). Risk of napping: Excessive daytime sleepiness and mortality in an older community population. Journal of the American Geriatrics Society, 44(6), 693-698.

Hayter, J. (1983). Sleep behaviors in older adults. Nursing Research, 32(4), 242-246.

Hayter, J. (1985). To nap or not to nap?. Geriatric Nursing, 6, 104-106.

Haythorthwaite, J. A., Hegel, M. T. & Kerns, R. D. (1991). Development of a sleep diary for chronic pain patients. Journal of Pain and Symptom Management, 6(2), 65-72.

Herscovitch, J. & Broughton, R. (1981). Sensitivity of the Stanford Sleepiness Scale to the effects of cumulative partial sleep deprivation and recovery oversleeping. Sleep, 4(1), 83-92.

Hoch, C. C., Reynolds, C. R., & Houck, P. R. (1988). Sleep patterns in Alzheimer, depressed and healthy elderly. Western Journal of Nursing Research, 10(3), 239-256.

Hoddes, E., Zarcone, V., Smythe, H., Phillips, R. & Dement, W. C. (1973). Quantification of sleepiness: A new approach. Psychophysiology, 10(4), 431-436.

Hodgson, L. A. (1991). Why do we sleep?: Relating theory to nursing practice. Journal of Advanced Nursing, 16, 1503-1510.

Horne, J. A. (1983). Human sleep and tissue restitution: Some qualifications and doubts. Clinical Science, 65, 569-578.

Jacobs, D., Ancoli-Israel, S., Parker, L. & Kripke, D. F. (1989). Twenty-four hour sleep-wake patterns in a nursing home population. Psychology of Aging, 4(3), 352-356.

Jones, B. E. (1994). Basic mechanisms of sleep-wake states. In Kryger, M. H., Roth, T. & Dement, W. C. (Eds.), Principles and practice of sleep medicine (2<sup>nd</sup> ed.) (pp. 145-159). Philadelphia: W. B. Saunders.

Jonsson, P. V., Lipsitz, L. A., Kelley, M. & Koestner, J. (1990). Hypotensive responses to common daily activities in institutionalized elderly. Archives of Internal Medicine, 150, 1518-1524.

Khatri, I. M. & Freis, E. D. (1967). Hemodynamic changes during sleep. Journal of Applied Physiology, 22(5), 867-873.

Knowles, J. B., Coulter, M., Wahnnon, S., Reitz & MacLean, A. W. (1990). Variations in process S: Effects on sleep continuity and architecture. Sleep, 13(2), 97-107.

Livingston, G., Blizzard, B. & Mann, A. (1993). Does sleep disturbance predict depression in elderly people?: A study in inner London. British Journal of General Practice, 43, 445-448.

Lombardi, F., Malliani, A. & Pagani, M. (1997). Age-related changes in heart rate including sympathetic and parasympathetic tone. Cardiology in the Elderly, 5(1), 14-17.

MacWilliam, J. A. (1923). Blood pressure and heart action in sleep and dreams. The British Journal of Medicine, 2, 1196-1200.

Maczaj, M. (1993). Pharmacological treatment of insomnia. Drugs, 45(1), 44-55.

Newmann, J. & Broughton, R. (1991). Pupillometric assessment of excessive daytime sleepiness in narcolepsy-cataplexy. Sleep, 14(2), 121-129.

O'Rourke, D. J. & Burke, K. S. (2000). Redesigning nighttime care for personal care residents. Manuscript submitted for publication.

Parmeggiani, P. L. (1988). Thermoregulation during sleep from the viewpoint of homeostasis. In Lydic, R. & Biebuyck, J. F. (Eds), Clinical physiology of sleep (pp. 159-169). Bethesda, MD: American Physiological Society.

Parmeggiani, P. L. (1994). The autonomic nervous system in sleep. In Kryger, M. H., Roth, T. & Dement, W. C. (Eds.), Principles and practice of sleep medicine (2<sup>nd</sup> ed.) (pp. 194-203). Philadelphia: W. B. Saunders.

Pressman, M. R. & Fry, J. M. (1988). What is normal sleep in the elderly? Clinics in Geriatric Medicine, 4(1), 71-81.

Prinz, P. N., & Vitiello, M. V. (1993). Sleep loss in aging. Facts and Research in Gerontology, 7, 55-68.

Prinz, P. N., Vitiello, M. V., Raskind, M. A. & Thorpy, M. J. (1990). Geriatrics: Sleep disorders and aging. The New England Journal of Medicine, 323(8), 520-526.

Raiha, I, Seppala, M., Impivaara, O., Hyypa, M. T., Knuts, L.-R. & Sourander, L. (1994). Chronic illness and subjective quality of sleep in the elderly. Aging: Clinical and Experimental Research, 6(2), 91-96.

Rediehs, M. H., Reis, J. S. & Creason, N. S. (1990). Sleep in old age: Focus on gender differences. Sleep, 13(5), 410-424.

Regestein, Q. R. & Morris, J. (1987). Daily sleep patterns observed among institutionalized elderly residents. The Journal of the American Geriatrics Society, 35(8), 767-772.

Reynolds, C. F., Jennings, J. R., Hoch, C. C., Monk, D. J., Berman, S. R., Hall, F. T., Matzzie, J. V., Buysse, D. J. & Kupfer, D. J. (1991). Daytime sleepiness in the healthy 'old-old': A comparison with young adults. Journal of the American Geriatric Society, 39 (10), 957-962.

Richards, K. C., Curry, N., Lyons, W. & Todd, B. (1996). Cardiac dysrhythmia during sleep in the critically ill: A pilot study. American Journal of Critical Care, 5(1), 26-33.

Rogers, A. E., Caruso, C. C. & Aldrich, M. S. (1993). Reliability of sleep diaries for assessment of sleep-wake patterns. Nursing Research, 42(6), 368-371.

Roth, T. Roehrs, T. A., Carskadon, M. A. & Dement, W. C. (1994). Daytime sleepiness and alertness. In Kryger, M. H., Roth, T. & Dement, W. C. (Eds.), Principles and practice of sleep medicine (2<sup>nd</sup> ed.) (pp. 40-49). Philadelphia: W. B. Saunders.

Sandford, S. (1983). Sleep and the cardiac patient. Cardiovascular Nursing, 19(5), 19-24.

Schnelle, J. F., Alessi, C. A., Ouslander, J. G. & Simmons, S. F. (1993). Noise and predictors of sleep in a nursing home environment. Facts and Research in Gerontology, 7, 89-99.

Schnelle, J. F., Cruise, P. A., Alessi, C. A., Al-Samarrai, N. & Ouslander, J. G. (1998). Individualizing nighttime incontinence care in nursing home residents. Nursing Research, 47(4), 197-204.

Schnelle, J. F., Ouslander, J. G., Simmons, S. F., Alessi, C. A. & Gravel, M. D. (1993a). The nighttime environment, incontinence care and sleep disruption in nursing homes. Journal of the American Geriatrics Society, 41(9), 910-914.



Schnelle, J. F., Ouslander, J. G., Simmons, S. F., Alessi, C. A. & Gravel, M. D. (1993b). Nighttime sleep and bed mobility among incontinent nursing home residents. Journal of the American Geriatrics Society, 41(9), 903-909.

Seppala, M., Rajala, T. & Sourander, L. (1993). Subjective evaluation of sleep and the use of hypnotics in nursing homes. Aging: Clinical and Experimental Research, 5(3), 199-205.

Siegel, J. M. (1994). Brainstem mechanisms generating REM sleep. In Kryger, M. H., Roth, T. & Dement, W. C. (Eds.), Principles and practice of sleep medicine (2<sup>nd</sup> ed.) (pp. 125-143). Philadelphia: W. B. Saunders.

Smirne, S., Ferini-Stambi, L., Montanari, C. & Canal, N. (1987). Age-related modifications of heart rate during sleep-wakefulness cycle. Functional Neurology, 2(4), 465-470.

Smith, O. A. (1988). Sleep and other behavioral states reflected in cardiovascular response patterns. In Lydic, R. & Biebuyck, J. F. (Eds), Clinical physiology of sleep (pp. 53-65). Bethesda, MD: American Physiological Society.

Snyder-Halpern, R. & Verran, J. A. (1987). Instrumentation to describe subjective sleep characteristics in healthy subjects. Research in Nursing and Health, 10, 155-163.

Spenceley, S. M. (1993). Sleep inquiry: A look with fresh eyes. Image: Journal of Nursing Scholarship, 25(3), 249-256.

Swift, C. G. & Shapiro, C. M. (1993). Sleep and sleep problems in elderly people. British Journal of Medicine, 306, 1468-1471.

Synder, F., Hobson, J. A., Morrison, D. F. and Goldfrank, F. (1964). Changes in respiration, heart rate and systolic blood pressure in human sleep. Journal of Applied Physiology, 19, 417-422.

Tombaugh, T. N. & McIntyre, N. J. (1992). The mini-mental state examination: A comprehensive review. Journal of the American Geriatric Society, 40(9), 922-935.

Tri-Council Working Group. (1998). Tri-council policy statement: Ethical conduct for research involving humans. Ottawa: Government of Canada.

Turpin, G. (1986). Psychophysiology of sleep. Nursing (Oxford), 3, 313-320.

Wauquier, A. & van Sweden, B. (1992). Aging of care and optional sleep. Biological Psychiatry, 31, 866-880.

Webb, W. B. (1988). An objective behavioral model of sleep. Sleep, 11(5), 488-496.

Webb, W. (1989). Age-related changes in sleep. Clinics in Geriatric Medicine, 5(2), 275-287.

Webb, W. B. & Aber, W. R. (1984). Relationships between sleep and retirement-nonretirement status. International Journal of Aging and Human Development, 20(1), 13-19.

Welch, A. J. & Richardson, P. C. (1973). Computer sleep state classification using heart rate data. Electroencephalography and Clinical Neurophysiology, 34, 145-152.

Wewers, M. E. & Lowe, N. K. (1990). A critical review of visual analogue scales in the measurement of clinical phenomena. Research in Nursing and Health, 13, 227-36.

Wooten, V. (1992). Sleep disorders in geriatric patients. Clinics in Geriatric Medicine, 8(2), 427-439.

Zemaityte, D., Varoneckas, G. & Sokolov, E. (1984). Heart rhythm control during sleep. Psychophysiology, 21(3), 279-289..

## Appendix A

## Interactions of Brain Structures in a Homeostatic Model of Sleep Control

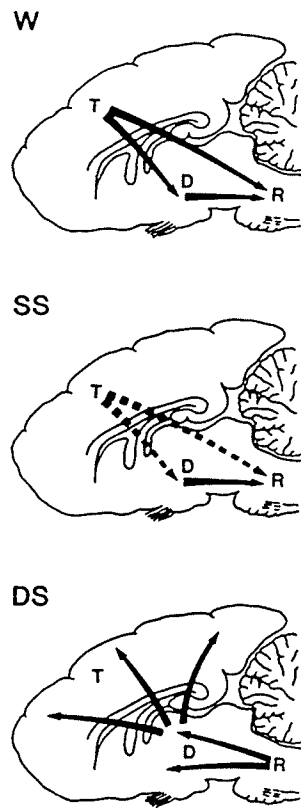


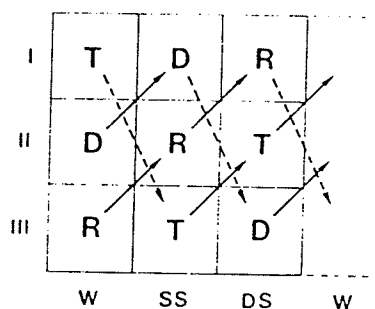
FIG. 3. Schematic representation of state-dependent changes in the interaction between telencephalic (T), diencephalic (D), and rhombencephalic (R) structures. Homeostatic control of hypothalamus on brain stem and spinal effector mechanisms is affected by telencephalic influences during wakefulness (W). During synchronized sleep (SS), stability of automatic functions is maximal as a result of the suspension of the functional dominance of telencephalic structures. Diminished telencephalic influence is schematized by dashed lines. During desynchronized sleep (DS) rhombencephalic structures exert a dominant influence. Strong rhombencephalic influence also is postulated to break the hierarchical coherence between the morphological and functional organizations of the encephalon.

(Parmeggiani, 1988, p. 165)

## Appendix B

# Ranking of Functional Dominance of Brain Structures in a Homeostatic Model of Sleep Control

FIG. 4. Ranking (*ordinate*) of functional dominance of telencephalon (T), diencephalon (D), and rhombencephalon (R) during wakefulness (W) and sleep (SS, synchronized sleep; DS, desynchronized sleep). Relative ranking of structures was inferred from various measures of waking and sleeping phenomenology (see text). Permutations in the pattern of functional organization are postulated to occur in an orderly way, as indicated by *arrows*. [From Parmeggiani (25).]



(Parmeggiani, 1988, p. 166)

## Appendix C

**Stanford Sleepiness Scale**

ID#: \_\_\_\_\_

**At noon today, please circle a number beside the line that best describes how you feel:**

- 1     Feeling active and vital; alert; wide awake
- 2     Functioning at a high level; but not at peak; able to concentrate
- 3     Relaxed; awake; not at full alertness; responsive
- 4     A little foggy; not at peak; let down
- 5     Fogginess; beginning to lose interest in remaining awake; slowed down
- 6     Sleepiness; prefer to be lying down; fighting sleep; woozy
- 7     Almost in reverie; sleep onset soon; lost struggle to remain awake

(Hoddes et al, 1973)

Appendix D

VSH Sleep Scale

ID#: \_\_\_\_\_

Mid-Sleep Awakenings

- Number of awakenings during the sleep period

\_\_\_\_\_

Didn't wake

Awake off and on

Score - \_\_\_\_

Movement during Sleep

- Estimate of the amount of movement during sleep

\_\_\_\_\_

Didn't move

Tossed all night

Score - \_\_\_\_

Total Sleep Period

- Total time from settling down for sleep to awakening in the morning

\_\_\_\_\_

No sleep

Ten hours sleep

Score \_\_\_\_

Sleep Latency

- Amount of time from settling down to sleep until falling asleep

\_\_\_\_\_

Fell asleep immediately

Didn't sleep at all

Score - \_\_\_\_

Soundness of Sleep

- Estimate of depth

---

Slept lightly

Slept deeply

Score \_\_\_\_\_

Rested Upon Awakening

- Estimate of how rested you feel on awakening

---

Awoke exhausted

Awoke refreshed

Score \_\_\_\_\_

Method of Awakening

- Spontaneity with which you awoke in the morning

---

Awoke abruptly

Awoke spontaneously

Score \_\_\_\_\_

Subjective Quality of Sleep

- Estimate of sleep along dimensions of satisfaction, quality and disturbance

---

Bad night

Good night

Score \_\_\_\_\_

Total Score: /80

(Snyder-Halpern & Verran, 1987)



## Appendix E

## Sleep Diary

ID#: \_\_\_\_\_

Date: \_\_\_\_\_

Time Monitor On: \_\_\_\_\_ Time Monitor Off: \_\_\_\_\_

**For all entries, please record the time in hours and minutes.**

**Before you get into bed, record:**

Time you started sitting on the bed/chair for 5 minutes: \_\_\_\_\_

What time did you get into bed for the night? \_\_\_\_\_

**If you wake up during the night, record the time of each awakening:**

\_\_\_\_\_

\_\_\_\_\_

**When you awoke in the morning for the day, record:**

What time did you fall asleep last evening: \_\_\_\_\_

**For each time you woke up in the night, how long did it take to fall asleep again?**

\_\_\_\_\_

\_\_\_\_\_

What time did you wake up in the morning for the day? \_\_\_\_\_

What time did you get up out of bed for the day? \_\_\_\_\_

Researcher use:

Any equipment concerns (functioning, comfort, skin condition)?

---

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---

Any unusual occurrences overnight?

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---

---

Any identified causes of sleep disturbance?

---

---

---

Use of sleep aid medications previous night?

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---

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Appendix F

Demographic Information Form

Study ID#: \_\_\_\_\_

Age: \_\_\_\_\_

Length of Stay (on unit in days): \_\_\_\_\_

MMSE score: \_\_\_\_\_ Date completed: \_\_\_\_\_ By Whom: \_\_\_\_\_

Medical Diagnoses:

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Current Medications (including sleep aid meds):

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Usual bedtime: \_\_\_\_\_ Usual time to get up in AM: \_\_\_\_\_

## Appendix G

## Consent Form

You are invited to participate in the research project "Comparison of Subjective Reports and Heart Rate Profiles during Sleep in Older Institutionalized Adults". This study is being conducted by Deanne O'Rourke, R.N., B.N., graduate student from the Faculty of Nursing, University of Manitoba. The purpose of this study is to explore the use of heart rate monitoring as a method to determine quality and quantity of sleep. This study has received ethical approval from the Faculty of Nursing Ethical Review Committee.

Your signature below indicates that you agree to participate and you understand the following details about your involvement in the study:

- The above named researcher will collect information from your health record on your age, length of stay on your unit, a memory test score, medical diagnoses and any medication that you are taking.
- If a memory test score is not available from your chart, you will be given a short 10-minute test of your memory and concentration. The researcher will also check your ability to see typed print and sample your handwriting. You will be asked about your usual bedtime routines and time of getting out of bed in the morning.
- You will be asked to try on the heart monitor equipment and wear it for a short period of time (15 to 30 minutes) so you have the chance to see how it feels.
- The researcher will return on a pre-arranged evening before your bedtime and place the heart rate monitor around your chest and a watch around one wrist that you would wear during the night. Before going to bed, you will be asked to record on a sleep diary what time you got into bed for the night. In the morning, you will be asked to write in the diary what time you fell asleep the previous evening, how many times and how long you were awake during the night, what time you woke up in the morning and what time you rose from bed for the day.
- The researcher will return that morning to remove the heart rate monitor, review your sleep diary and to ask you a short 10-15 minute questionnaire about your sleep the past night. At this time, the researcher will also examine your skin for any sign of breakdown related to the monitor. If any exist, your nurse will be notified to treat the skin condition.
- At noontime that same day, you will be asked to answer a one-question scale that the researcher will leave with you that determines how sleepy you feel at that time.

All of the information collected in the study will be kept strictly confidential. Your name will not be used on any reports about the study or in future publications. Only the researcher and her advisor will have access to the information.

While participation in this study may have minimal benefit to you personally, it is expected that the shared experiences of people like yourself will offer a better understanding of the use of heart rate monitors during sleep in older persons. The costs

to you involve the time you spend filling out the sleep diary, wearing the heart rate monitor overnight and answering the sleep-related questions the next day.

Participation in this study is completely voluntary. You are under no obligation to participate and you may withdraw from the study at any time. If you decide not to participate in the study, this will not affect your present or future health care in any manner. If you have any questions about the study, you may ask them at any time during the study or by calling the researcher or her adviser at the Faculty of Nursing at the numbers listed below. You will be given a copy of this consent form. If you wish, a summary of the study's findings will be sent to you.

I recognize the importance of your participation and thank you.

Resident Signature: \_\_\_\_\_ Date: \_\_\_\_\_

I have provided a thorough verbal explanation of the research study and content of this consent form to the above resident:

Researcher Signature: \_\_\_\_\_ Date: \_\_\_\_\_

Deanne O'Rourke, R.N., B.N.  
Graduate Student  
Faculty of Nursing  
University of Manitoba  
222-1255 (home)  
935-8394 (pager)

Lorna Guse, R.N., Ph.D.  
Thesis Chair/Advisor  
Faculty of Nursing  
University of Manitoba  
474-6220

## Appendix H

## Frequencies of Medical Diagnoses Appearing in the Health Record

<b>Medical Diagnosis</b>	<b>Frequency (%)</b>	<b><u>n</u></b>
Cerebral vascular accident/Transient ischemic attack	47.8	11
Osteoarthritis	39.1	9
Congestive heart failure	34.8	8
Depression	30.4	7
Osteoporosis	30.4	7
Chronic obstructive pulmonary disease	30.4	7
Hypertension	30.4	7
Sensory disorders	26.1	6
Diabetes mellitus	26.1	6
Hip fracture/replacement	21.7	5
Ischemic heart disease	17.4	4
Peripheral vascular disorders	13.0	3
Gastrointestinal disorders	13.0	3
Parkinson Disease	13.0	3
Cancer	13.0	3
Myocardial infarction	8.7	2
Angina	8.7	2
Kyphoscoliosis	8.7	2
Anxiety disorders	8.7	2
Seizure disorder	8.7	2
Glaucoma	8.7	2
Compressions fractures to spine	8.7	2
Other	69.6	16

N =23

## Appendix I

## Frequencies of Regular and PRN Medications Recorded in the Health Record

<b>Medication Classification</b>	<b>Frequency (%)</b>	<b><u>n</u></b>
Bowel aid/laxative	69.5	16
Diuretic	65.2	15
Analgesic – non-narcotic	52.2	12
Antidepressant	52.2	12
Vitamin/mineral supplement	47.8	11
Cardiac – anti-anginal/nitrate	43.5	10
Anti-thrombolytic (ASA, Coumadin)	39.1	9
Anti-hypertensive – ACE inhibitor	39.1	9
Analgesic – narcotic	34.8	8
Anxiolytic	30.4	7
Bronchodilator	21.7	5
Cardiac – anti-arrhythmic	21.7	5
GI medications (H <sub>2</sub> blocker, motility)	21.7	5
NSAID	17.4	4
Cardiac – B-blocker	17.4	4
Anti-osteoporotic	13.0	3
Anti-emetic	13.0	3
Electrolyte replacement	13.0	3
Steroid	13.0	3
Anti-parkinson	13.0	3
Other	95.7	22

N =23

## Appendix J

## Record of Instrument Artifacts removed from the HR Data

Study ID	Cells Removed	Comments
ID#1	- 0 values: present between cells 120-1344	- loss of contact likely due to interference from resident's laptop computer used prior to bed
ID#2	- 0 values: cells 6052-6153 (8.4 minutes) - high artifact: cell 6352	- artifacts coincide with movement and waking in the AM
ID#3	- 0 values: cells 110-114, 189-193, 314-325, 333-341, 2097-2148	- loss of contact related to pre-bedtime activities and transfer into bed at hs.
ID#4	No adjustments needed	
ID#5	- 0 values: cells 337-345, 5213-5217, 7504-7515, 7648-7652, 7692-7717, 7722-7730, 7749-7762, 7771-7788, 7800-7824 - high artifacts: cells 5098-5099, 7386-7389	- artifacts coincide with one WASO and AM waking/activity
ID#6	- 0 values: cells 81-2, 107-120, 123-127, 145-146, 149-155, 630-646	- loss of monitor contact likely related to pre-bedtime movement
ID#7	- 0 values: cells 151-155, 171-212, 215-228, 278-287, 313-321, 335-339, 350-354	- loss of monitor contact likely related to pre-bedtime movement
ID#8	- 0 values: cells 1812-1819, 1864-1869, 1929-1938, 7153-7175, 7185-7207, 7293-7299, 7320-7325 - high artifacts: cells 5846-5847, 6003-6004	- loss of monitor contact likely related to AM movement once out of bed. - explanation of high artifacts may be movement in sleep
ID#9	- 0 values: cells 4047-4111 - 'plateau data': cells 4477-5102 (52 minutes)	- squared appearance of data in the HR profile suggests the recording of inaccurate HR values likely related to poor contact between the electrodes and skin
ID#10	- 0 values: cells 36-37, 88-89, 1011-1013, 3110-3114, 3117-3120, 7114-7116, 7238-7247, 7540-7549	- appears to coincide with SL movement and after rising from bed in the AM.

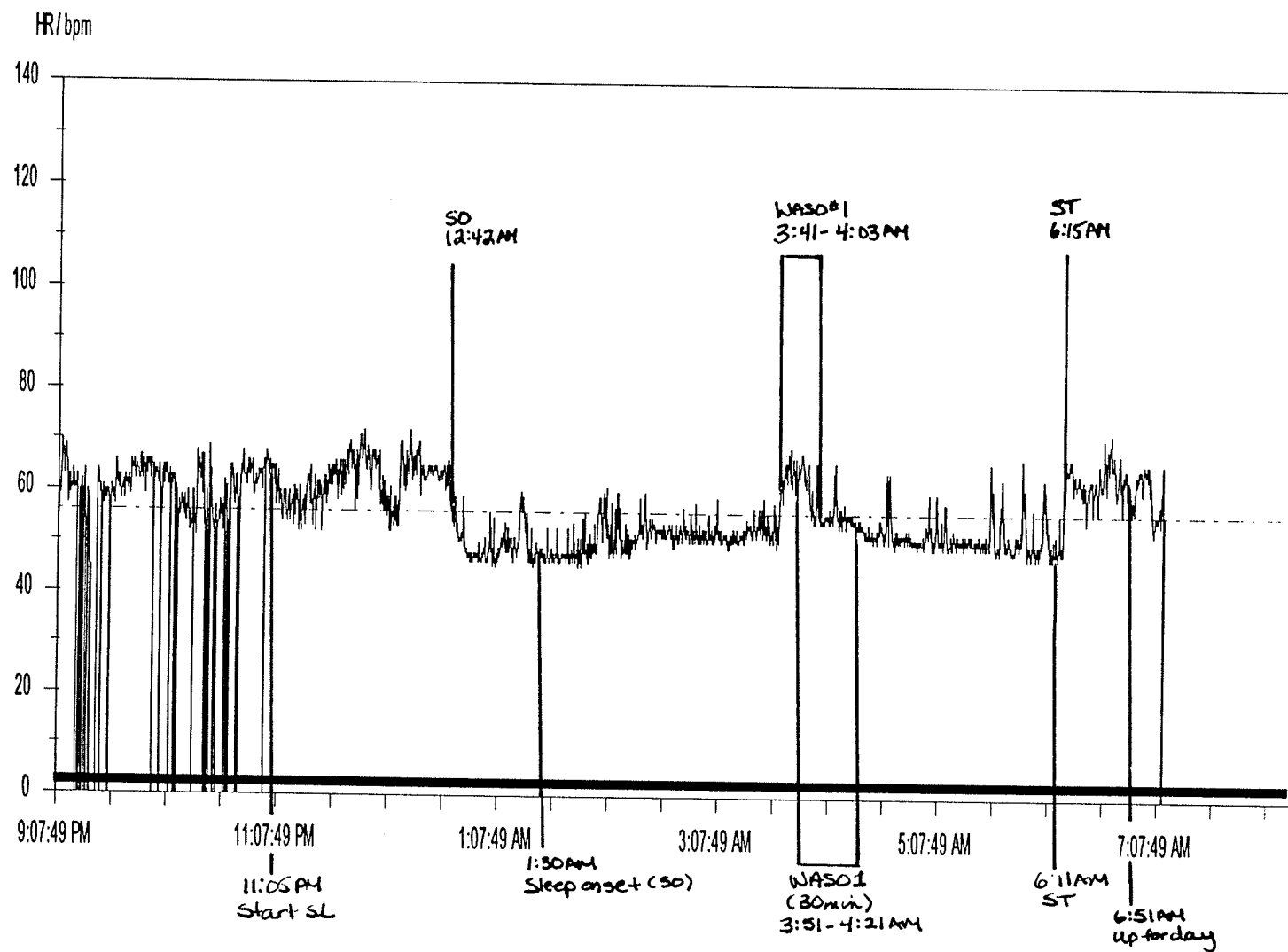


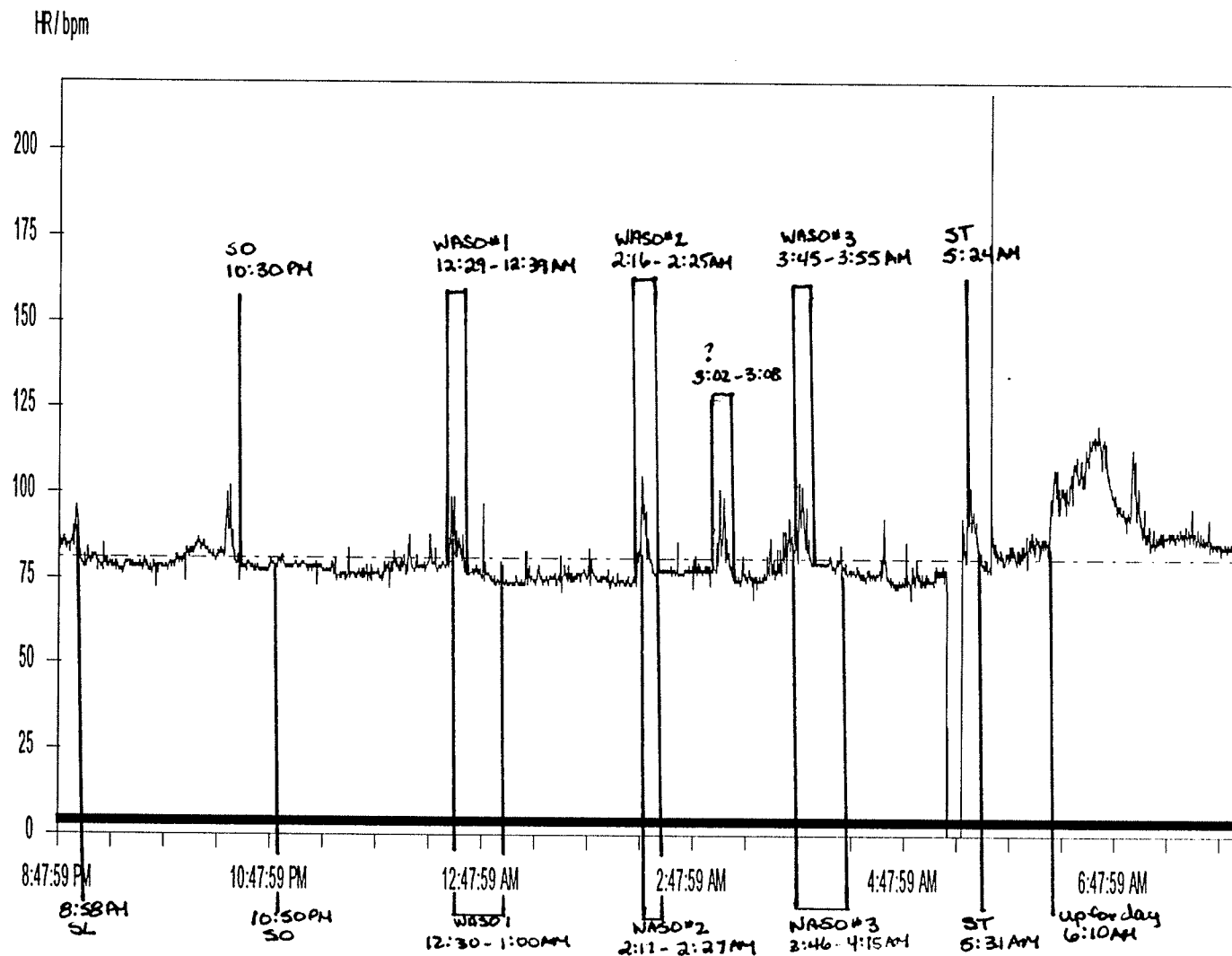
Study ID	Cells Removed	Comments
ID#11	No adjustments needed	
ID#12	No adjustments needed	
ID#13	- 0 values: cells 374-382, 389-395, 1931-1941, 2033-2043, 6833-6836, 7035-7038	- 0 values coincide with pre-bedtime movement and activity in the AM once out of bed
ID#14	- 0 values: cells 319-327	- 0 values coincide with getting into bed at hs
ID#15	- 0 values: cells 149-156, 201-208, 406-412, 418-423, 438-444, 514-520, 523-526, 2169-2176	- 0 values coincide with pre-bedtime activity and SL movement
ID#16	- 0 values: cells 1438-1443 - high artifact: cells 5478-5481	- 0 values associated with pre-bedtime movement
ID#17	- high artifacts: cells 506, 3768-3769 - low artifacts: cells 1379-1387, 4065-4089, 4657-4671, 4762-4767, 4792-4817, 4987-5012, 6523-6524 - 0 values: cells 3994-4009, 4052-4064, 4090-4098, 4457-4477, 4502-4618, 4653-4656, 4753-4761, 4768-4775, 4818-4836, 4945-4959, 4977-4986, 5032-5056, 5077-5093, 6757-6773, 6916-6927 - cells 5164-6184 (85 minutes) were removed and not included in analysis	- largest number of alterations done on this profile - monitor artifacts do not appear to be related to movement or awakenings - electrodes may have lost contact with the skin due to movement of the chest strap or inadequate amounts of conduction jelly
ID#18	No adjustments needed	
ID#19	- high artifact: cells 6746-6747	- no obvious cause of artifact noted on profile
ID#20	No adjustments needed	
ID#21	- 0 values: cells 7235-7241	- 0 values coincide with rising from bed in the AM

Study ID	Cells Removed	Comments
ID#22	- 0 values: cells 5942-5963, 7064-7068, 7538-7543, 7548-7559, 7561-7569, 7571-7575 - high artifacts: cells 1675, 1773, 1978-1979, 3360, 3543-3544, 4567-4568, 7524-7525	- no obvious explanation or pattern is observed on the profile
ID#23	0 value: cell 3690	- no obvious explanation for 0 value in HR profile

Note: Cell number corresponds to location of value in Excel spreadsheet. Each cell represents a 5 second interval of time (i.e., 12 cells = 1 minute)

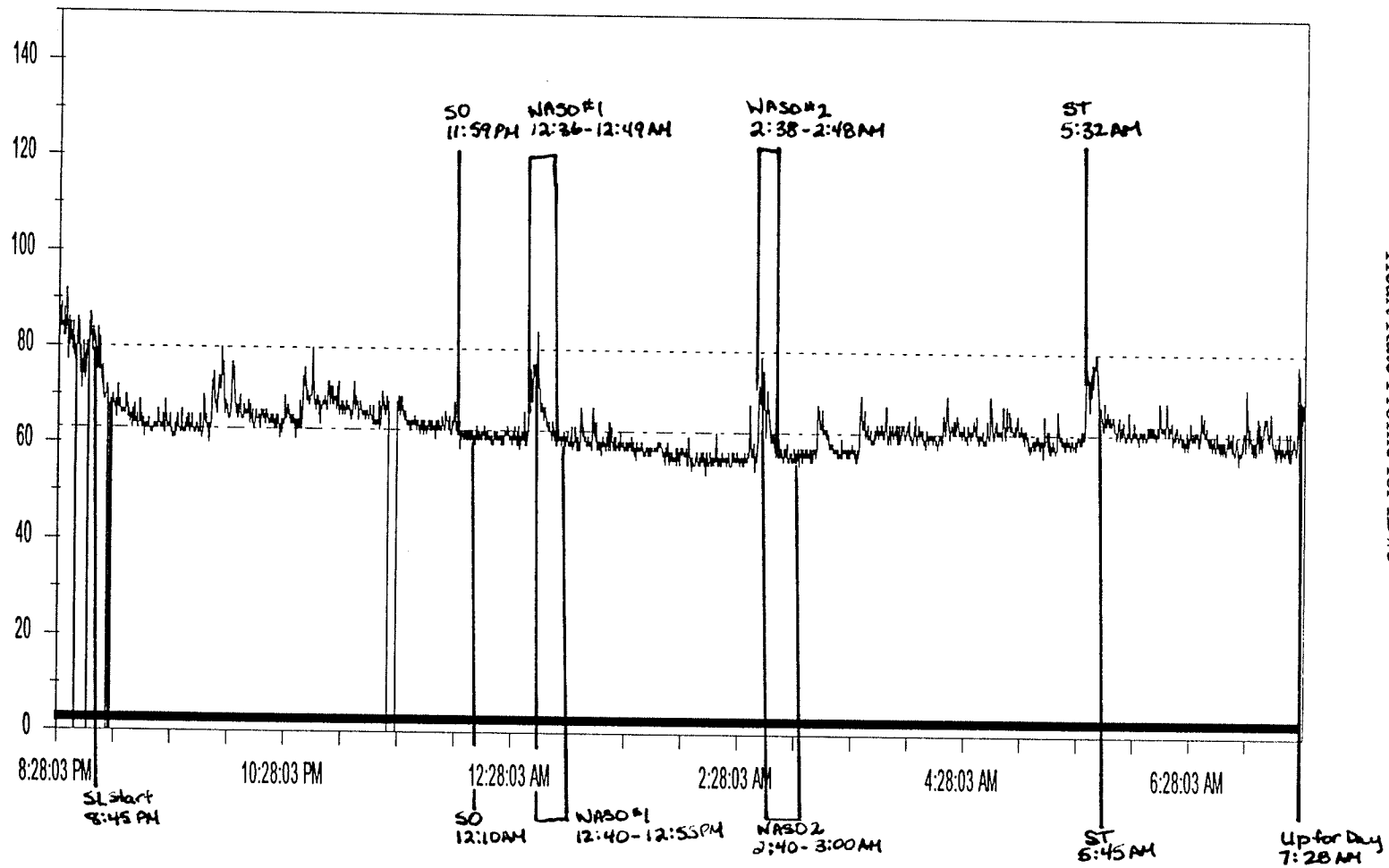
Heart Rate Profile for ID#1





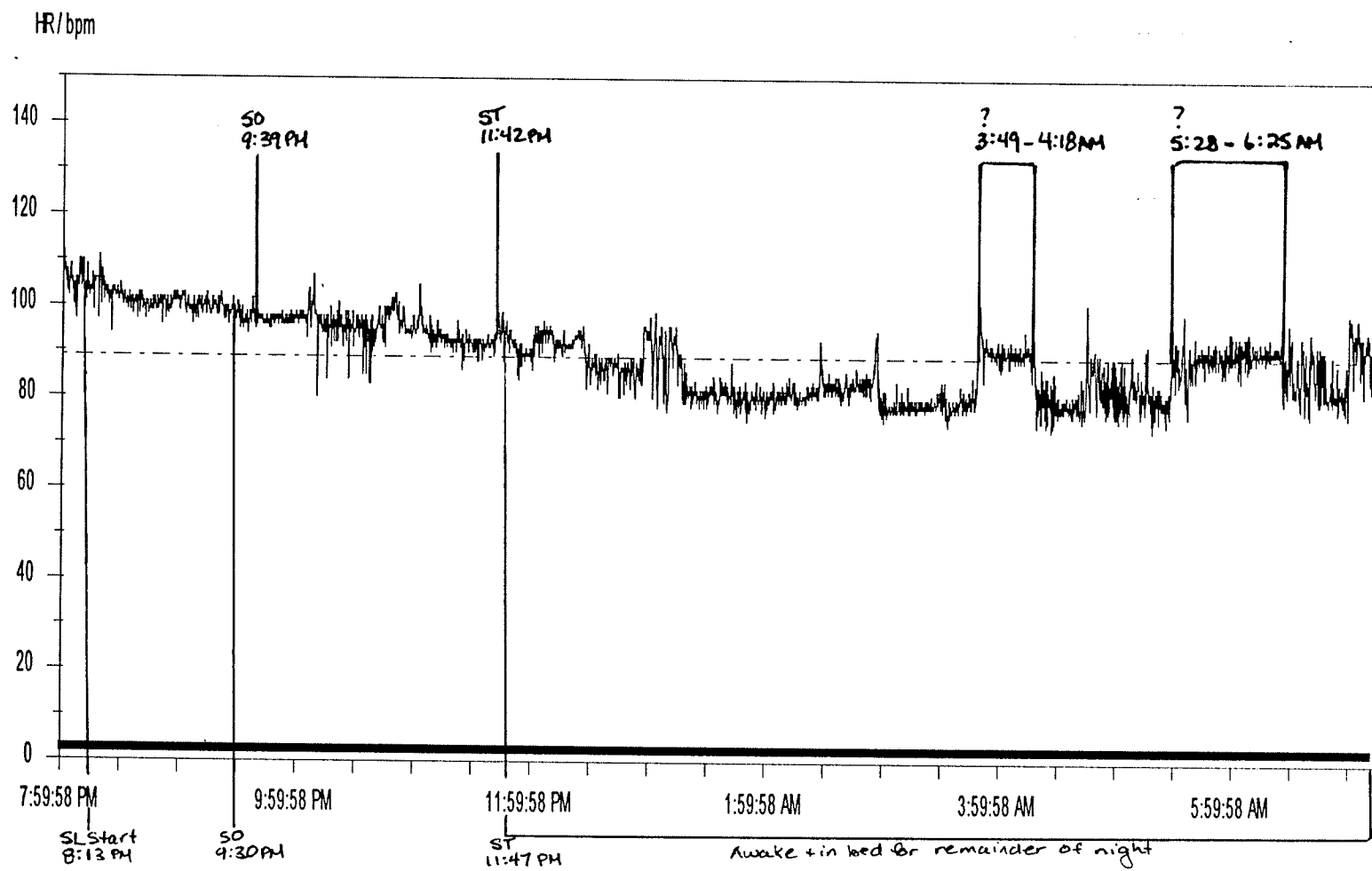
Heart Rate Profile for ID#2

HR/bpm

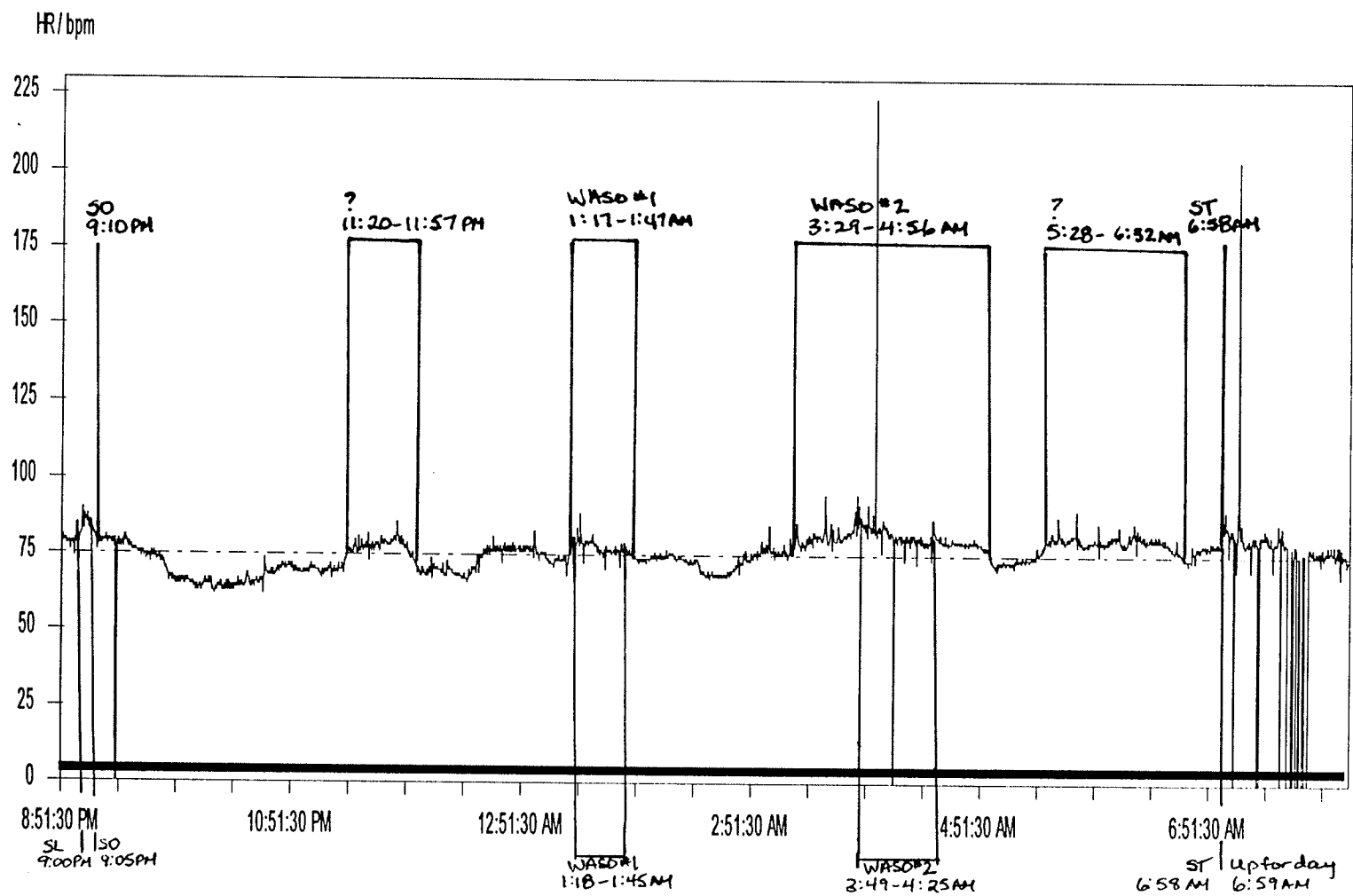


Heart Rate Profile for ID#3

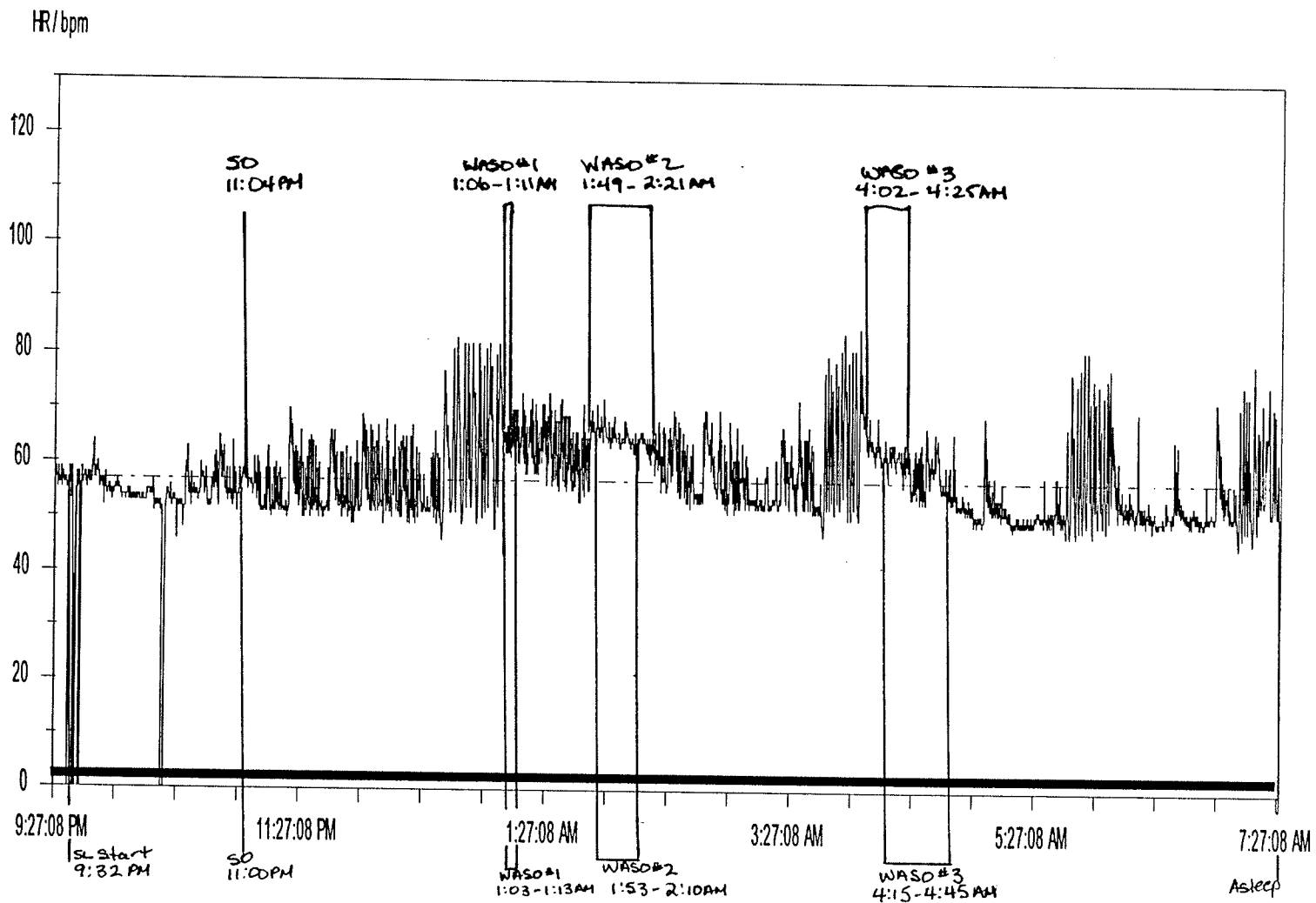
Sleep 107



Heart Rate Profile for ID#4

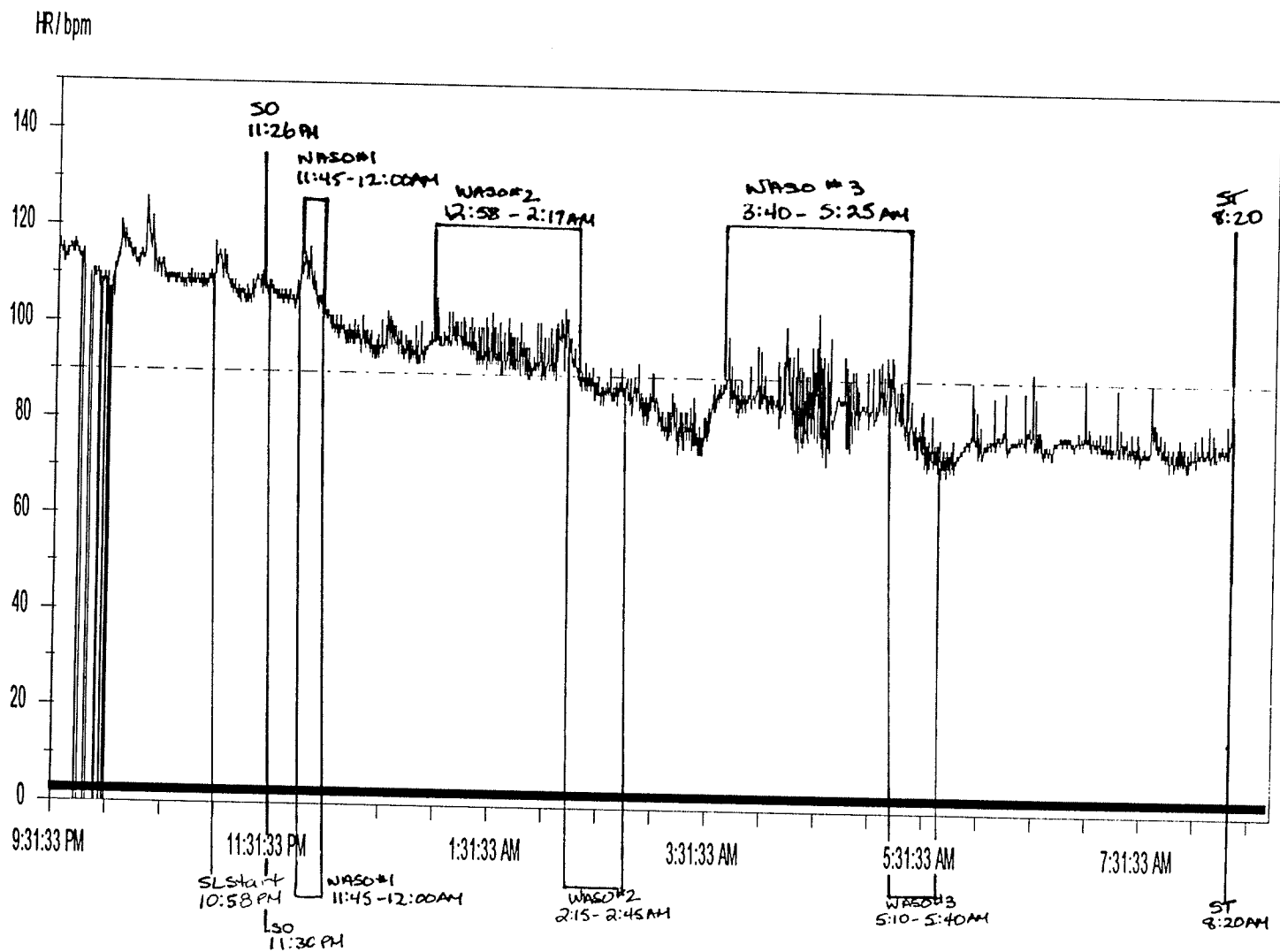


Heart Rate Profile for ID#5

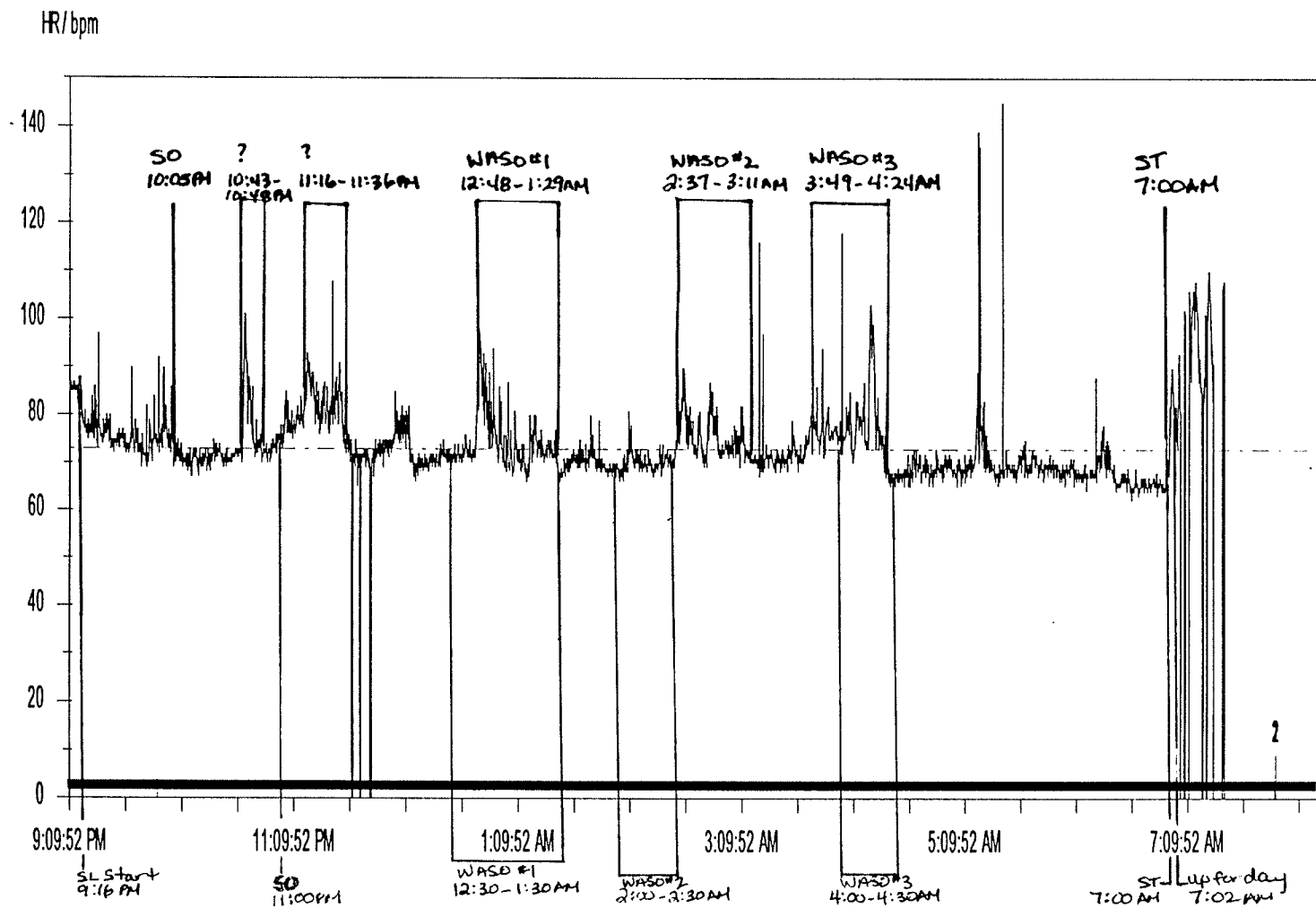


Heart Rate Profile for ID#6



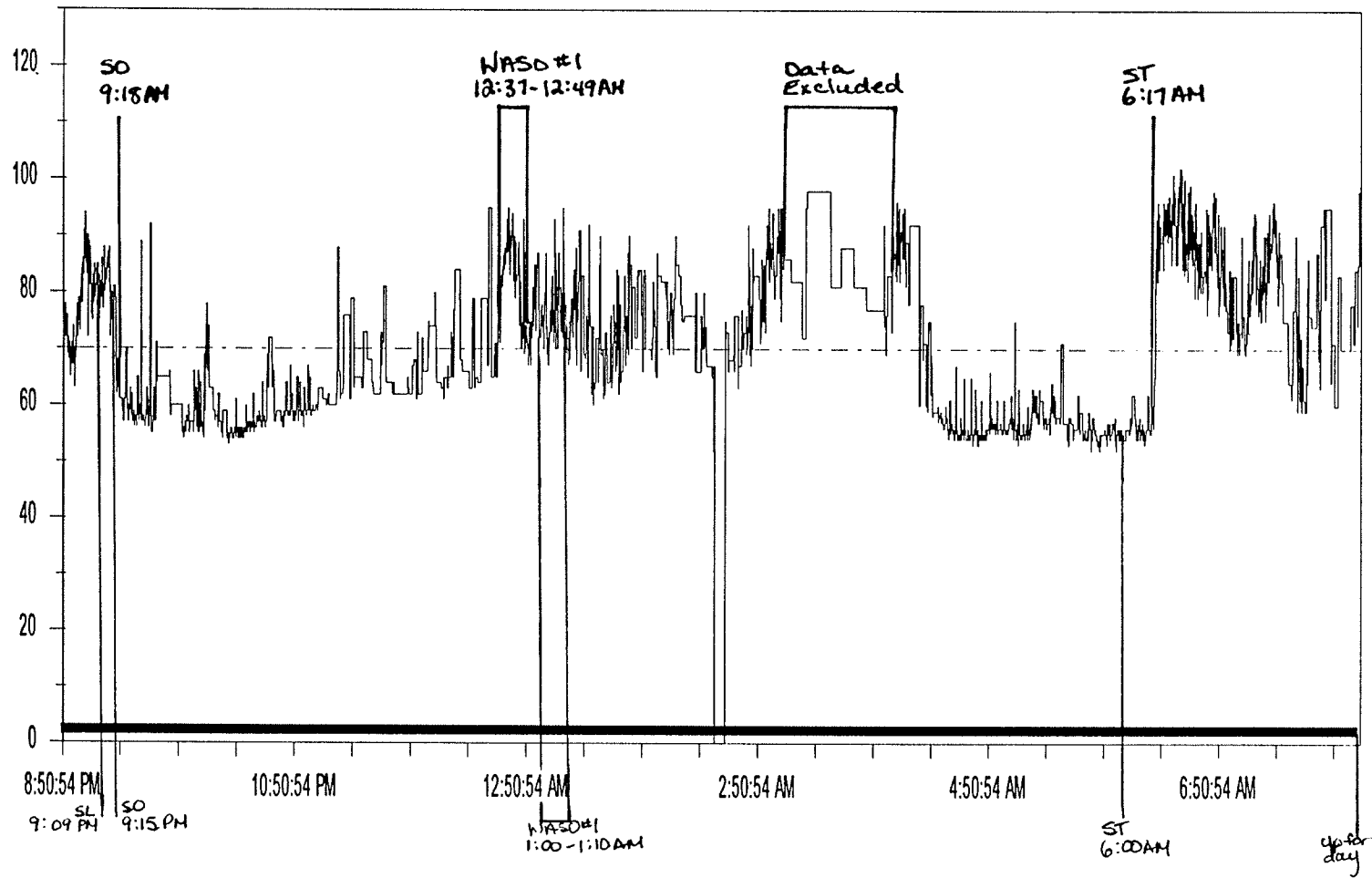


Heart Rate Profile for ID#7



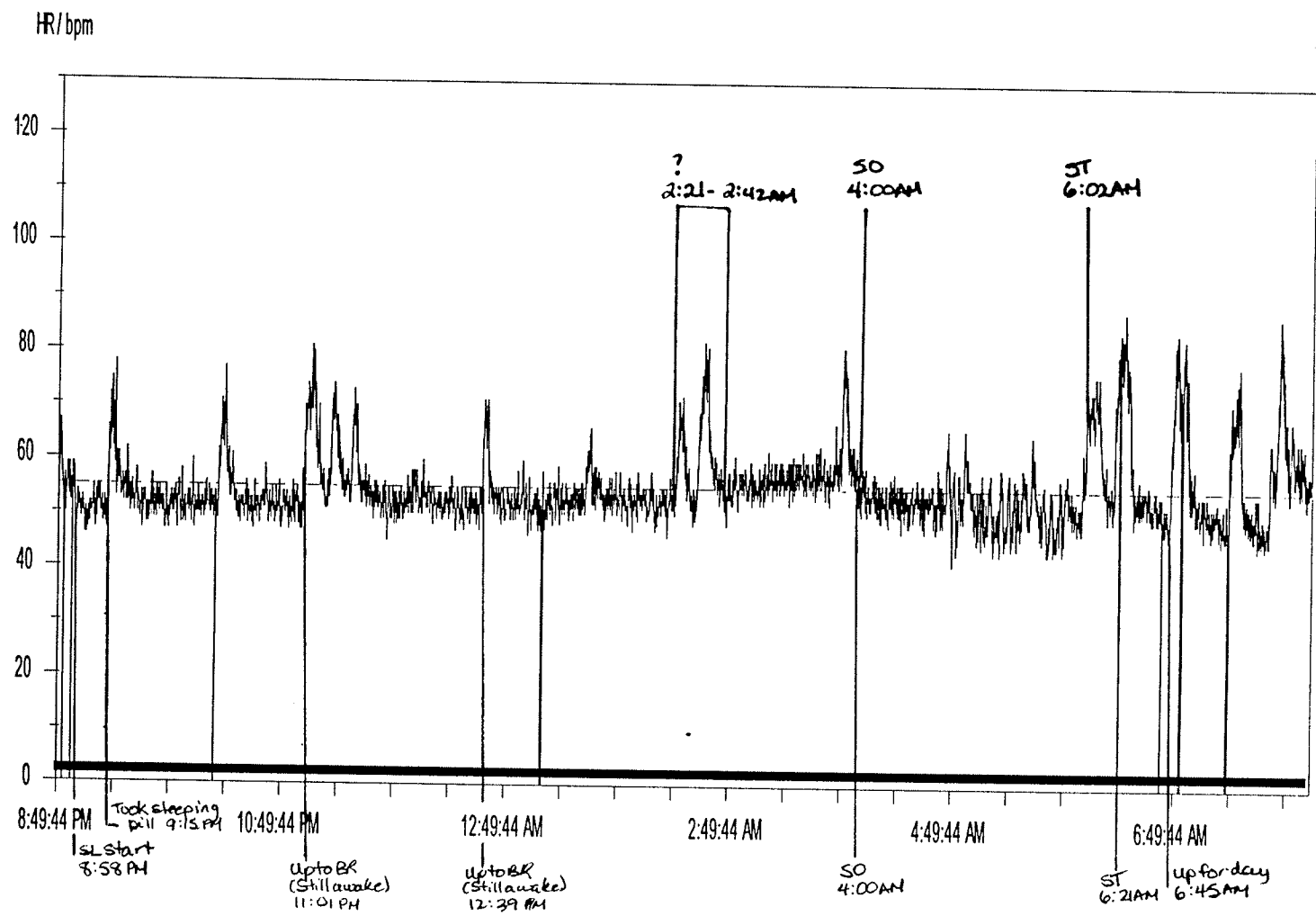
Heart Rate Profile for ID#8

HR/bpm

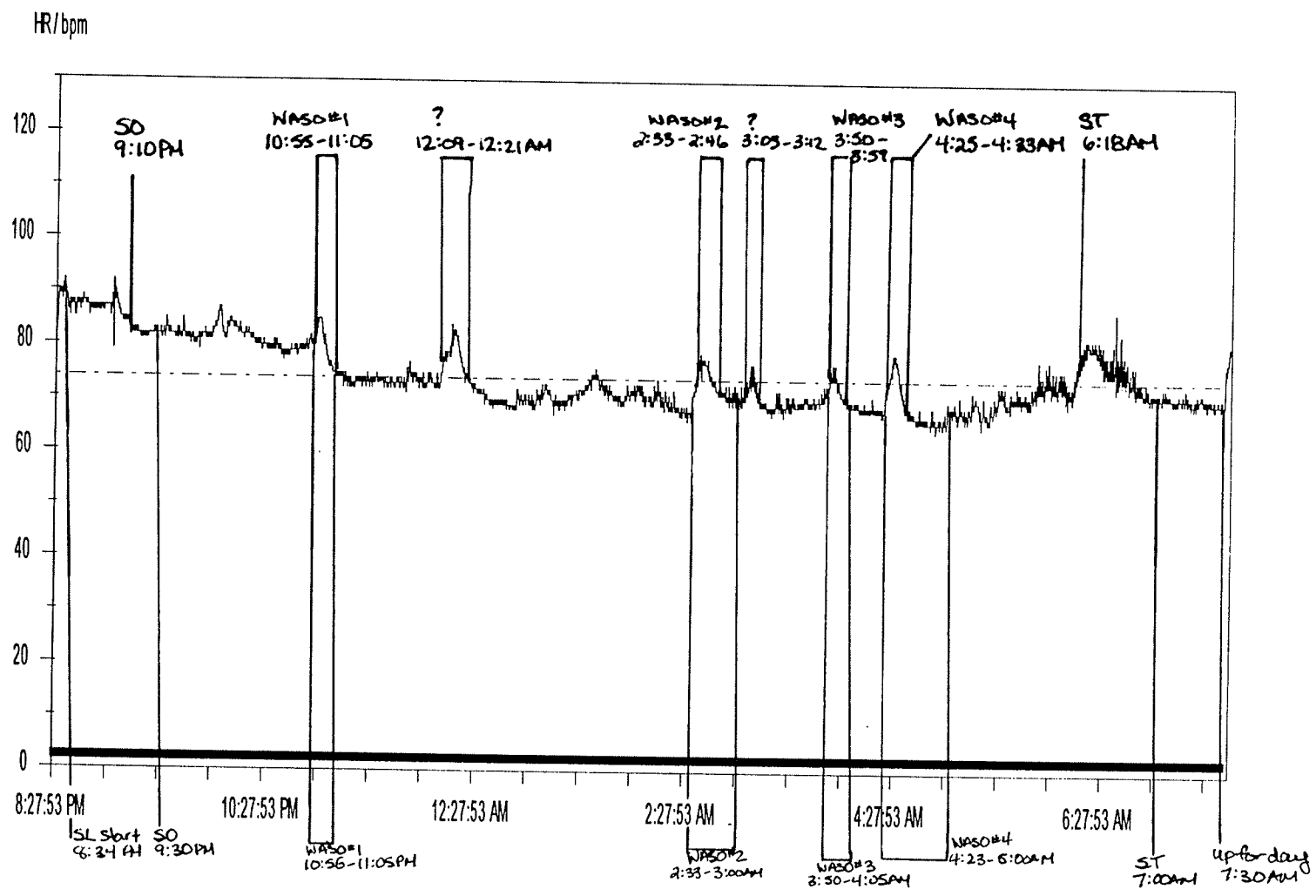


Heart Rate Profile for ID#9

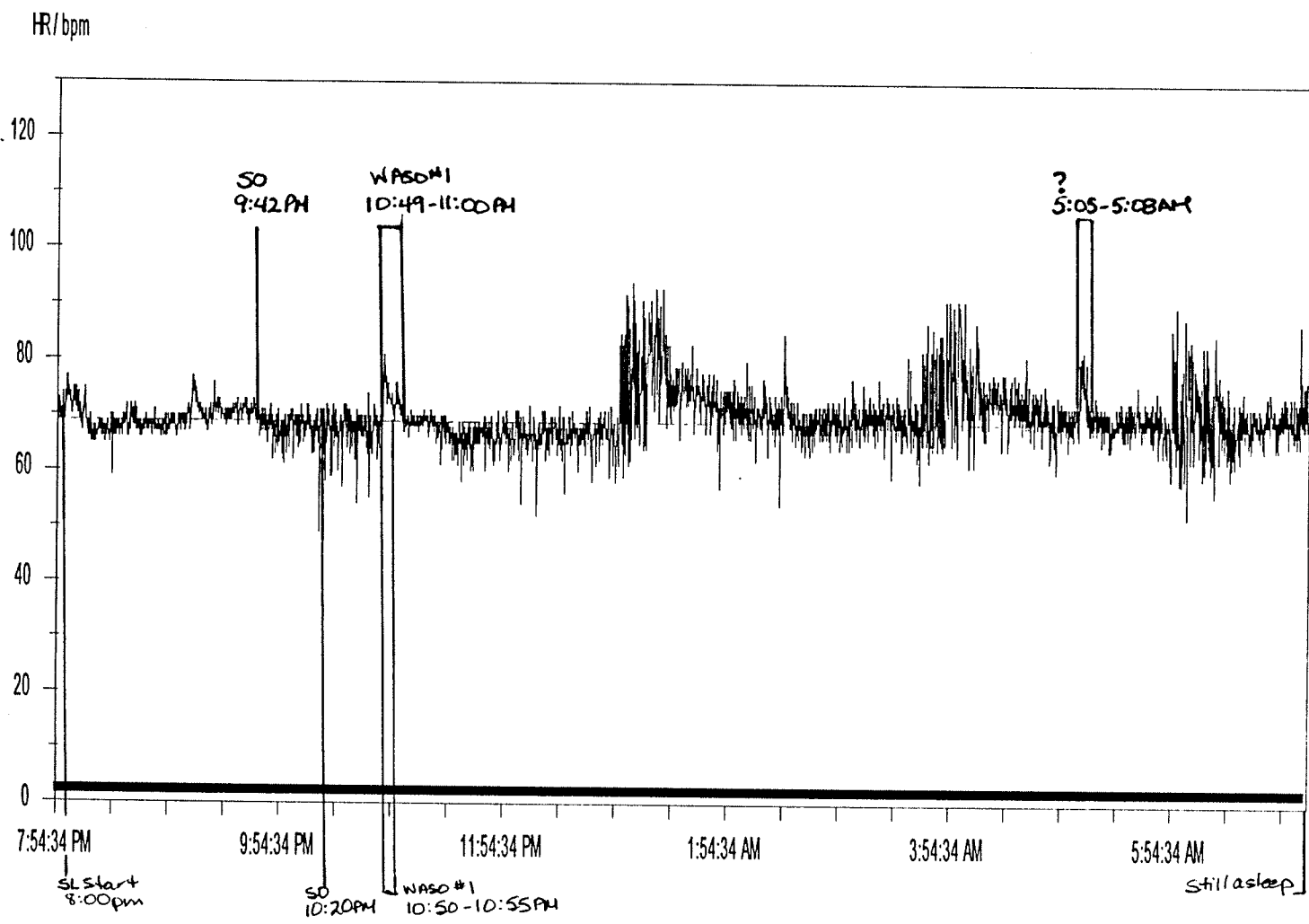
Sleep 113



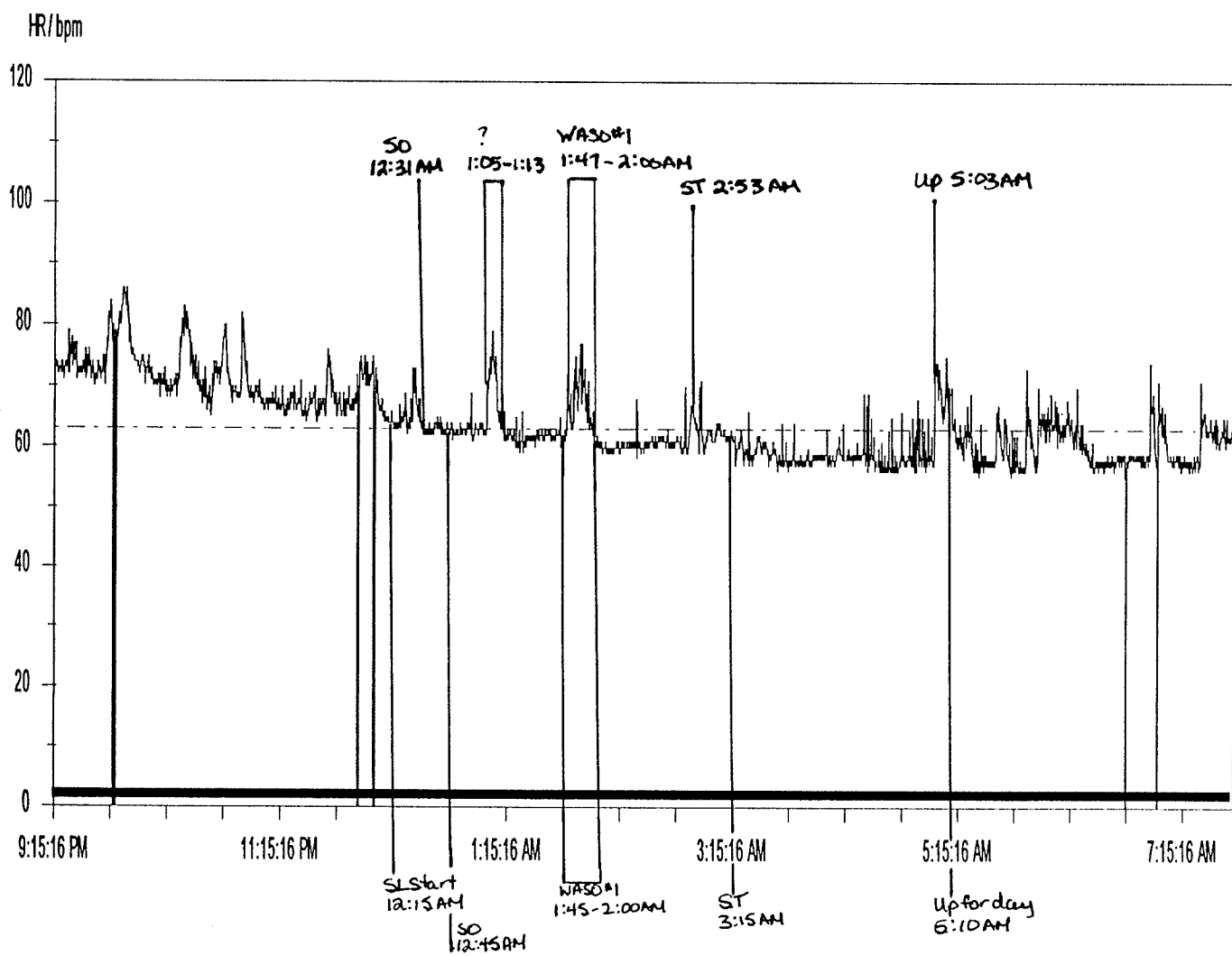
Heart Rate Profile for ID#10



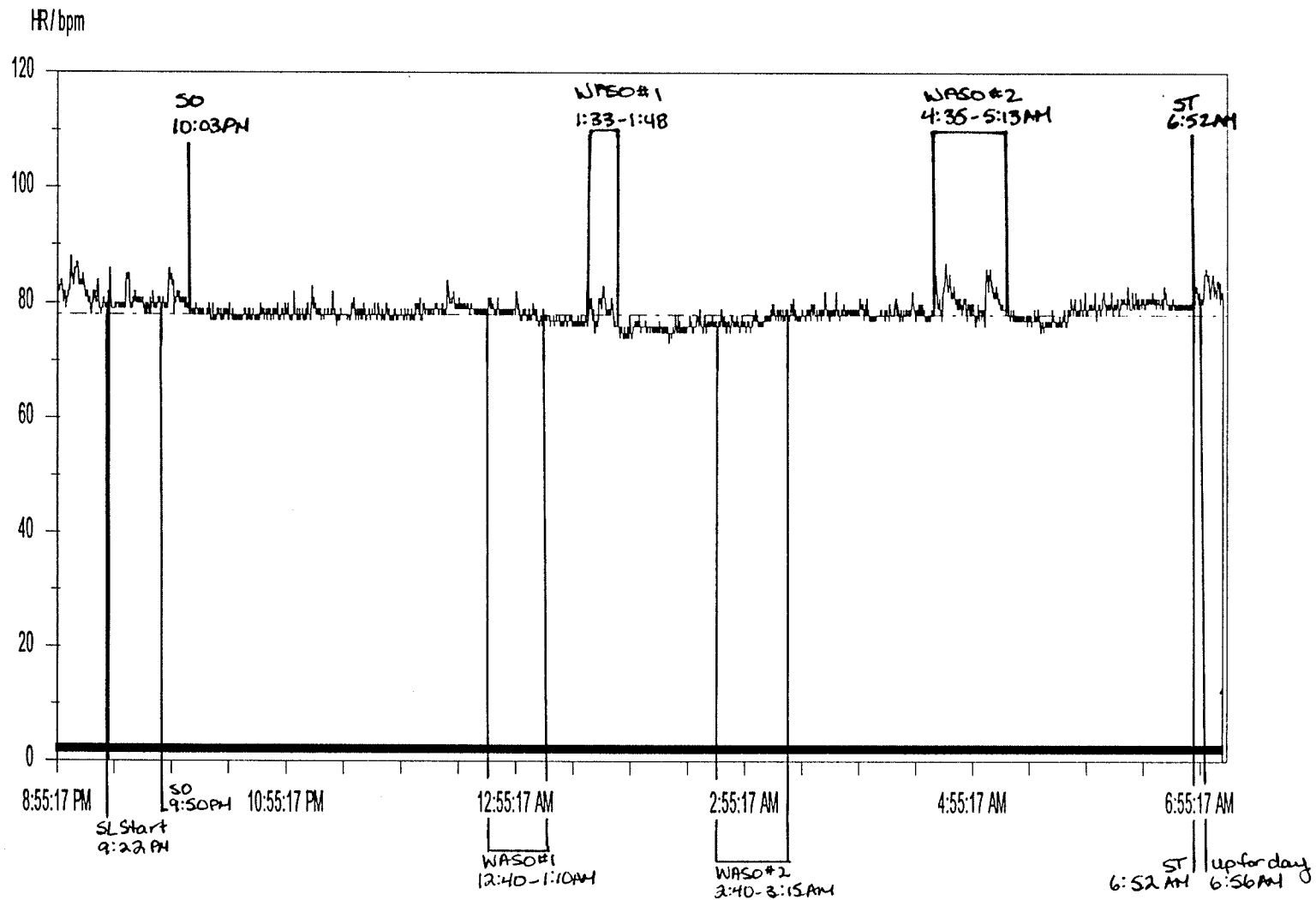
Heart Rate Profile for ID#11



Heart Rate Profile for ID#12

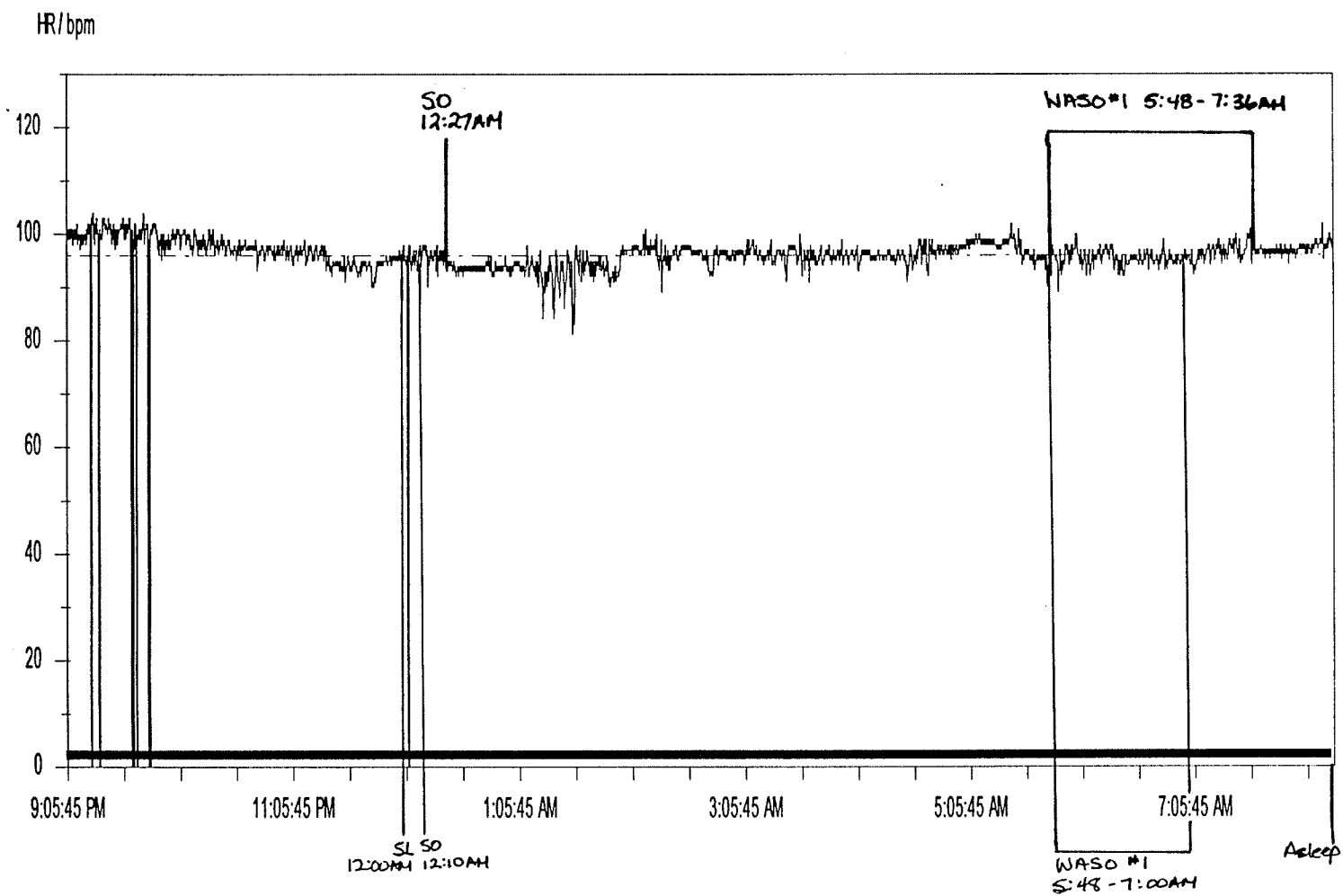


Heart Rate Profile for ID#13

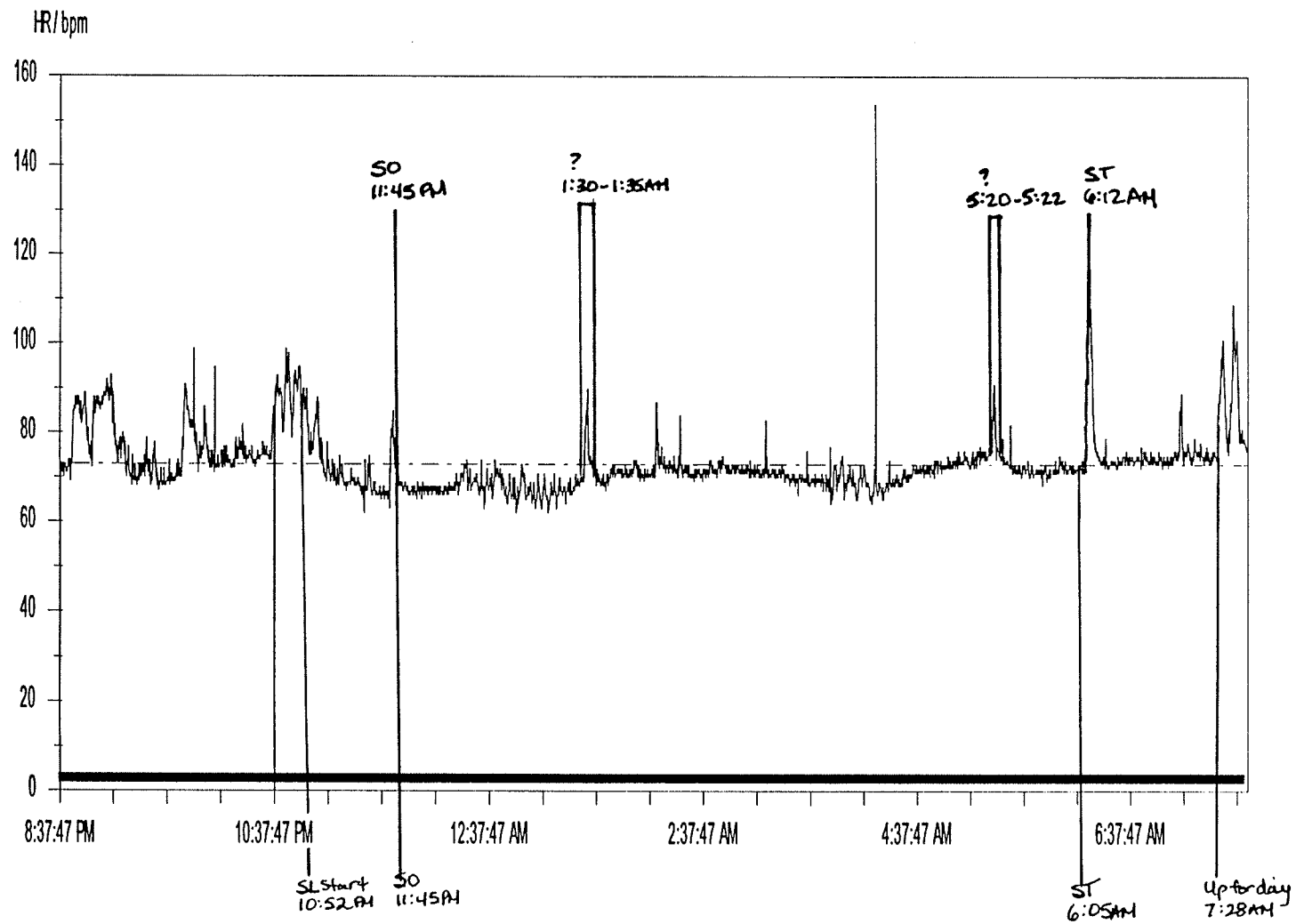


Heart Rate Profile for ID#14



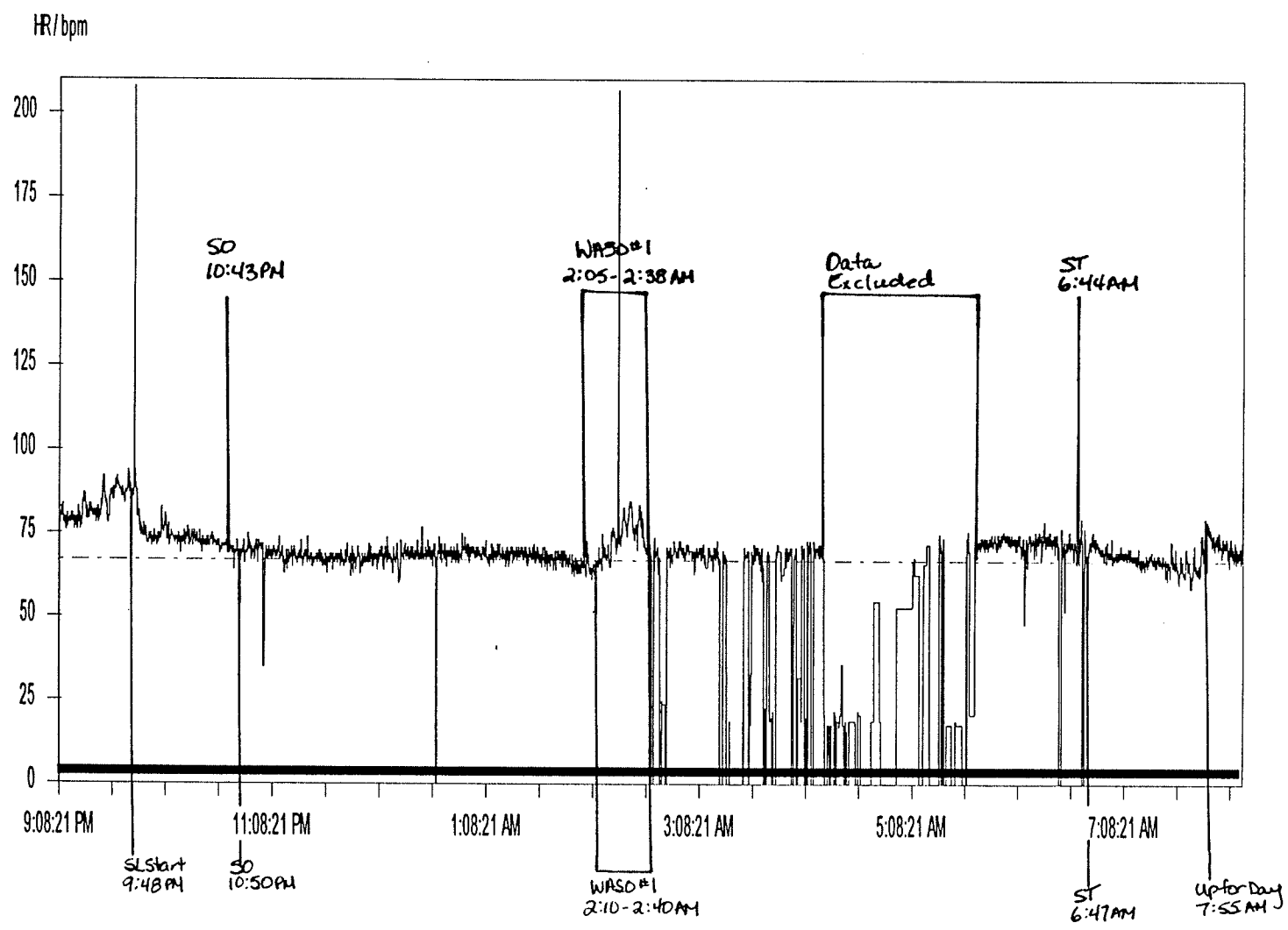


Heart Rate Profile for ID#15

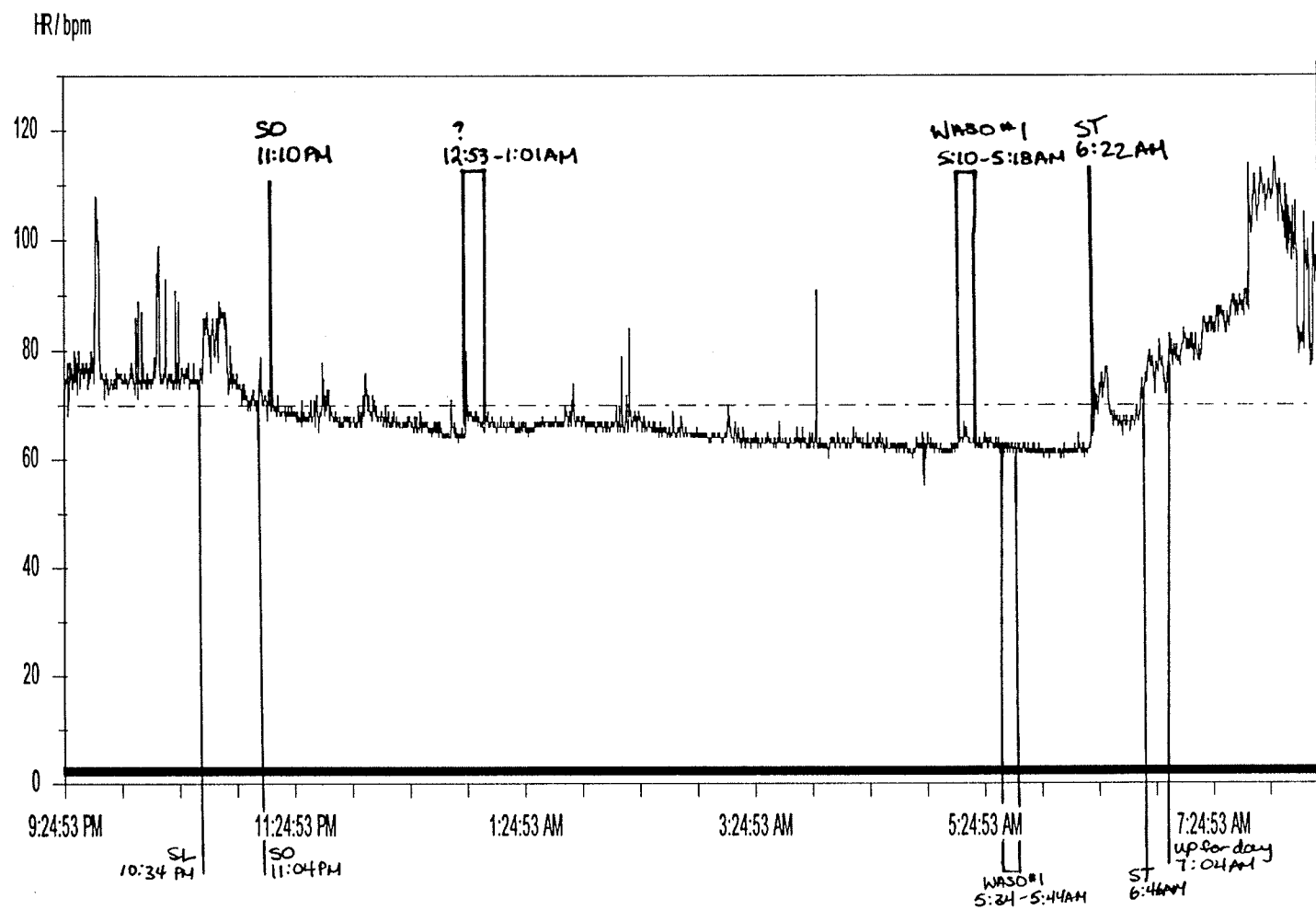


Heart Rate Profile for ID#16

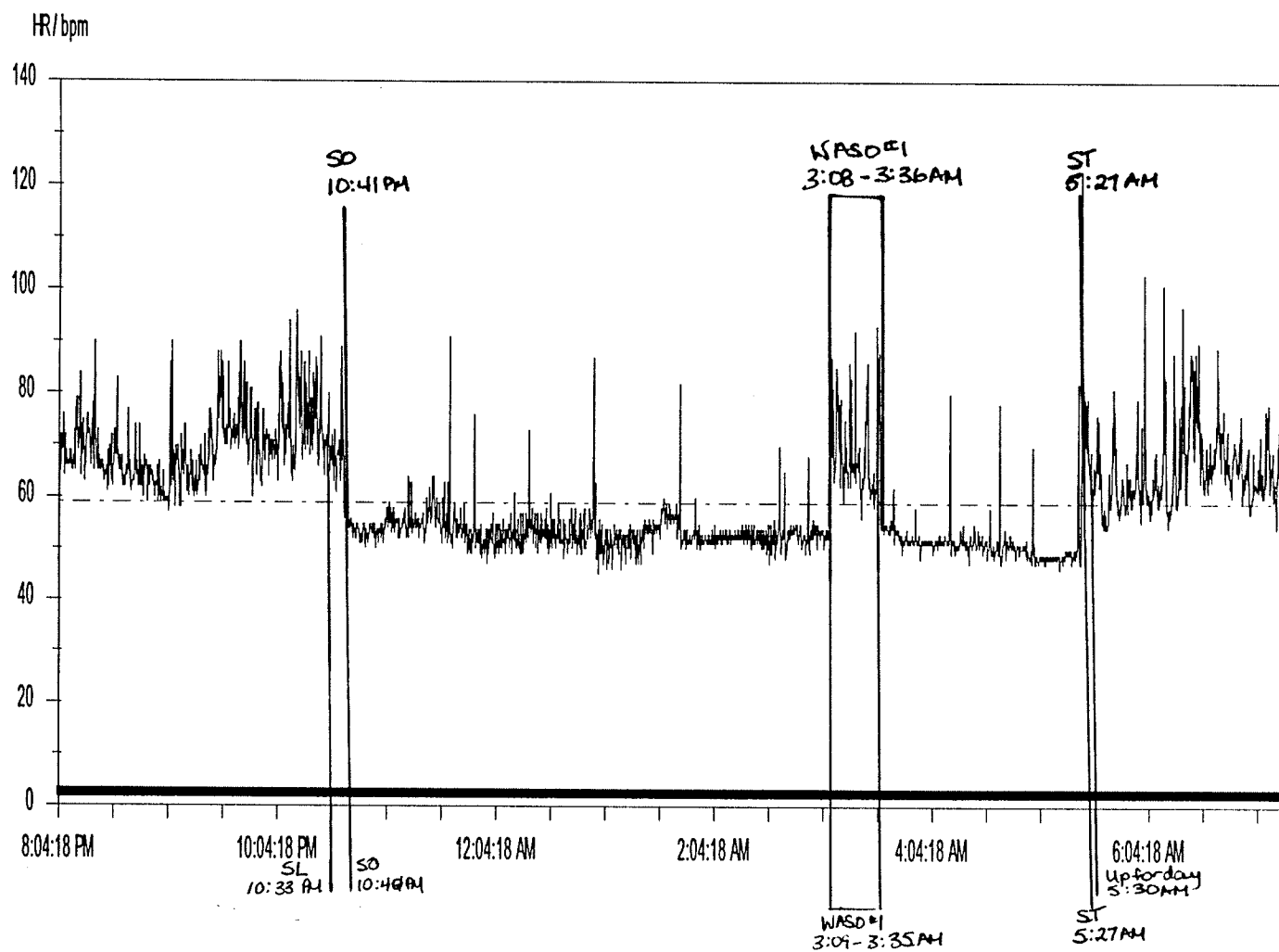
Sleep 120



Heart Rate Profile for ID#17

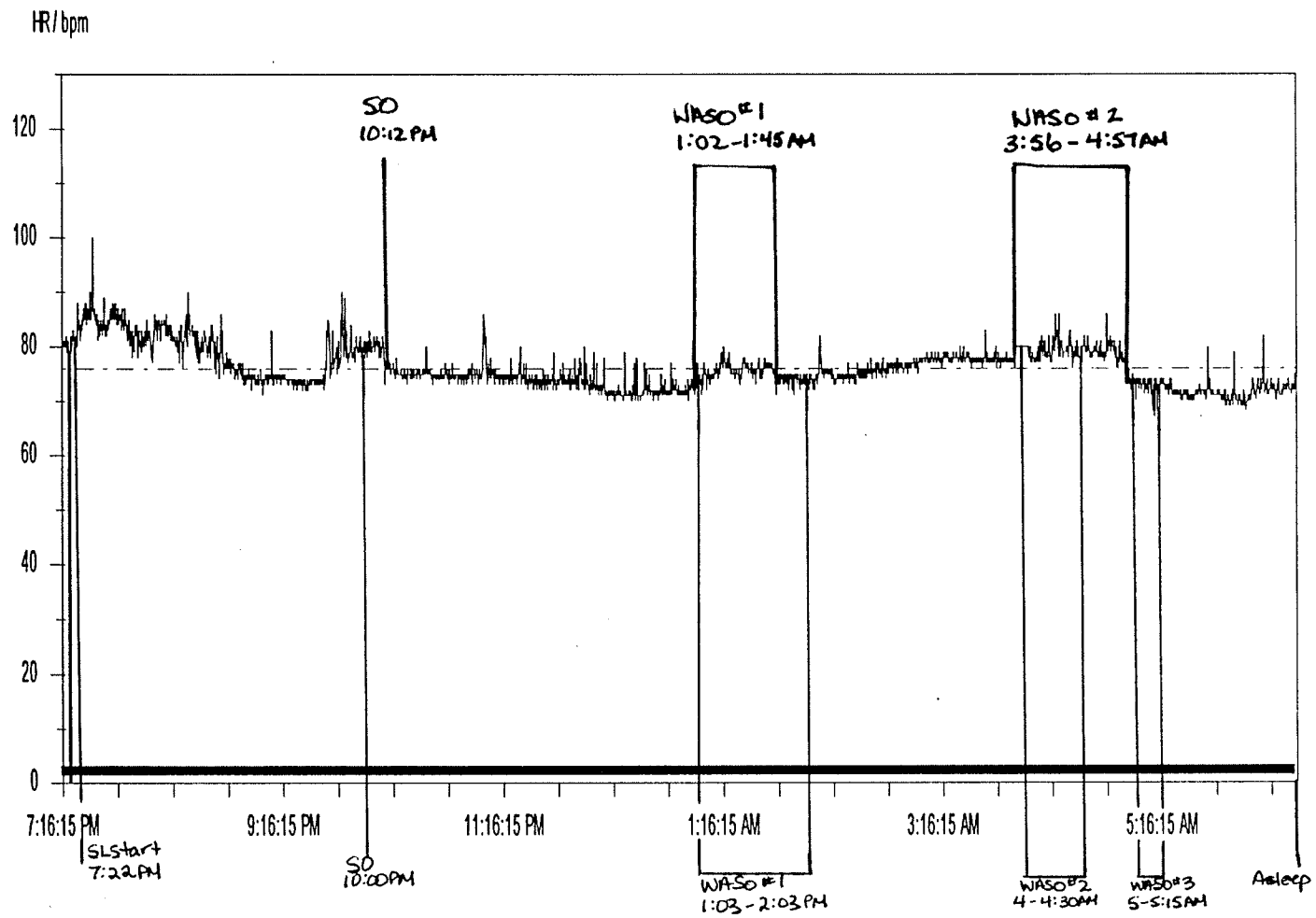


Heart Rate Profile for ID#18



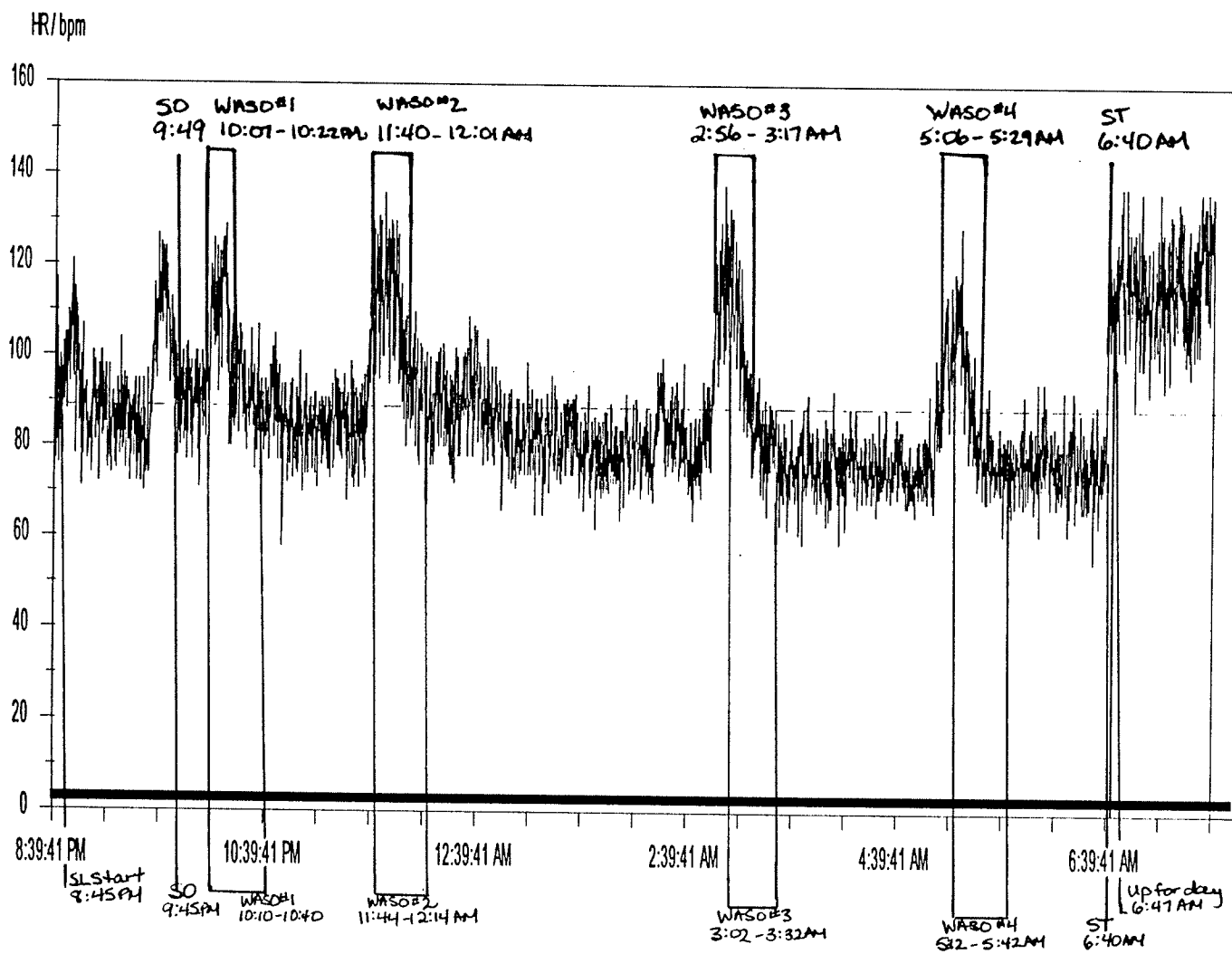
Heart Rate Profile for ID#19

Sleep 123

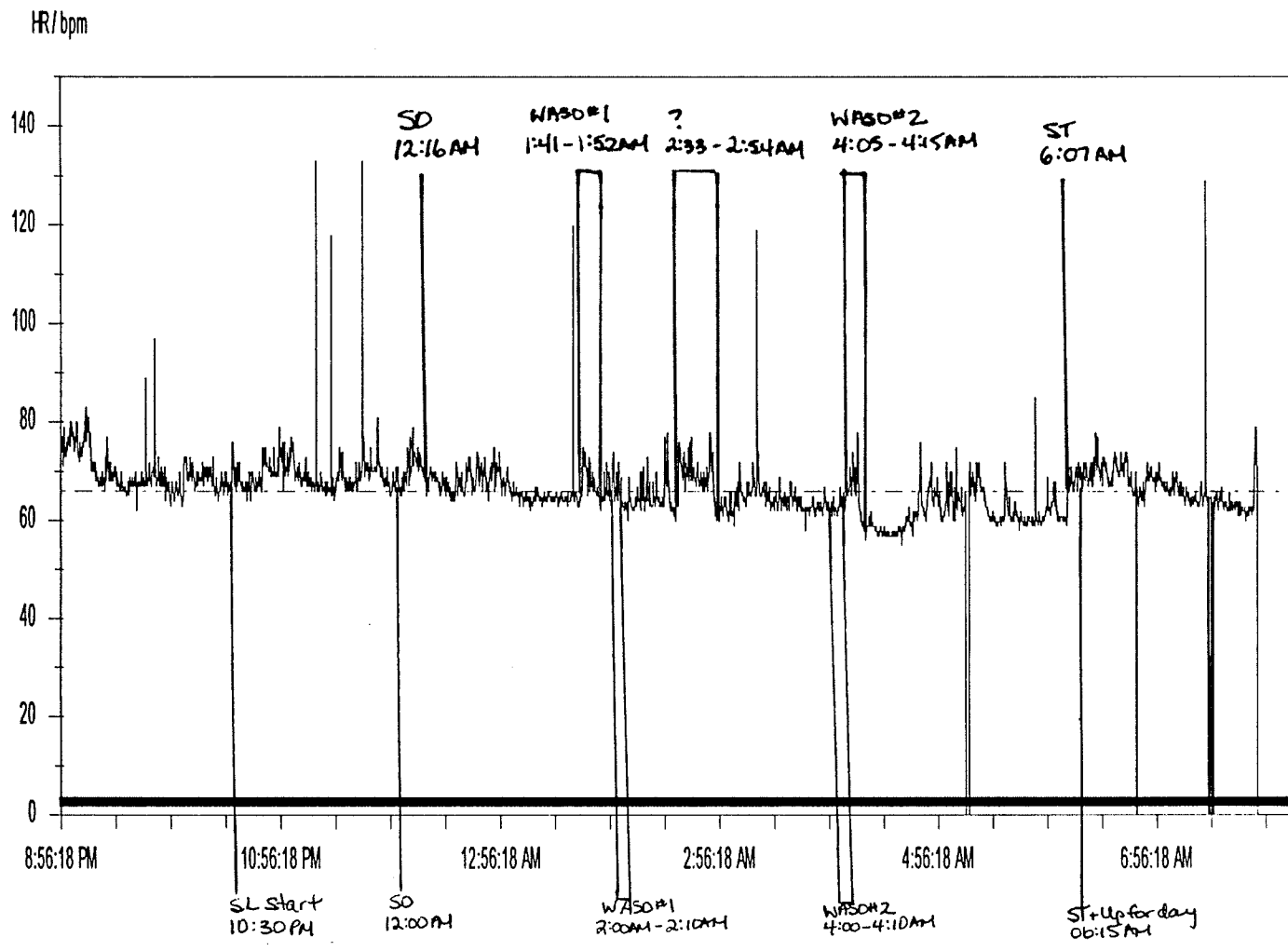


Heart Rate Profile for ID#20

Sleep 124

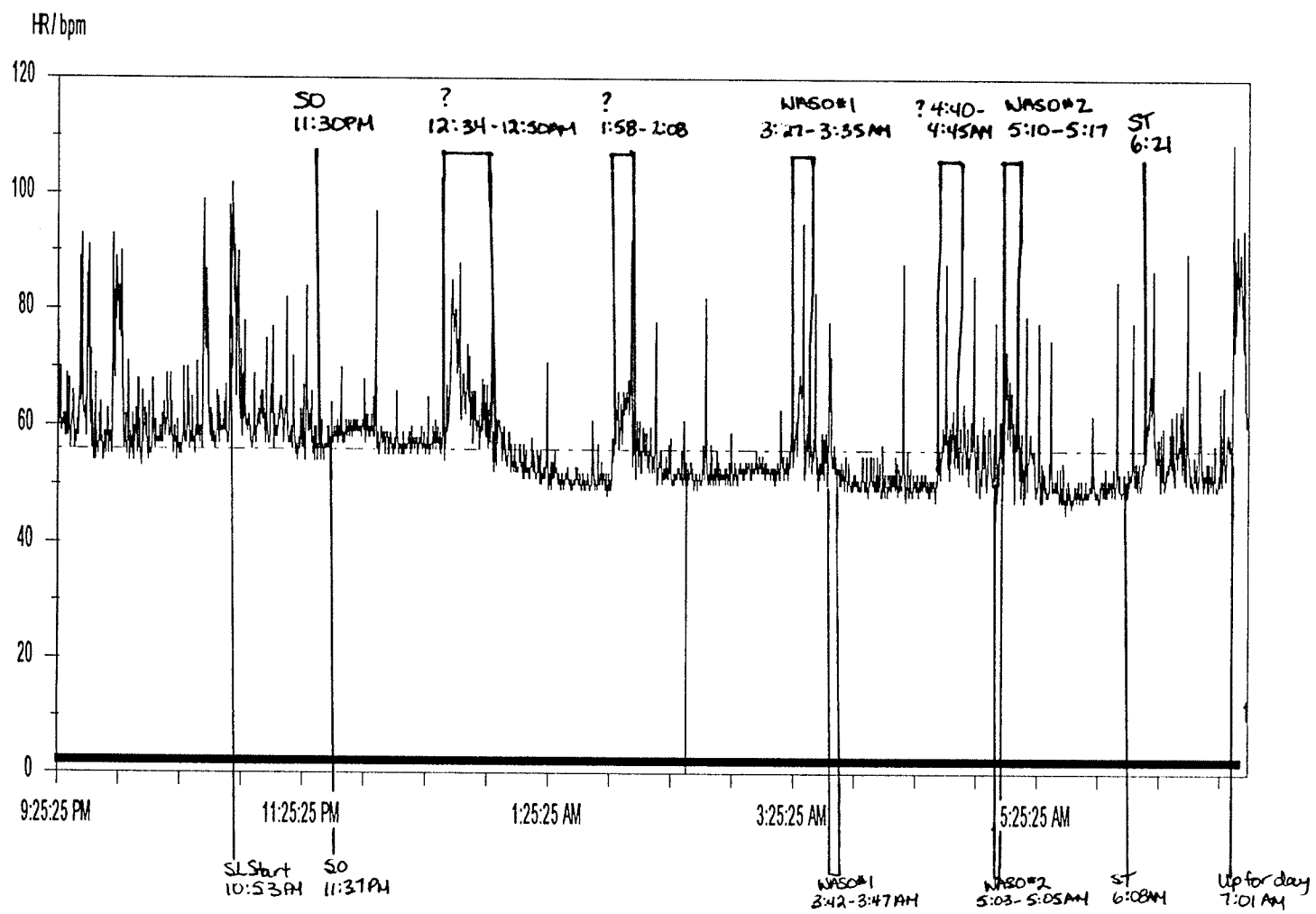


Heart Rate Profile for ID#21



Heart Rate Profile for ID#22





Heart Rate Profile for ID#23

Table 1

Use of Sleep Medications during the Study Night

Medication Used	Frequency (%)	<u>n</u>
Tylenol plain	37.5	3
Anxiolytic	25.0	2
Antidepressant	25.0	2
Tylenol plain, antidepressant, anxiolytic and anti- psychotic	12.5	1

n=8

Table 2

Summary of the Study's Nighttime Variables

<b>Variable</b>	<b>Mean</b>	<b><u>SD</u></b>	<b>Minimum Value</b>	<b>Maximum Value</b>
TST	384.3 minutes	121.6	135 minutes	541 minutes
#WASO	1.8	1.2	0	4
SSS score	3	2	1	7
VSH score	47.2	12.1	27.5	67.4
Overall HR	70.5 bpm	12.4	52.5 bpm	95.7 bpm
Resting HR	78.9 bpm	15.0	56.7 bpm	114.4 bpm
Corrected HR1	8.4 bpm	6.8	-0.75 bpm	28.1 bpm
SD1	5.1 bpm	2.7	1.7 bpm	12.3 bpm
Corrected HR2	8.5 bpm	6.7	-0.76 bpm	27.9 bpm
SD2	5.0 bpm	2.7	1.65 bpm	12.3 bpm

N=23

Table 3

Summary of VSH Sub-scores

<b>VSH Sub-scale</b>	<b>Mean</b>	<b><u>SD</u></b>	<b>Minimum</b>	<b>Maximum</b>
<b>VSH 1</b>	5.2	2.7	0.6	9.6
<b>VSH 2</b>	6.2	2.6	2.1	10.0
<b>VSH 3</b>	6.2	2.1	1.1	10.0
<b>VSH 4</b>	6.5	2.0	2.1	9.9
<b>VSH 5</b>	6.1	2.0	1.9	10.0
<b>VSH 6</b>	5.8	2.3	0.8	10.0
<b>VSH 7</b>	5.8	2.5	1.6	9.9
<b>VSH 8</b>	5.5	2.8	0.1	9.9
<b>Total VSH</b>	47.2	12.1	27.5	67.4

N=23

Note: Sub-scale scores are out of 10, total score out of 80.

Table 4

Correlation Matrix for Sleep Diary, Sleep Scales and HR Variables (r and p values)

	WASO	TST	SSS	VSH	Corr Mean1	SD1	Corr Mean 2	SD2
<b>WASO</b>	-							
<b>TST</b>	.43** .04	-						
<b>SSS</b>	-.01 .98	-.33 .12	-					
<b>VSH</b>	-.06 .79	.65*** .001	-.46** .03	-				
<b>Corr Mean1</b>	-.33 .12	.10 .65	-.0 .67	.24 .28	-			
<b>SD1</b>	.38* .08	.19 .40	.09 .67	.19 .38	-.185 .40	-		
<b>Corr Mean2</b>	-.33 .13	.11 .62	-.10 .65	.24 .27	1.00*** .000	-.189 .388	-	
<b>SD2</b>	.39* .07	.22 .32	.12 .59	.28 .30	-.188 .39	.997*** .000	-.191 .384	-

N = 23

\* Significance level &lt; .10

\*\* Significance level &lt; .05

\*\*\* Significance level &lt; .01

Table 5

Multiple Regression Model Results for VSH Score (Dependent Variable) versus Total Sleep Time, Wakenings after Sleep Onset (Independent Variables)

Model	B	Std. Error	Beta	Partial t	Significance
(Constant)	22.930	5.906		3.882	.001
TST	.0825	.016	.829	5.086	.000
WASO	-4.159	1.639	-.414	-2.539	.020

$R^2 = .565$ , Adjusted  $R^2 = .522$ , Standard error = 8.368

Table 6

Multiple Regression Model Results for VSH Score (Dependent Variable) versus Total Sleep Time, Wakenings after Sleep Onset and Interaction Term (Independent Variables)

Model	B	Std. Error	Beta	Partial t	Significance
(Constant)	28.281	7.819		3.617	.002
TST	.0676	.022	.679	3.130	.006
WASO	-10.597	6.395	-1.055	-1.657	.114
Interaction	.0159	.015	.734	1.041	.311

$R^2 = .589$ , Adjusted  $R^2 = .524$ , Standard error = 8.350

Table 7

Multiple Regression Model Results for SSS Score (Dependent Variable) versus Total  
Sleep Time, Wakenings after Sleep Onset, Interaction Term (Independent Variables)

Model	B	Std. Error	Beta	Partial t	Significance
(Constant)	8.400	1.541		5.452	.000
TST	-.0164	.004	-.978	-3.863	.001
WASO	-2.464	1.260	-1.451	-1.956	.065
Interaction	.00713	.003	1.946	2.369	.029

$R^2 = .441$ , Adjusted  $R^2 = .353$ , Standard error = 1.65



Table 8

Multiple Regression Model Results for Total Sleep Time (Dependent Variable) versus  
Corrected Mean1, SD1, Interaction Term (Independent Variables)

Model	B	Std. Error	Beta	Partial t	Significance
(Constant)	430.393	100.842		4.268	.000
Mean1	-11.577	10.551	-.644	-1.097	.268
SD1	-2.311	16.345	-.051	-.141	.889
Interaction	1.369	1.478	.642	.926	.366

$R^2 = .095$ , Adjusted  $R^2 = -.048$ , Standard error = 124.54

Table 9

Multiple Regression Model Results for Total Sleep Time (Dependent Variable) versus  
Corrected Mean2, SD2, Interaction Term (Independent Variables)

Model	B	Std. Error	Beta	Partial t	Significance
(Constant)	418.394	98.327		4.255	.000
Mean2	-10.520	9.892	-.581	-1.063	.301
SD2	.150	16.292	.003	.009	.993
Interaction	1.190	1.400	.560	.850	.406

$R^2 = .105$ , Adjusted  $R^2 = -.037$ , Standard error = 123.84

Table 10

Multiple Regression Model Results for Wakenings after Sleep Onset (Dependent Variable) versus Corrected Mean1, SD1 (Independent Variables)

Model	B	Std. Error	Beta	Partial t	Significance
(Constant)	.626	.555		1.128	.273
Mean1	.0488	.036	.274	1.359	.189
SD1	.146	.091	.325	1.612	.123

$R^2 = .214$ , Adjusted  $R^2 = .135$ , Standard error = 1.12

Table 11

Multiple Regression Model Results for Wakenings after Sleep Onset (Dependent Variable) versus Corrected Mean2, SD2 (Independent Variable)

Model	B	Std. Error	Beta	Partial t	Significance
(Constant)	.623	.547		1.138	.269
Mean2	.0474	.036	.265	1.314	.204
SD2	.150	.090	.337	1.674	.110

$R^2 = .218$ , Adjusted  $R^2 = .140$ , Standard error = 1.12

Table 12

Multiple Regression Model Results for VSH Score(Dependent Variable) versus  
Corrected Mean1, SD1, Interaction Term (Independent Variables)

Model	B	Std. Error	Beta	Partial t	Significance
(Constant)	47.573	9.914		4.798	.000
Mean1	.0715	1.037	-.400	-.690	.499
SD1	.824	1.607	.183	.513	.614
Interaction	.0318	.145	.150	.219	.829

$R^2 = .116$ , Adjusted  $R^2 = -.023$ , Standard error = 12.244

Table 13

Multiple Regression Model Results for VSH Score (Dependent Variable) versus  
Corrected Mean2, SD2, Interaction Term (Independent Variables)

Model	B	Std. Error	Beta	Partial t	Significance
(Constant)	46.705	9.617		4.857	.000
Mean2	-.683	.967	-.380	-.706	.489
SD2	1.040	1.593	.232	.653	.522
Interaction	.0244	.137	.115	.178	.860

$R^2 = .135$ , Adjusted  $R^2 = -.002$ , Standard error = 12.113

Table 14

Multiple Regression Model Results for SSS Score (Dependent Variable) versus Corrected Mean1, SD1, Interaction Term (Independent Variables)

Model	B	Std. Error	Beta	Partial t	Significance
(Constant)	2.030	1.749		1.161	.260
Mean1	.144	.183	.477	.787	.441
SD1	.141	.283	.185	.498	.624
Interaction	.0209	.026	-.582	-.814	.426

$R^2 = .036$ , Adjusted  $R^2 = -.116$  Standard error = 2.16

Table 15

Multiple Regression Model Results for SSS Score (Dependent Variable) versus Corrected Mean2, SD2, Interaction Term (Independent Variables)

Model	B	Std. Error	Beta	Partial t	Significance
(Constant)	2.348	1.725		1.361	.189
Mean2	.108	.173	.355	.621	.542
SD2	.0897	.286	.119	.314	.757
Interaction	-.0156	.025	-.435	-.633	.534

$R^2 = .026$ , Adjusted  $R^2 = -.128$  Standard error = 2.17



Table 16

Summary of Comparison between Sleep Diaries and HR Profiles for Sleep Onset, Sleep Termination and Wakenings after Sleep Onset

ID#	SO (min)	ST (min)	WASO Matches	Comments (Sleep diary WASO)	Unexplained events	Comments
1	48	-4	1/1	- began 10 min later and was 8 min longer	0	
2	20	7	3/3	- all beginning times agreed - overestimated by 19, 2 and 20 min respectively	1	- 6 min in length
3	11	13	2/2	- reported began 2-4 min later - 2-10 min longer in length	0	
4	-9	5	N/A		2	- difficult profile to interpret - 2 rises of 29 and 57 min towards end of night when reported awake
5	-5	0	1\2	- #1 matched - #2 recorded 20 min later and 51 min less than HR rise	2	- 37 and 64 min in length
6	-4	N/A	3/3	-#1 recorded 3 min earlier and 5 min longer -#2 recorded 5 min later and 15 min less -#3 recorded 13 min later and 7 min longer	0	

ID#	SO (min)	ST (min)	WASO Matches	Comments (Sleep diary WASO)	Unexplained events	Comments
7	4	0	1/3	-#1 matched -#2 recorded 79 min later and was 49 min shorter -#3 recorded 90 min later and 75 min shorter	0	
8	55	0	3/3	-#1 reported 18 min earlier and 19 min longer -#2 reported 37 min early and 4 min longer	2	- 5 and 20 min rises in HR
9	-3	-17	1/1	-recorded 23 min later and 2 min shorter	0	
10	0	19	N/A		1	- 21 min rise prior to SO - ? up to BR
11	20	42	4/4	-#1 matched -all others start time recorded accurately but overestimated length by 14,6, and 27 min	2	-12 and 7 min unexplained HR rise
12	38	N/A	1/1	- start time matched, underestimated length by 6 min	1	- 3 min rise
13	14	22	1/1	- matched	1	- 8 min rise
14	-13	0	0/2	-both are quite offset and if times were shifted down, would match	0	
15	-17	N/A	1/1	- start time matched, overestimated length by 36 min	0	

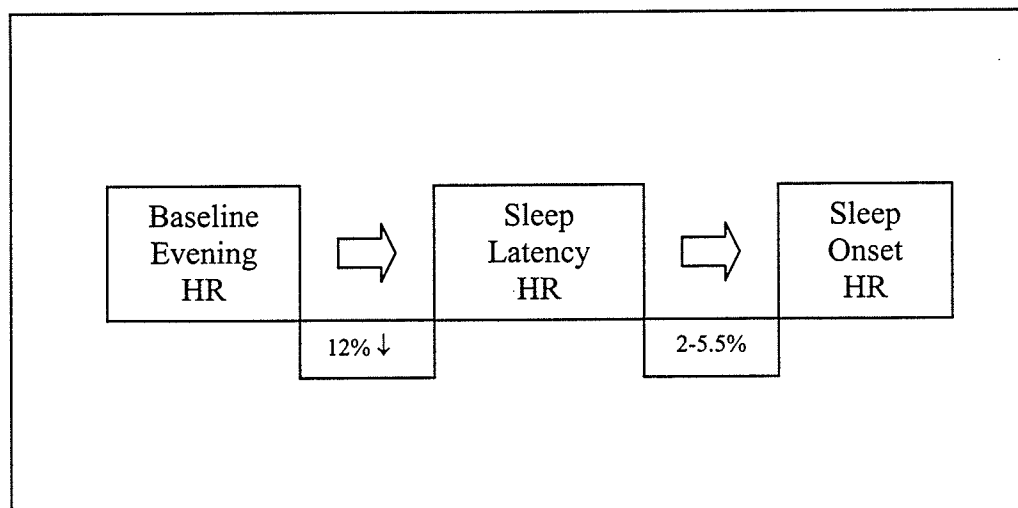
ID#	SO (min)	ST (min)	WASO Matches	Comments (Sleep diary WASO)	Unexplained events	Comments
16	0	-7	N/A		2	- 5 and 2 min - ? temporary arousals
17	7	3	1/1	- reported as starting 5 min later and lasting 3 min longer	0	
18	-6	24	1/1	- offset - length = but reported starting 24 min later	1	- 8 min rise
19	0	0	1/1	- matched	0	
20	-12	N/A	2/3	-#1 reported start a match, 17 min longer -#2 start 4 min later and 31 min shorter -#3 no corresponding rise in HR	0	- possible that WASO #2 and 3 may be combined
21	-4	0	4/4	-all starts reported later by 3, 4, 6 and 6 respectively -all lengths overestimated by 15, 9, 9 and 7 min respectively	0	
22	-16	8	2/2	-offset -both match in length but #1 reported to begin 19 min later and #2 5 min earlier	1	-21 min rise in HR
23	7	-13	2/2	-offset -#1 reported to start 15 min later and 3 min shorter -#2 start 7 min earlier and 5 min shorter	3	-16, 10 and 5 min rising -? REM HR variation

Table 17

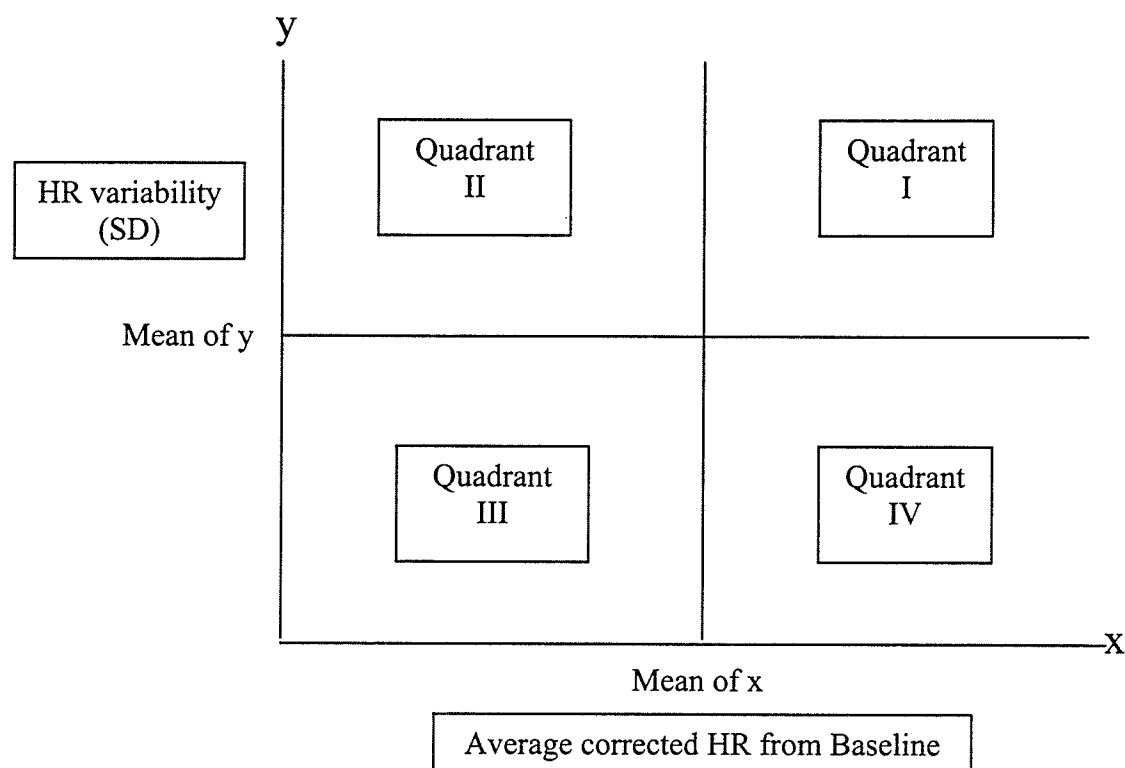
Comparison of Mean HR Values from the Sleep Diary and HR Profiles to the Conceptual Framework Predicting Decreases in HR from Baseline → Sleep Latency → Sleep Onset

#	Baseline	SL (Diary)	% ↓	SO (Diary)	% ↓	SL (Profile)	% ↓	SO (Profile)	% ↓
1	56.67	57.85	-	47.29	18.3	61.86	-	54.88	11.3
2	85.37	80.37	5.86	79.55	1.02	80.85	5.29	77.88	<b>3.67</b>
3	83.44	66.22	20.64	62.10	6.22	66.47	20.34	62.29	6.29
4	104.66	100.54	3.94	97.88	<b>2.65</b>	100.21	4.25	96.45	<b>3.75</b>
5	78.66	84.30	-	82.90	-	83.11	-	79.02	<b>4.68</b>
6	57.05	55.17	3.30	56.33	-	55.22	3.21	55.65	-
7	114.43	108.30	5.36	106.27	<b>1.87</b>	108.42	5.25	107.39	0.95
8	85.68	74.33	<b>13.25</b>	74.76	-	75.66	<b>11.69</b>	72.37	<b>4.35</b>
9	70.98	81.68	-	74.51	-	80.39	-	63.29	21.3
10	57.38	54.81	4.48	54.76	0.09	54.81	4.48	54.76	0.09
11	88.57	85.00	4.03	82.69	<b>2.72</b>	86.64	2.18	82.27	<b>5.04</b>
12	70.93	68.94	2.81	67.24	<b>2.47</b>	69.29	2.31	69.00	0.42
13	72.87	64.09	<b>12.05</b>	62.47	<b>2.53</b>	65.09	<b>10.68</b>	62.49	<b>3.99</b>
14	81.75	80.24	1.85	79.80	0.55	80.50	1.53	79.18	<b>1.64</b>
15	99.78	95.56	4.23	95.47	0.09	95.72	4.07	94.10	<b>1.69</b>
16	72.02	72.54	-	70.20	<b>3.23</b>	72.55	-	68.78	<b>5.20</b>
17	79.13	74.00	6.48	69.18	<b>6.51</b>	74.55	5.79	69.84	<b>6.32</b>
18	74.80	77.71	-	71.90	-	76.69	-	70.41	8.19
19	67.70	68.87	-	56.35	18.2	67.56	0.21	55.20	18.3
20	80.49	79.36	1.40	80.08	-	79.40	1.35	76.37	<b>3.82</b>
21	95.10	92.52	2.71	100.24	-	93.00	2.21	92.57	0.46
22	75.27	69.15	8.13	67.35	<b>2.60</b>	69.33	7.89	69.24	0.13
23	61.68	60.64	1.69	57.98	<b>4.39</b>	61.38	0.49	56.80	7.46

Note: Numbers in bold represent values and profile that match conceptual framework within 2% of SL and 1% of SO.



**Figure 1.** Conceptual model of the percent decrease in HR values from evening baseline to sleep onset.



**Figure 2.** Hypothetical sleep scenarios as indicated by the four-quadrant graph.

Legend: Quadrant I =  $\uparrow$  HR,  $\uparrow$  SD – mediocre sleep (awake on and off)  
 Quadrant II =  $\downarrow$  HR,  $\uparrow$  SD – mediocre sleep (awake for long periods)  
 Quadrant III =  $\downarrow$  HR,  $\downarrow$  SD – worst sleep  
 Quadrant IV =  $\uparrow$  HR,  $\downarrow$  SD – best sleep

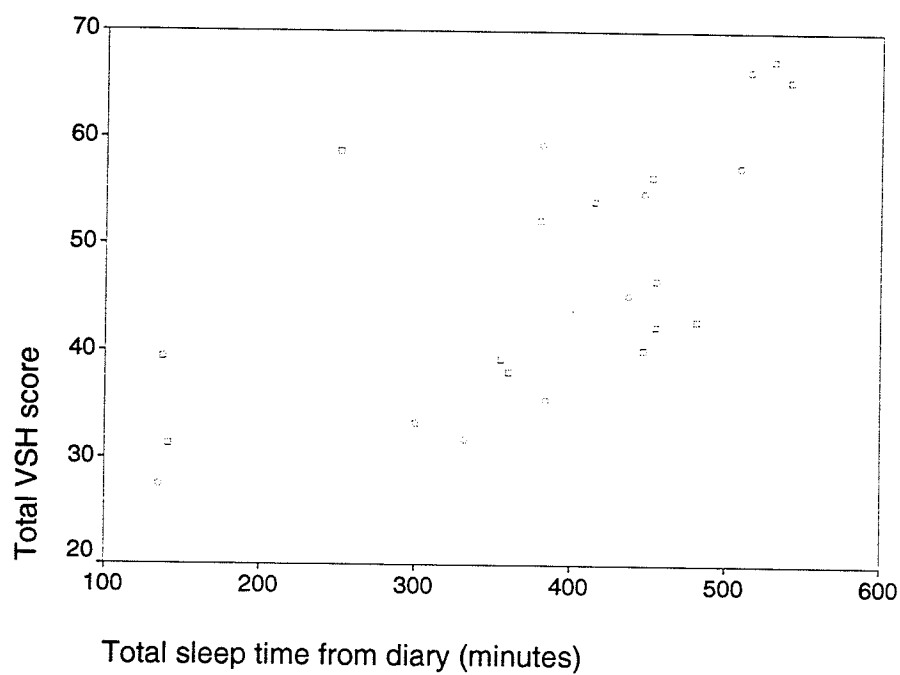


Figure 3. Scatter plot Total Sleep Time by VSH score

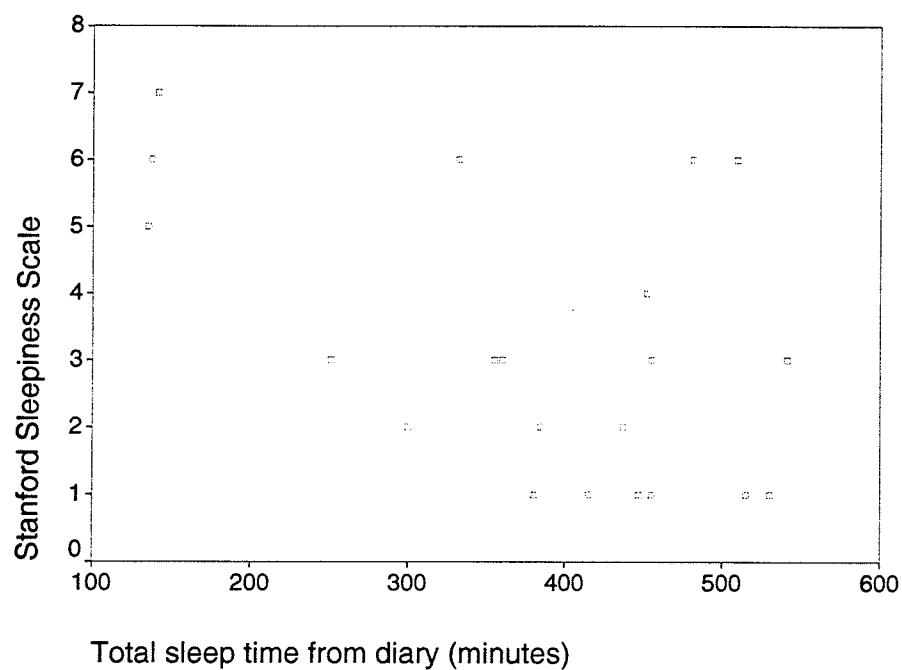


Figure 4. Scatter plot Total Sleep Time by SSS score



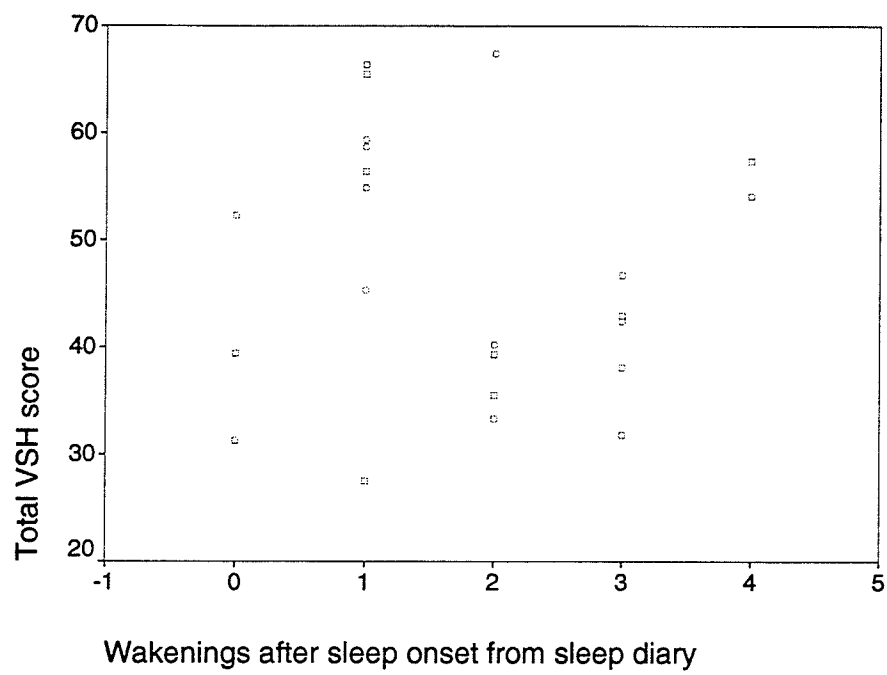


Figure 5. Scatter plot Wakenings after Sleep Onset by VSH score

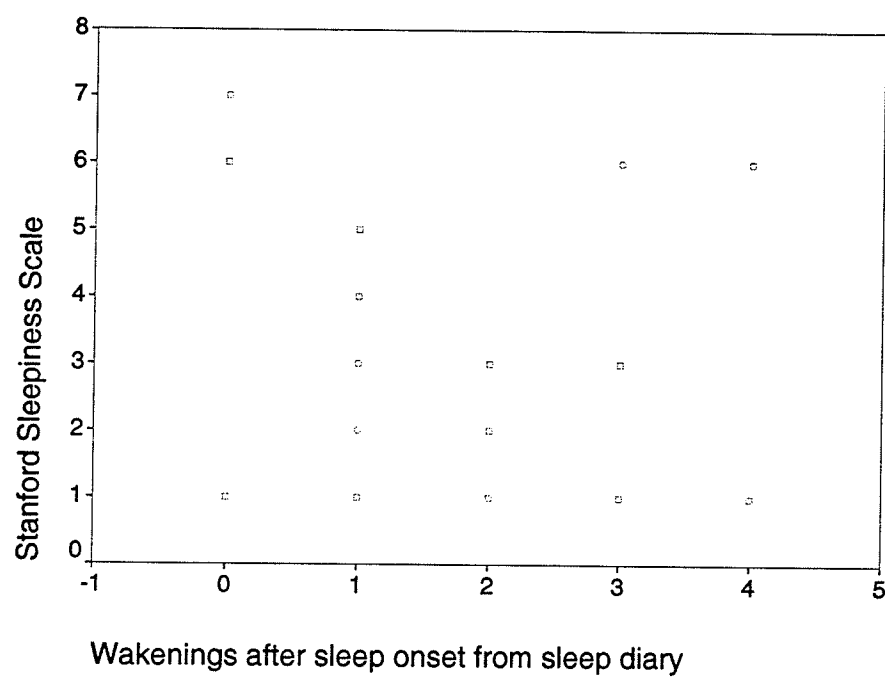


Figure 6. Scatter plot Wakenings after Sleep Onset by SSS score

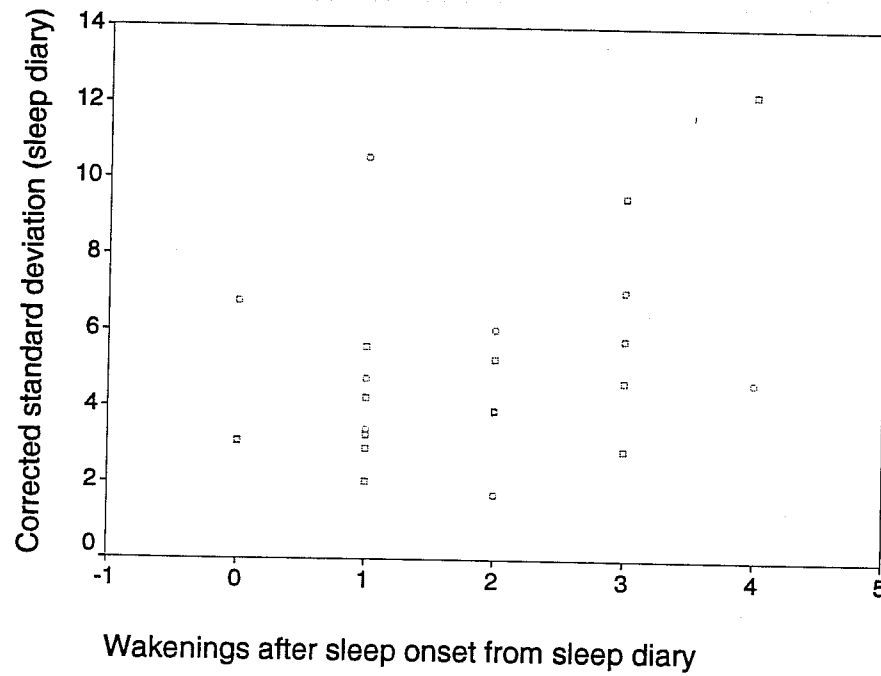


Figure 7. Scatter plot Wakenings after Sleep Onset by SD1

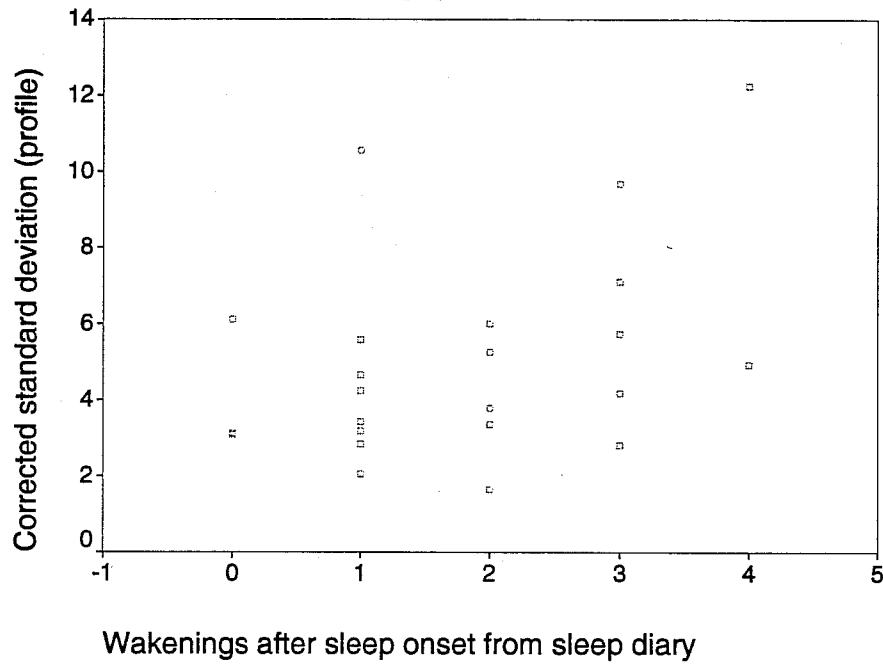


Figure 8. Scatter plot Wakenings after Sleep Onset by SD2

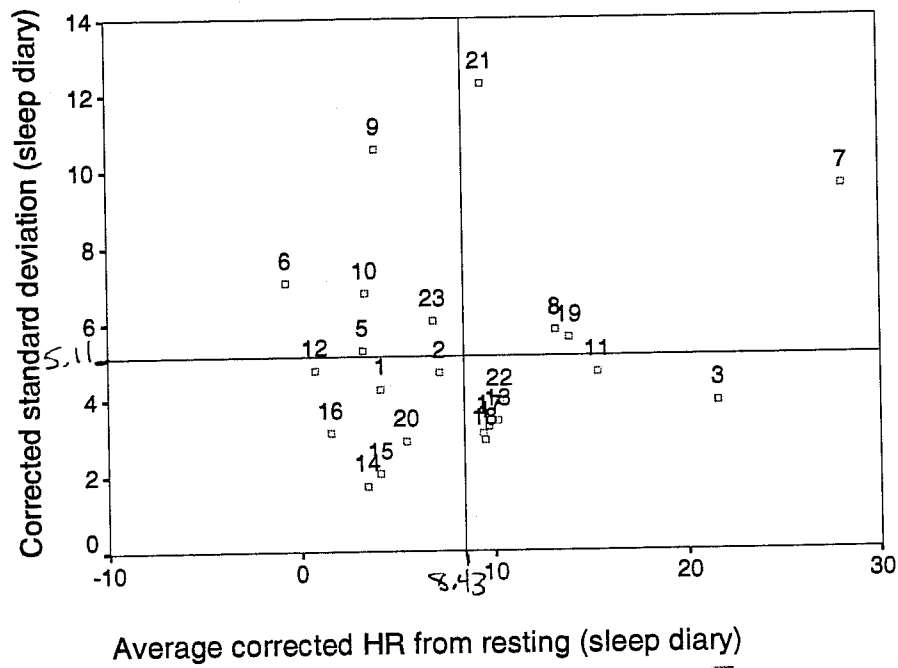


Figure 9. Four-quadrant graph – Corrected Mean1 by SD1

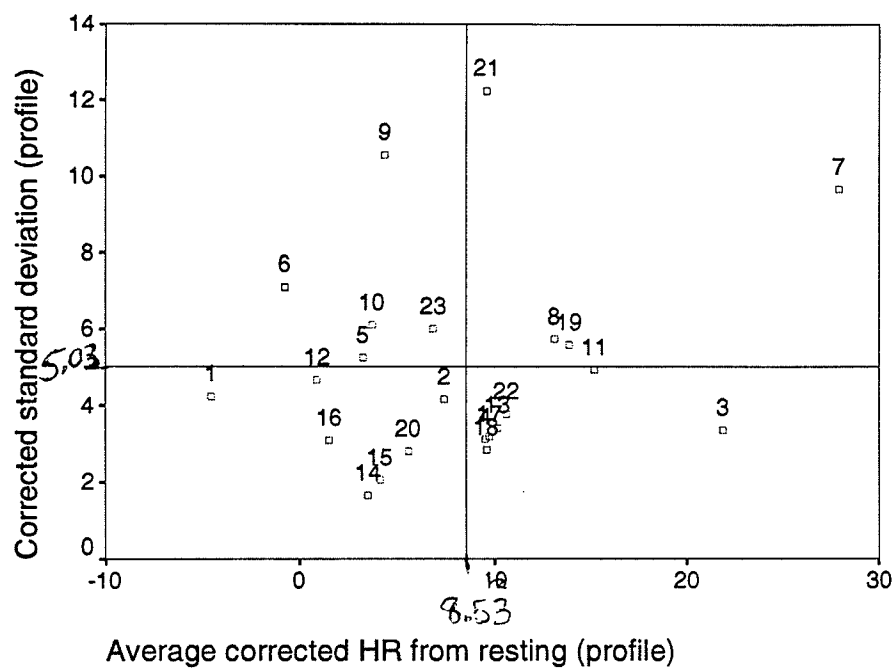


Figure 10. Four-quadrant graph – Corrected Mean2 by SD2