THE IMPACT OF ACOUSTIC STIMULATION AND DAYTIME NAPPING ON HEART RATE VARIABILITY AND SLEEP ARCHITECTURE

by

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NOMENCLATURE

AASM	American Academy of Sleep Medicine
AHI	Apnea-Hypopnea Index
ANOVA	Analysis of Variance
ANS	Autonomic Nervous System
AS	Acoustic Stimulation
AU	Arbitrary Units
BAI	Beck Anxiety Inventory
BMI	Body Mass Index
BP	Blood Pressure
C	Control Condition
CES-D	Center for Epidemiologic Studies Depression Scale
DAP	Diastolic Arterial Pressure
ECG	Electrocardiogram
EEG	Electroencephalogram
EMG	Electromyography
EOG	Electrooculogram

ESS	Epworth Sleepiness Scale
FFT	Fast Fourier Transform
HF	High Frequency
HRV	Heart Rate Variability
ISI	Insomnia Severity Index
KSS	Karolinska Sleepiness Scale
LF	Low Frequency
LF/HF	Low Frequency/High Frequency Ratio
MAP	Mean Arterial Pressure
MSNA	Muscle Sympathetic Nerve Activity
N1	Stage 1 Sleep
N2	Stage 2 Sleep
N3	Stage 3 Sleep
NREM	Non-Rapid Eye Movement
pNN50	Percentage of R-R Intervals That Varied By 50 ms or More
PNS	Parasympathetic Nervous System
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index

REM	Rapid Eye Movement
RMSSD	Root Mean Squared of Successive Difference
RRI	R-R Interval
SAP	Systolic Arterial Pressure
SCN	Suprachiasmatic Nucleus
SNS	Sympathetic Nervous System
STAI	Spielberger State Anxiety Inventory
SWS	Slow Wave Sleep
TSD	Total Sleep Deprivation
TST	Total Sleep Time
WASO	Wake After Sleep Onset

ABSTRACT

Receiving adequate overnight sleep is imperative for proper autonomic nervous system function. Recent studies utilizing acoustic stimulation (AS) have shown an improvement in sleep quality when applied during nighttime sleep, although the implications of AS on napping are not known. The purpose of the current study was to assess the impact of AS during a daytime nap on sleep and autonomic function. We hypothesized that AS (0.8 Hertz monaural beats at 30 decibels) would improve heart rate variability (HRV) during non-rapid eye movement (NREM) sleep and increase time spent in slow wave sleep in young adults. 25 young healthy adult subjects (12 men, 13 women, age: 23 ± 1 yrs, BMI: 24 ± 1 kg/m^2) took part in the study. Participants were enrolled into a randomized, placebo-controlled crossover design where they completed two 90-minute afternoon nap opportunities with or without AS, at least one day apart. During each testing session, participants were fitted with gold-standard polysomnographic (PSG) equipment and five-lead electrocardiogram (ECG). Subjective questionnaires assessing sleepiness (Epworth Sleepiness Scale) and anxiety (Spielberger State Anxiety Inventory) were given prior to and following each nap opportunity. ECG was recorded continuously starting 5 minutes prior to lights out until awakening, allowing assessment of wake and sleep HRV. Contrary to our hypothesis, there were no significant changes in total sleep time (Control: 73±4 vs. AS: 72±4 min, P=0.850), slow wave sleep (Control: 30±4 vs. AS: 31±4 min, P=0.855), or other objective sleep measurements between conditions (All P>0.05). AS did not impact sleep HRV measures (All P>0.05). However, there was a significant main effect for time where napping resulted in a significant increase in the R-R interval ($F(1,19)=25.683$, $P<0.001$) and reduction of self-report anxiety ($F(1,24)=8.229$, $P=0.008$), but these responses were not different across conditions (time x condition, P>0.05). Napping elicited a paradoxical increase of diastolic ($F(1,24)=14.483$, $P=0.001$) and mean ($F(1,24)=10.857$, $P=0.003$) arterial pressure, but again these responses were not different across conditions (time x condition, P>0.05). In summary, the current study supports beneficial impacts of daytime napping on anxiety levels, but minimal effects of AS on sleep architecture and autonomic function.

CHAPTER ONE

LITERATURE REVIEW

Introduction to Sleep Medicine

Sleep plays a critical role in both physiological and cognitive function. However, despite the importance of sleep in physiological functioning across numerous health domains, the study of sleep and sleep-related processes is fairly new (Shepard et al., 2005). Briefly, building upon the work of the late 1920s, Loomis et al. (1937) were the first to use electroencephalography (EEG) in the United States to classify non rapid eye movement (NREM) stages of sleep, while rapid eye movement sleep (REM) wasn't characterized until research conducted by Dement and Kleitman (Aserinsky & Kleitman, 1953; Dement & Kleitman, 1957) in the 1950s. A standardized set of guidelines for the scoring of sleep stages in human subjects was not defined until 1968 (Kales & Rechtschaffen, 1968), with these guidelines being modified and replaced in the early 2000's by the American Academy of Sleep Medicine (AASM) (Iber, 2004). In sum, the field of sleep medicine is unique in reference to some other physiological fields of research, in that it has only gained traction within human subjects in the scientific community within the past century. Nonetheless, significant strides have been made in our understanding of sleep, and its importance on cognition and emotional regulation (Walker, 2009, 2010) and cardiovascular regulation (Trinder et al., 2012). However, many questions remain surrounding the role of sleep on physiological function.

The purpose of the current study is to build upon the growing literature focused on sleep and cardiovascular health. More specifically, the current study utilizes a **randomized, crossover**

design to assess the impact of acoustic stimulation (AS) on sleep and cardiac autonomic function to determine if AS during daytime napping can positively impact cardiovascular regulation.

Sleep Architecture

While Rechtschaffen (Rechtschaffen A., 1968) was the first to publish guidelines for sleep stage scoring, the AASM has subsequently developed an updated scoring guide that characterizes sleep staging on EEG, respiratory, and muscle tone characteristics. The AASM scoring guidelines are the most utilized standards for defining stages of sleep in sleep research laboratories today (Berry et al., 2020). NREM and REM sleep staging is critical in distinguishing normal human sleep cycling. A normal cycle of sleep between NREM and REM stages is approximately 90-minutes long (Carskadon & Dement, 2005). The earlier portion of overnight sleep is predominantly NREM, while the later part of the night is characterized by increases of REM sleep (Carskadon & Dement, 2005). The stages of sleep are separated as follows:

Wake

Wakefulness is characterized by a range of low-amplitude EEG brainwave activity primarily consisting of beta (16-32 Hz) and alpha (8-13 Hz) frequencies with eyes open. However, with eye closure alpha rhythm will predominantly occur from the occipital region of the brain.

Stage 1

Sleep onset is usually characterized by the first epoch of stage 1 (N1). The onset of N1 is usually characterized with low-amplitude, mixed-frequency EEG activity that occur

simultaneously with slow eye rolling movements and vertex sharp waves. N1 is generally defined as the transitionary stage from wake to other stages of sleep.

Stage 2

Stage 2 (N2) sleep is well-defined by the occurrence of K complexes and/or sleep spindles. K complexes are characterized by a sharp negative wave followed by a positive inflection wave. Sleep spindles have a frequency of 11-16 Hz and last more than half a second. Most individuals spend approximately 50% of their time asleep in N2.

Stage 3

Stage 3 (N3), also well known as slow wave sleep (SWS), is established when 20% or more of an epoch consists of delta waves (0.5-2 Hz) or slow wave activity. Slow wave activity is characterized by delta frequency and a peak amplitude of great than 75 microvolts (μV). Normal young adults spend around 25% of the night in N3 sleep, predominately in the first half of the night.

REM

REM sleep displays low-amplitude, mixed-frequency activity that is similar to N1 sleep. However, REM sleep also displays a specific brain wave deemed “sawtooth waves”, rapid eye movements, low chin electromyography (EMG), and atonia. In normal young adults, REM sleep is found predominately in the second half of sleep.

Two-Process Model of Sleep Regulation

The two-process model developed by Borbely et al. (Borbély, 1982; Borbély et al., 2016) outlines a model whereby sleep is regulated by both circadian (process C) and homeostatic

(process S) influences that work in tandem with one another (**Figure 1**). The independent effects of these two processes have spurred numerous research questions surrounding how perturbations to either process can impact sleep propensity and quality.

Process C

Process C is driven by an internal biological clock for a circadian rhythm (Borbély, 1982). The primary circadian clock is found within the suprachiasmatic nucleus (SCN) of the hypothalamus. The SCN is largely responsible for maintaining consistent rhythms and release of hormones such as melatonin and cortisol, and also for regulating the cyclical variations of thermoregulatory processes (Gilbert et al., 2004). Evidence for the key role of the SCN in circadian output comes from animal studies whereby destruction of the SCN leads to alterations in the cyclical patterns of core body temperature, cortisol, feeding, among other processes (Abe et al., 1979). Similarly, in vitro studies show that cultured neurons from the SCN maintain cyclical patterns of activity, indicating an inherent circadian rhythmicity in these neurons (Gillette & Reppert, 1987). Measures such as melatonin, core body temperature, and others are generally used in studies assessing process C (Reid, 2019). Furthermore, analyses have also indicated an effect of circadian rhythm on parasympathetic activity at the level of the heart in humans (Burgess et al., 1997; Hilton et al., 2000; Hu et al., 2011).

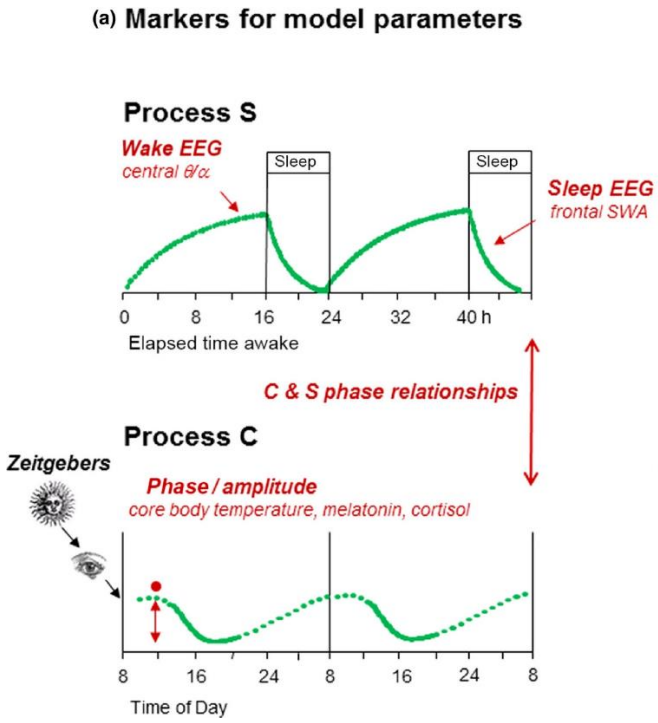

Figure 1: Time Lapse of Process S and Process C.
Process C and process S during regular sleep and waking periods. Process S is the sleep-dependent component, while the process C highlights the circadian dependent curve. Reproduced with permission from (Borbely et al., 2016). Copyright John Wiley & Sons.

Process S

In contrast to process C, process S is regulated by a homeostatic drive and sleep itself (Borbély, 1982). A primary regulator of process S and sleep propensity is through the release of adenosine which builds throughout the day and is subsequently reduced upon sleep attainment. It has been shown that with total sleep deprivation there is a buildup of adenosine (Porkka-Heiskanen et al., 1997) and an increase in slow wave activity (Dijk et al., 1993). Additionally, when caffeine is consumed, which is often accompanied with poor sleep and/or sleep deprivation, it causes a prolonged wakefulness through inhibition of adenosine binding (Ribeiro

& Sebastião, 2010). Lastly, perturbations of process S can lead to autonomic dysfunction (Carter et al., 2012; Carter et al., 2019; Greenlund & Carter, 2022), indicating a role for both process S and process C in autonomic regulation of cardiovascular function.

Sleep and Cardiovascular Health

Receiving the proper amount of overnight sleep is crucial for many physiological processes. However, evidence from large scale epidemiological studies such as the Behavioral Risk Factor Surveillance System (Liu Y, 2016) and the US National Health and Nutrition Examination Survey (Grandner et al., 2014) indicates that a large proportion of the general population are not receiving the recommended nightly sleep allowance. This is important, as insufficient sleep falling below the recommended 7-9 hours of sleep per night can lead to an increased risk in numerous diseases and disorders. Relevant to the present study, individuals that have either acute (Mullington et al., 2009) or chronic (Tobaldini et al., 2017) short sleep have been shown to have a dramatic increase in the risk for cardiovascular disease. Similarly, large-scale associations have been observed between short sleep duration and an increased risk of incident hypertension (Cappuccio et al., 2007; Gangwisch et al., 2006), and an exacerbated cardiovascular dysfunction in individuals with established sleep disorders such as insomnia (Fernandez-Mendoza et al., 2012; Vgontzas et al., 2010). The high prevalence of poor sleep in the general population and its comorbidity with cardiovascular dysfunction highlight the necessity for finding a mechanism underlying these associations, as well as the development of mitigation strategies to offset the adverse cardiovascular outlook associated with short sleep.

Sleep and the Autonomic Nervous System

A suspected mediator of this relationship between poor sleep and the observed increase in cardiovascular disease is the autonomic nervous system (ANS). The ANS is divided into two distinct parts; the sympathetic division, often associated with the “flight-or-fight” response, and the parasympathetic division which is often referred to as the “rest and digest” response. The ANS and sleep are both highly influenced by each other. This is a concept which has been supported through numerous experimental studies (Burgess et al., 1997; Carter et al., 2012; Carter et al., 2019; Grimaldi et al., 2016; Kato et al., 2000; Ogawa et al., 2003; Somers et al., 1993; Trinder et al., 2001).

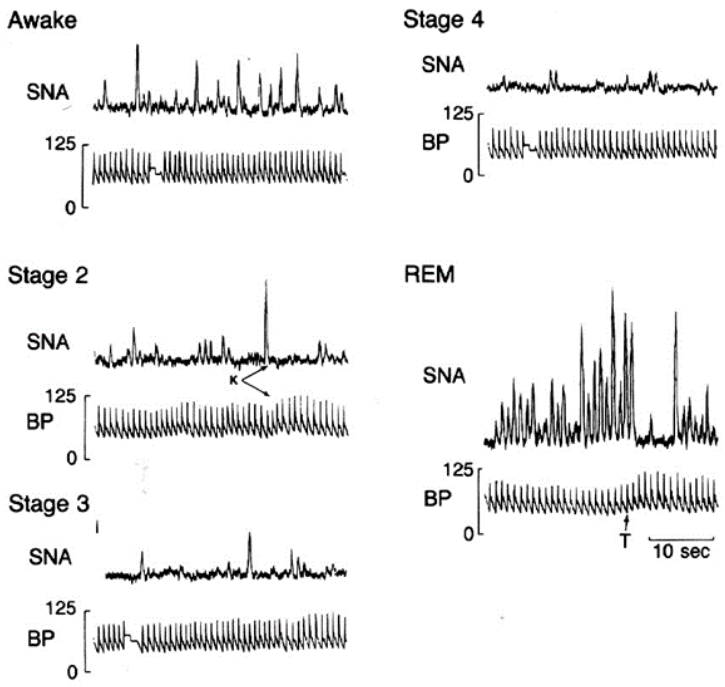

Figure 2: Muscle Sympathetic Nerve Activity and Blood Pressure During Wake and Sleep. Direct recordings of muscle sympathetic-nerve activity (MSNA) and blood pressure (BP) during wake, stages 2, 3, 4, and rapid eye movement (REM) sleep. Reproduced with permission from (Somers et al., 1993), Copyright Massachusetts Medical Society.

Through the use of directly recorded muscle sympathetic nerve activity (MSNA), it has been demonstrated that the sympathetic nervous system (SNS) is significantly altered during various sleep stages (Somers et al., 1993) (**Figure 2**). Specifically, MSNA was elevated during REM sleep and reduced during N2 and SWS compared to wakefulness (Somers et al., 1993), indicative of an inherent relation between sleep process and output of the SNS. Other studies have been conducted to assess whether total sleep deprivation (TSD) in young healthy adults would impact the ANS. 24-hours of TSD resulted in a reproducible increase of morning blood pressure (BP) (Carter et al., 2012; Carter et al., 2019; Kato et al., 2000; Ogawa et al., 2003). This

pressor response was observed with a decrease in MSNA in early studies that primarily examined male participants (Kato et al., 2000; Ogawa et al., 2003), but TSD did not alter MSNA premenopausal women (Carter et al., 2012), and was even increased in postmenopausal women (Carter et al., 2019). These divergent responses of MSNA in females were present despite reductions of MSNA in males (Carter et al., 2012; Carter et al., 2019; Kato et al., 2000; Ogawa et al., 2003) that has been repeatedly observed (Kato et al., 2000; Ogawa et al., 2003). Accordingly, both age and sex appear to impact SNS function during TSD. To date, no studies have assessed a circadian effect on MSNA, likely due to technical limitations associated with this approach. However, some have shown a compounding effect of both short sleep and circadian dysregulation on elevations in SNS activity through indirect measurements (Grimaldi et al., 2016).

The parasympathetic nervous system (PNS), on the other hand, has been repeatedly shown to be influenced by both circadian and homeostatic influences depending on the experimental protocol. Constant routine protocols focused on parasympathetic measurements, such as heart rate and respiratory sinus arrythmia, follow a circadian rhythm even under constant wakefulness (Burgess et al., 1997). However, it has also been shown that HRV is impacted by sleep with HRV increasing with sleep and SWS onset and decreasing with the onset of REM (Trinder et al., 2001). Trinder et al. (2001) additionally showed that HRV was not impacted by the time asleep, but only by sleep stage changes, hinting at a stronger homeostatic regulation of parasympathetic activity. It is likely that parasympathetic regulation is mediated by both circadian and sleep phases, and discrepant findings are likely due to the experimental methodologies. The stance of dual input from both circadian and homeostatic mechanisms on

PNS activity is supported once more by the combined effect of short sleep and circadian misalignment, which resulted in reduced nocturnal cardiac PNS function compared to short sleep alone (Grimaldi et al., 2016). These findings highlight that inadequate and misaligned sleep can impair both nighttime and daytime autonomic function (**Figure 3**). For this reason, it is necessary to explore interventions to compensate for this loss of sleep and reduce its impact on cardiovascular function.

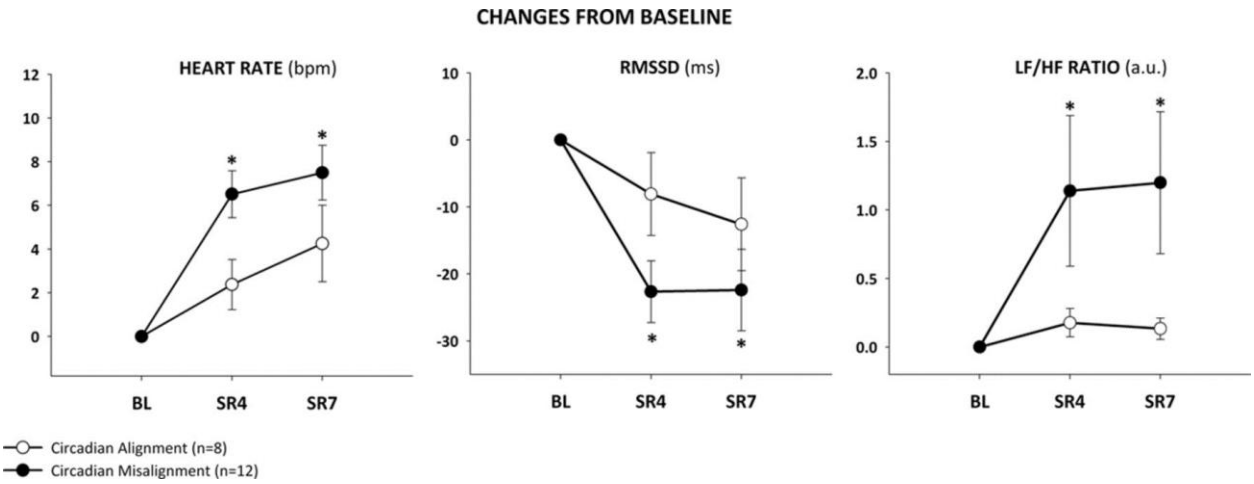

Figure 3: Impact of Sleep Restriction and Circadian Alignment on Heart Rate Variability. Increases in heart rate and low frequency high frequency ratio (LF/HF ratio), and reductions in root mean squared of successive differences (RMSSD) are exaggerated following four (SR4) and seven (SR7) nights of sleep restriction in slow wave sleep compared to baseline (BL). Reproduced for permission from D. Grimaldi, J. R. Carter, E. Van Cauter, R. Leproult, Adverse Impact of Sleep Restriction and Circadian Misalignment on Autonomic Function in Healthy Young Adults, *Hypertension*, Vol. 68, No. 1, 243-250, https://doi.org/10.1161/HYPERTENSIONAHA.115.06847

Napping

With the high rate of poor sleep prevalence within the general population, it is imperative to obtain countermeasures to offset this loss of sleep to prevent or slow the augmented risk of morbidity and mortality. About 81% of U.S. adults have taken a nap within the past three months, with around 75% of the adults that do take an afternoon nap reporting it is due to tiredness or fatigue (Sommer, 2022). With the epidemic of poor sleep in adults, it is important to distinguish whether napping is beneficial or harmful to individuals' overall health. There are mixed findings stating that napping can either be beneficial or detrimental to health. For instance, it has been shown that chronic daytime napping is associated with an increased health risk (Leng

et al., 2014; Liu et al., 2015; Yan et al., 2019), excessive daytime sleepiness (Foley et al., 2007; Hays et al., 1996), and poor nighttime sleep (Melodee Mograss et al., 2022; Owens et al., 2010). In contrast, it has also been reported that afternoon naps improve alertness and performance (Milner & Cote, 2009) and have a beneficial effect on the risk of coronary artery disease in healthy individuals (Naska et al., 2007). Further, findings utilizing HRV have shown that daytime sleep helps modulate parasympathetic activity similarly to that of nighttime sleep, which validates the idea that an afternoon nap could in fact be beneficial for the loss of nighttime sleep and aid in mitigating the increased cardiovascular risks associated with insufficient sleep (AlQatari et al., 2020; Cellini et al., 2018; Cellini et al., 2016).

Since daytime naps elicit a reprieve in cardiac activity and increase parasympathetic activity similar to nighttime sleep (Cellini et al., 2018), investigation into modalities which improve the depth of napping, and potentially augment its positive impact on cardiac autonomic function are warranted. Additionally, the mechanisms underlying what makes a nap beneficial versus detrimental remain equivocal.

Acoustic Stimulation and Sleep

The use of AS has been suspected to be capable of manipulating the microarchitecture of SWS during the night (Fehér et al., 2021; Grimaldi et al., 2019). Having the ability to augment slow wave activity throughout an individual's sleep cycle with the use of AS represents a novel opportunity to enhance slow wave activity and perhaps vagal tone during NREM sleep. It has been shown that with the application of auditory stimulation during sleep both slow wave oscillations (Koo-Poeggel et al., 2022; Ladenbauer et al., 2016; Ngo et al., 2013; Simor et al.,

2018) and slow wave activity can be enhanced (Grimaldi et al., 2019; Huwiler et al., 2022; Ong et al., 2016; Papalambros et al., 2019) indicating increased sleep depth. Further, it has been shown that with the use of AS throughout an overnight study it is possible to improve the amount of PNS activity observed during SWS (Grimaldi et al., 2019). Associated with this increase in PNS throughout overnight sleep, sympathetic activity measured via plasma cortisol release, was decreased the following morning (Grimaldi et al., 2019). These findings are substantial due to the simultaneous effect of AS on both sleep and the ANS which may improve both sleep quality and cardiovascular health.

There is a lack of research dedicated to the potential benefits of AS during the daytime in afternoon napping studies. The few studies that have investigated the potential benefits of noninvasive stimulations on afternoon naps showed that with certain stimulations (i.e., transcranial slow oscillatory stimulation and rhythmic acoustic stimulation) there was an enhancement of slow oscillatory waves (Koo-Poeggel et al., 2022; Ladenbauer et al., 2016; Ngo et al., 2013; Simor et al., 2018). However, the main focus of these studies was to see whether or not enhanced sleep could provide better memory consolidation following an afternoon nap (Koo-Poeggel et al., 2022; Ladenbauer et al., 2016; Ngo et al., 2013). The current paucity of research exploring the effects of AS on afternoon naps, combined with the potential benefits an increase in slow wave activity, underpins the necessity for future investigation within this area.

Summary

Sleep is beneficial to overall human health and function. Many studies have shown that the cardiovascular system can be greatly impacted by sleep (Cappuccio et al., 2007; Gangwisch

et al., 2006; Mullington et al., 2009; Tobaldini et al., 2017). However, a large amount of individuals receive poor or inadequate sleep in the United States (Grandner et al., 2014; Liu et al., 2015), a fact that is more concerning given the association between poor sleep and cardiovascular and metabolic diseases (Cappuccio et al., 2007; Fernandez-Mendoza et al., 2012; Gangwisch et al., 2006; Vgontzas et al., 2010). This has led to a resurgence of efforts directed towards establishing means by which poor sleep can be attenuated. Daytime napping and the use of AS during sleep represents a potential low-cost countermeasure. Some have shown AS to have a beneficial effect on sleep quality, specifically slow wave sleep activity or oscillations (Grimaldi et al., 2019; Huwiler et al., 2022; Koo-Poeggel et al., 2022; Ladenbauer et al., 2016; Ngo et al., 2013; Ong et al., 2016; Papalambros et al., 2019; Simor et al., 2018). Interventions that enhance SWS could positively impact the ANS (Grimaldi et al., 2019). However, there is a lack of studies that implement AS during the daytime to see the potential benefits on the ANS.

Purpose and Hypothesis

The **purpose** of the current study was to assess if the application of AS during an afternoon nap would improve HRV measurements and sleep quality in young healthy adults who habitually receive less than 8 hours of sleep per night. The current study utilizes a **double-blind, randomized, controlled, crossover design** to investigate the impact of AS on sleep HRV and sleep quality in young healthy adults. We **hypothesized** that the implementation of AS during a daytime nap would 1) improve HRV measures with a more pronounced vagal drive throughout NREM sleep stages, and 2) that AS would be associated with an increased slow wave sleep and delta power.

CHAPTER TWO

THE IMPACT OF ACOUSTIC STIMULATION AND DAYTIME NAPPING ON HEART RATE VARIABILITY AND SLEEP ARCHITECTURE

Methods

Subjects

25 young healthy adults (12 men, 13 women, age: 23 ± 1 yrs, BMI: 24 ± 1 kg/m^2) were recruited and enrolled in the study. Recruitment included online advertisements and word-of-mouth at Montana State University and the surrounding area. Subjects were excluded from the study if they were not between 18-30 years old, had a body mass index (BMI) $\geq$ 35 kg/m^2, had unstable or serious medical conditions (i.e., any sleep disorder, cardiovascular or metabolic disease, autonomic dysfunction, hearing impairment, etc.), had a self-imposed irregular sleep schedule, were a habitual smoker or drinker, or were pregnant or breastfeeding. Further, participants were not allowed to currently be taking any cardiovascular or autonomic medications, or any hypnotic or stimulant medications. All females were required to have a regular menstrual cycle (lasting 26-30 days). Lastly, all participants were required to self-report sleeping less than 8 hours per night. 64 participants were recruited and screened for the study. 39 individuals were excluded due to inclusion/exclusion criteria. All subjects were orientated and provided with a written informed consent that was approved by the Montana State University Institutional Review Board.

Experimental Design

Orientation & Initial Questionnaires. Participants completed a brief screening over the phone to determine initial eligibility for the study. After eligibility was confirmed, participants were scheduled for a visit to the laboratory for a detailed orientation session. Participants completed a series of questionnaires through a secure online database after voluntarily signing the written informed consent. The validated questionnaires used to assess anxiety levels and sleepiness were Spielberger State and Trait Anxiety Inventory (STAI) and Epworth Sleepiness Scale (ESS). Participants were also provided an at-home sleep apnea test (ApneaLink, Resmed, San Diego, CA) to confirm the absence of sleep-disordered breathing (i.e., AHI < 15 event/hour). Briefly, the at-home apnea test monitored respiration and blood oxygen saturation throughout the sleep period for one-night in each participant.

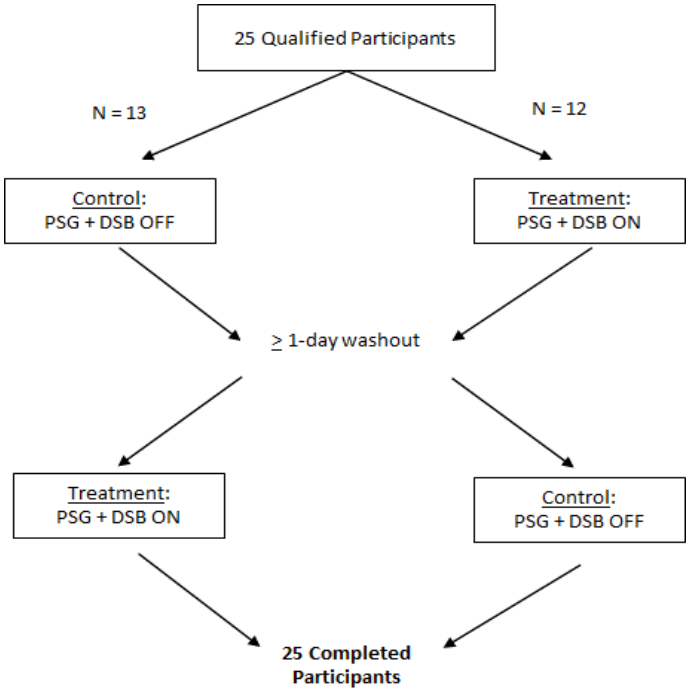

Figure 4: Crossover design used in protocol.

Conditions. Participants were randomized into a controlled crossover design where they received one of two conditions: the control napping condition with no acoustic stimulation (C) or acoustic stimulation (AS) napping condition (**Figure 4**). There was a minimum one-day washout period in-between testing sessions. The AS condition consisted of an auditory frequency (0.8 Hertz monaural beats at 30 decibels) that was emitted from a small device placed at the top of the pillow. In both conditions, participants were given a 90-minute sleep opportunity which was initiated after sleep onset was established. The device was turned on after 5 minutes of stable N2 sleep was observed by trained sleep technicians. After the device was turned on it was not turned off until the 90-minute nap opportunity concluded. The C condition acted as a placebo to the

auditory stimulation with the device still placed at the top of the pillow, although it was not turned on at any time throughout the nap period.

Protocol. Prior to the first scheduled napping session participants were asked to wear an Actigraphy wristwatch (Actiwatch Spectrum Pro, Philips Respironics, Bend, OR) and to fill out a daily subjective sleep diary starting one week prior to the first scheduled napping session and ending after their last napping session. This provided assessment of both subjective and objective habitual sleep. For both conditions (C and AS) participants were asked to refrain from alcohol, exercise, and caffeine 12-hours prior to their scheduled session, and were asked to arrive to the laboratory after fasting for at least 3-hours prior.

Participants arrived at the laboratory between 1:30-2:00 pm for their scheduled afternoon napping sessions. Upon arrival, participants were asked to sit quietly for at least 5 minutes, after which time 3 resting seated BP measurements were taken (Omron, Kyoto, Japan), with the average of these 3 measurements used for all subsequent analysis. Additionally, anthropometric information (i.e., height and weight), and subjective questionnaires assessing sleepiness and anxiety levels were conducted (ESS and STAI). Following the completion of baseline measurements, the participants were fitted with gold-standard polysomnography (PSG) equipment by trained laboratory technicians. The participants then completed a 5-minute resting supine baseline recording before lights out, during which continuous heart rate was monitored. Following lights out, participants were given 20 minutes to fall asleep. If sleep onset was not observed within 20 minutes, the protocol was concluded. Only those participants who fell asleep within 20 minutes in both conditions were included in the final analysis (N=25). The start of sleep onset, determined by trained sleep technicians, initiated the 90-minute timed napping

period. Following the 90-minute period the protocol was completed. Participants were once again asked to sit quietly for 5-minutes upon awakening, after which 3-more seated BP measurements were taken, with the average used in the remainder of analyses. Additionally, participants were asked to once again complete subjective questionnaires assessing sleepiness and anxiety levels (i.e., ESS and STAI). The participant then had at least a one-day wash-out period and arrived to the laboratory once more to complete the opposite protocol condition with the same parameters.

Measures

Polysomnography. PSG (Natus Medical, Middleton, WI) was used to quantify sleep stages. Sleep EEG was conducted based on the guidelines of the AASM, including electrodes placed in pairs of central, frontal, and occipital leads on the scalp. Reference leads were placed contralaterally on the opposite mastoid processes of the head. Electrooculography (EOG) was used to monitor eye movement, while EMG recorded muscle movement on the chin. Respiratory effort was monitored using thoracic and abdominal piezoelectric effort belts. A thermistor and nasal cannula monitored respiratory flow. Pulse oximetry measured blood oxygen concentrations. All PSG scoring of sleep stages, apneic events, and arousals was performed after conclusion of each study by a trained PSG technician (J.R.N.) and confirmed by a board-certified sleep physician. Importantly, the sleep physician who confirmed all scoring was blinded to the experimental conditions.

Heart Rate.Continuous heart rate was recorded with a 5-lead electrocardiogram (ECG) recorded throughout the sleep period at 250 Hz.

Seated Blood Pressure.Participants arrived to the laboratory and were asked to sit quietly for a minimum of 5-minutes before taking three seated BPs on the upper arm (Omron, Kyoto, Japan), which were then averaged to assess their pre nap resting BP. Following the 90-minute nap opportunity participants then rested in a seated position for a minimum of 5-minutes before taking three more BPs that were averaged to assess their post nap resting BP.

Spielberger State Anxiety Inventory (STAI).The STAI is a subjective questionnaire that was filled out prior to and following the 90-minute nap opportunity to assess state anxiety levels. The questionnaire was administered using a Likert scale from 1-4 with 1 indicating "not at all" and 4 indicating "very much so" in different scenarios (i.e., (1) I feel calm (2) I feel secure (3) I feel tense, etc.). The current study only used the state form of the questionnaire, which only assesses state anxiety levels at the time of administration. The STAI has a cutoff of 39-40 for significant state anxiety levels (Julian, 2011).

Epworth Sleepiness Scale (ESS).The ESS is a subjective questionnaire that was filled out prior to and following the 90-minute nap opportunity to assess the level of sleepiness experienced by the participant. The questions were asked on a Likert scale from 0-3, with 0 being "no chance of dozing" and 3 being a "high chance of dozing" in different situations. The following scenarios were used: (1) Sitting and reading (2) Watching TV (3) Sitting inactive in a public place (e.g. a theater or a meeting) (4) As a passenger in a car for an hour without a break (5) Lying down to rest in the afternoon when circumstances permit (6) Sitting and talking to someone (7) Sitting quietly after a lunch without alcohol (8) In a car, while stopped for a few minutes in traffic. ESS total scores can be interpreted as 0-10 normal daytime sleepiness, 11-12

mild excessive daytime sleepiness, 13-15 moderate excessive daytime sleepiness, and 16-24 severe excessive daytime sleepiness.

Data Analysis

Power Spectral Analysis.EEG Spectral Analysis was performed in MATLAB (MathWorks, Natick, MA) using the EEGLAB toolbox (Delorme & Makeig, 2004). The data was pre-processed using a band-pass filter (0.3-35 Hz), referenced to the linked-mastoids, and sampled at 250 Hz. Artifact-free, 30-second epochs of N3 sleep were isolated throughout the nap period. These sections were then used to calculate the absolute power density in delta (Δ; 0.5-4 Hz) frequency across C3/C4, and F3/F4 electrodes, using a Fast Fourier Transform (FFT). All analyses presented were performed using the average absolute power across the central (C3 & C4) and frontal (F3 & F4) leads.

Heart Rate Variability.The continuous ECG recording from the nap was exported into analysis software (Labchart, ADInstruments, Sydney, Australia). R-waves of the ECG were automatically detected and manually confirmed by a trained investigator. This was used to quantify heart rate. HRV was quantified using the methodology of Trinder et al. (Trinder et al., 2001). Briefly, two-minute epochs of stable sleep that were not preceded or followed by sleep disruptions were used in HRV analysis. Spectral analysis was performed on all 2-minute periods of nocturnal ECG of stable sleep periods within each individual. Data were passed through a 60 Hz Notch filter prior to analysis. The integrated area within the high-frequency (HF; 0.15-0.4 Hz) range is reported. Time domain HRV was expressed using the percentage of R-R intervals that varied by 50 ms or more (pNN50) and the root mean squared of successive difference of R-R intervals (RMSSD). These HRV measures were used as a metric to estimate cardiac

parasympathetic activity. After HRV analysis of each 2-minute epoch, these epochs were grouped and averaged into either wake or NREM sleep for each participant. REM was not included as most participants did not achieve stable REM sleep during their 90-minute nap opportunity. The average wake and NREM HRV within each participant was used in subsequent analysis.

Statistical Analysis

All statistics were performed using commercially available software (SPSS Statistics, IBM Corp., Armonk, NY, USA). Descriptive statistics were used to characterize all participant attributes, as well as actigraphy defined sleep quantity and quality. Normality of the data was assessed. Two-tailed paired samples T-tests were used to compare objective PSG between nap conditions. A 2x2 Repeated measures mixed ANOVA was used to assess the main effects of condition (i.e., C vs. AS) and time (i.e., pre-nap vs. post-nap), as well as the interaction of the two effects (i.e., condition x time) on ESS, STAI, and seated BP. Similarly, a 2x2 repeated measures mixed ANOVA was used to assess the main effects of condition (i.e., C vs. AS) and time (i.e., wake vs. NREM), as well as the interaction of the two effects (i.e., condition x time) on HRV. All data are presented as means ± SE. A significance level of $\alpha = 0.05$ was used for all statistical tests.

Results

Participant Characteristics

Table 1 highlights participant characteristics and actigraphy measurements starting from a week prior to their first scheduled experimental study. The average TST (418 ± 9 mins) from

the actigraphy watch indicates that the participants in the current study obtained less than the recommended 7 hours of sleep per night according to the recommendations of the AASM.

	Male	**Female**	**P-Value**
N	12	13	---
Age (yrs)	23 ± 1	22 ± 1	0.294
BMI (kg/m^2)	24 ± 1	24 ± 1	0.835
	Actigraphy		**P-Value**
TST (mins)	430 ± 18	407 ± 6	0.241
Sleep Efficiency (%)	83 ± 2	82 ± 2	0.706
WASO (mins)	40 ± 6	37 ± 2	0.625

Table 1: Participant Characteristics: BMI, body mass index; TST, total sleep time; WASO, wake after sleep onset.

Objective Sleep Results

Table 2 displays the differences between the control and AS conditions in all PSG sleep measurements. AS had no significant effect on the PSG sleep parameters. **Figure 5** shows the individual TST for each participant in the control and AS on conditions.

	Control	Acoustic Stim.	P-Value
TST (min)	73 ± 4	72 ± 4	0.850
Sleep Efficiency (%)	76 ± 5	74 ± 4	0.680
Sleep Onset (min)	7 ± 1	7 ± 1	0.436
WASO (min)	17 ± 4	18 ± 4	0.853
Wake (min)	24 ± 5	26 ± 4	0.737
N1 (min)	18 ± 3	17 ± 2	0.711
N1 (%)	28 ± 5	27 ± 4	0.590
*N2 (min)	32 ± 3	28 ± 3	0.128
*N2 (%)	44 ± 3	38 ± 3	0.061
**SWS (min)	30 ± 4	31 ± 4	0.855
**SWS (%)	36 ± 4	40 ± 5	0.593
**SWS Onset (min)	27 ± 5	28 ± 5	0.944
‡REM (min)	12 ± 2	16 ± 3	0.262
‡REM (%)	15 ± 3	18 ± 4	0.290
‡REM Onset (min)	67 ± 5	65 ± 4	0.776

Table 2: Objective Sleep Assessment: TST, total sleep time; WASO, wake after sleep onset. SWS, slow wave sleep; REM, rapid eye movement; N1, stage 1 sleep; N2, stage 2 sleep. *N=23 with N2 sleep in both conditions; **N=15 with N3 in both conditions; ‡N=12 with REM in both conditions.

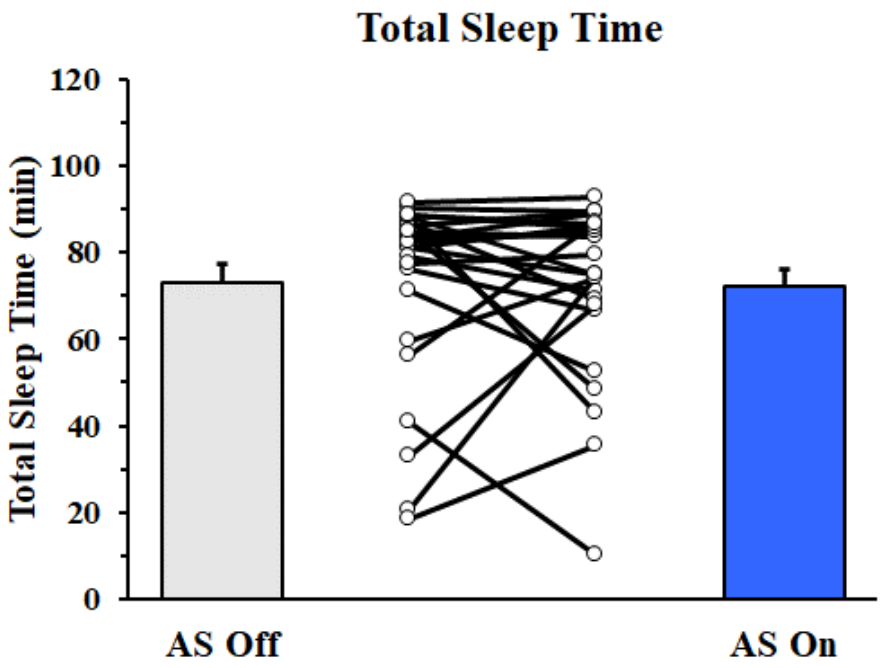

Figure 5**:** Effect of acoustic stimulation (AS) on and total sleep time (TST), with individual points for N = 25.

Sleep Disruptions

Table 3 shows the number of participants that exhibited an arousal and/or stage shift with the initiation of the AS. Of the 13 individuals that had an arousal with the initiation of AS, 12 of them shifted into a lighter stage of sleep (N1, N = 2) or were awoken (N = 10).

	DSB ON
Stage Shift	12/25
Arousal	13/25

Table 3: Stage Shifts and/or Arousals with The Acoustic Stimulation Initiation: Stage shifts and/or arousals associated with the AS device turned on or off throughout the treatment nap condition with all 25 participants in the experimental study.

Table 4 displays general sleep parameters from PSG for the 12 individuals that did not have a sleep disruption with the initial acoustic simulation of the device. There were no significant differences between the experimental conditions for this sub analysis.

	Control	Acoustic Stim.	P-Value
TST (min)	74 ± 7	82 ± 2	0.218
Sleep Efficiency (%)	78 ± 8	84 ± 4	0.270
Sleep Onset (min)	7 ± 2	8 ± 3	0.410
WASO (min)	16 ± 7	8 ± 2	0.206
Wake (min)	24 ± 9	17 ± 4	0.249

Table 4: Objective Sleep Assessment for Individuals with No Sleep Disruption: Sleep statistics for 12/25 participants that did not have a sleep disruption with initiation of acoustic stimulation. TST, total sleep time; WASO, wake after sleep onset.

EEG Spectral Analysis

Table 5 shows the delta power of the 15 individuals that had slow wave sleep in both experimental napping conditions. Due to technological difficulties, data from one participant with SWS was unavailable in the final analysis for the EEG spectral analysis, thus final analysis is N=14.

	Control	Acoustic Stim.	P-Value
C-leads	348 ± 65	369 ± 40	0.727
F-leads	646 ± 132	795 ± 190	0.402

Table 5: EEG Spectral Power: Delta Power: C- leads, central leads; F-leads, frontal leads.

Subjective Assessments

Figure 6 highlights the ESS subjective questionnaire taken prior to and following each nap in both experimental conditions. A 2 (AS off vs. AS on) x 2 (pre vs. post nap) repeated

measures ANOVA indicated a significant time effect of pre vs. post nap on ESS scores (F (1,24) = 6.216, P = 0.020), but no condition effect (F (1,24) = 0.416, P = 0.525) or time × condition interaction (F (1,24) = 0.291, P = 0.594).

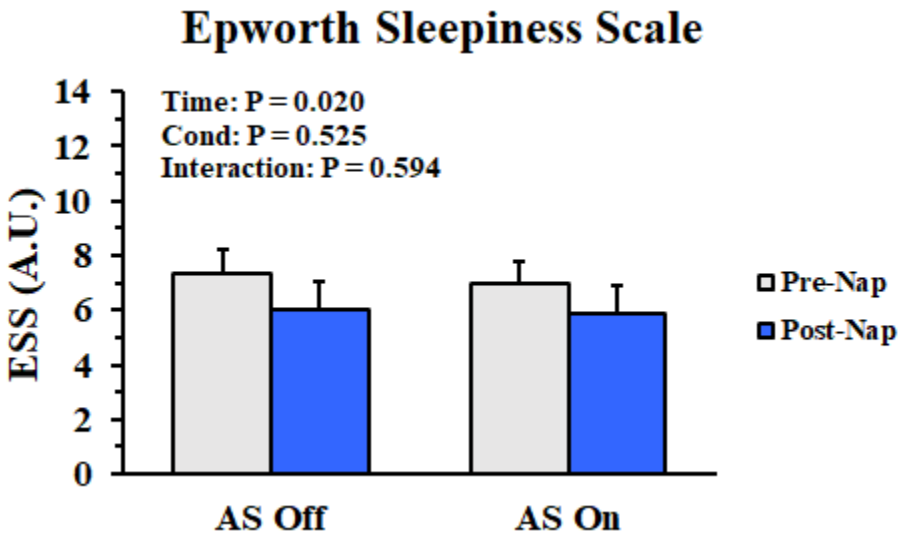

Figure 6: Comparison of the Epworth Sleepiness Scale (ESS) administered before and after each napping conditions (AS Off vs. AS On). AS, Acoustic Stimulation. N=25.

Figure 7 shows the STAI subjective questionnaire assessing state anxiety levels both prior to and following naps in both conditions. A 2 (AS off vs. AS on) x 2 (pre vs. post nap) repeated measures ANOVA indicated a significant time effect of pre vs. post nap on STAI scores (F (1,24) = 8.229, P = .008), but no condition effect (F (1,24) = 0.678, P = .419) or time × condition interaction between the variables was observed (F (1,24) = 0.079, P = .782).

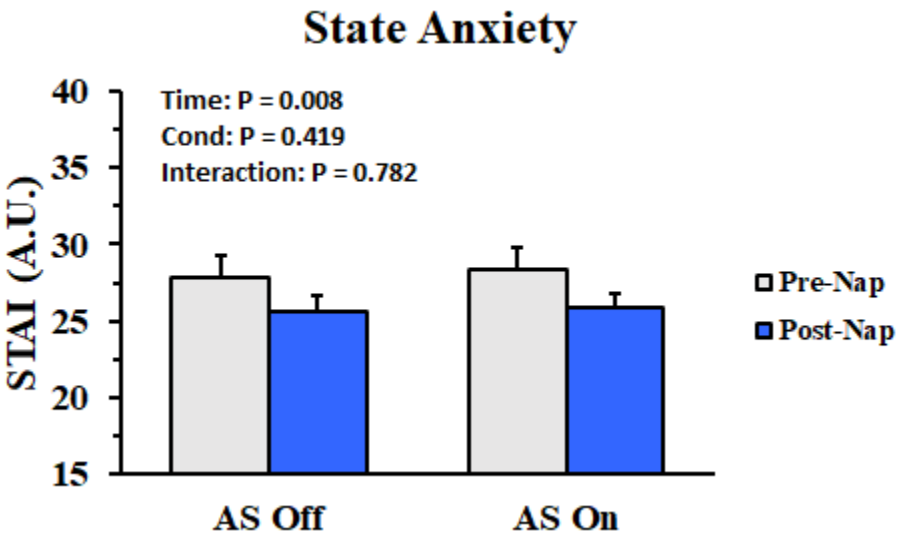

Figure *7*: Comparison of the Spielberger State Anxiety Inventory (STAI) administered before and after each napping conditions (AS Off vs. AS On). AS, Acoustic Stimulation. N=25.

Heart Rate Variability

Table 6 displays the HRV taken prior to the start of the nap during a wake baseline and throughout NREM sleep in both experimental napping conditions. A 2 (Control vs. Acoustic Stim.) x 2 (Wake vs. NREM) repeated measures ANOVA indicated a significant time effect of wake vs. NREM on average RRI ($F(1,19) = 25.683$, $P = < 0.001$). There were no significant time effects of wake vs NREM on pNN50 ($F(1,19) = 1.883$, $P = 0.186$), RMSSD ($F(1,19) = 0.106$, $P = 0.749$), or HF ($F(1,19) = 0.006$, $P = 0.941$). There was a significant condition effect on HF-HRV ($F(1,19) = 5.590$, $P = 0.029$). There was no condition effect or time × condition effects observed in any other variables (All $P > 0.05$).

	Control		Acoustic Stim.		AS Effect	Sleep Effect	AS x Sleep
	Wake	**NREM**	**Wake**	**NREM**	**P-Value**	**P-Value**	**P-Value**
Avg RRI	957 ± 35	1023 ± 40	960 ± 34	1038 ± 36	0.572	< 0.001	0.457
pNN50	37 ± 5	41 ± 5	40 ± 5	47 ± 5	0.101	0.186	0.266
RMSSD	75 ± 8	69 ± 7	81 ± 10	82 ± 9	0.071	0.749	0.415
HF	2451 ± 494	2295 ± 379	3188 ± 672	3286 ± 593	0.029	0.941	0.691

Table 6: Effects of Condition and Sleep on HRV: RRI, R-R interval; pNN50, proportion of RRIs differing by more than 50 ms; RMSSD, root mean square of successive differences; HF, high frequency. N = 20 with both wake and NREM HRV analysis.

Pre vs. Post Nap Blood Pressures

Table 7 shows the BPs taken pre and post nap in both experimental conditions. A 2 (C vs. AS) x 2 (pre vs. post nap) repeated measures ANOVA indicated a significant time effect of pre vs. post nap on MAP ($F(1,24) = 10.857$, $P = 0.003$), DAP ($F(1,24) = 14.483$, $P = 0.001$), but not SAP ($F(1,24) = 2.677$, $P = 0.115$). However, there were no condition effects or time × condition effects observed (All $P > 0.05$).

	Control		Acoustic Stim.		AS Effect	Sleep Effect	AS x Sleep
	Pre	**Post**	**Pre**	**Post**	**P-Value**	**P-Value**	**P-Value**
SAP	115 ± 2	117 ± 2	115 ± 3	117 ± 2	0.902	0.115	0.800
DAP	68 ± 2	71 ± 2	70 ± 2	72 ± 2	0.467	0.001	0.654
MAP	84 ± 1	86 ± 2	84 ± 2	87 ± 2	0.589	0.003	0.862

Table 7: Effect of Condition and Sleep on Blood Pressure: SAP, systolic arterial pressure; DAP, diastolic arterial pressure; MAP, mean arterial pressure. N=25 with pre and post nap blood pressures.

CHAPTER THREE

DISCUSSION AND FUTURE DIRECTIONS

Discussion

The current study aimed to assess the effects of AS on both nap HRV and sleep architecture. We report five new findings. First, a 90-minute daytime nap significantly decreased sleepiness (ESS) and state anxiety (STAI), but these effects were not influenced by AS. Second, there was an overall increase in RRI throughout the afternoon nap in both conditions, but again were not impacted by AS. Third, there was no change in sleep architecture when comparing the two experimental conditions. Fourth, approximately half of the 25 participants experienced an arousal when the AS was initiated during the nap. Lastly, napping resulted in a paradoxical increase of DAP and MAP, regardless of condition. In summary, these findings indicate that daytime napping could benefit individuals' mental health and wellbeing, although the implications for AS on daytime napping sleep architecture and autonomic function remain unclear.

Contrary to our initial hypothesis, AS did not impact parasympathetic HRV measures. We did observe increased RRI during the nap (main effect), consistent with other daytime nap studies that assessed HRV measures (AlQatari et al., 2020; Cellini et al., 2018; Cellini et al., 2016; Chen et al., 2020). Our findings are in contrast with AS during overnight studies, which report that parasympathetic activity via HRV is increased contrary to our findings (Grimaldi et al., 2019). A potential reason for the difference in the current AS study and that of Grimaldi et al. (2019) is the timing of AS administration. These discrepant findings could indicate a more

beneficial impact of AS on HRV when administered during nighttime circadian aligned sleep. Indeed, heart rate has been shown to be lower, and HF-HRV has been shown to be higher during nighttime sleep when compared to daytime naps (Whitehurst et al., 2018). Additionally, using a 72-hour ultradian sleep-wake cycle, Boudreau et al. (2013) observed reduced magnitude of sleep-related changes in heart rate and HRV during daytime sleep when compared to nighttime. When paired with the role of the circadian system in autonomic function (Burgess et al., 1997; Grimaldi et al., 2016), the combination of these findings indicates that the beneficial effects of AS on sleep HRV may be partially dependent on the timing of the sleep opportunity, as daytime sleep results in lessened cardiovascular and autonomic changes when compared to nighttime sleep. Future research would benefit from assessment of the impact of AS timing and subsequent effectiveness at different times of day.

The current study found that sleep architecture was not significantly altered by AS during an afternoon nap, and that delta power throughout SWS was not different. Other studies have report that the use of AS, sleep architecture is positively impacted with increases in slow wave activity (Grimaldi et al., 2019; Huwiler et al., 2022; Koo-Poeggel et al., 2022; Ladenbauer et al., 2016; Ngo et al., 2013; Ong et al., 2016; Papalambros et al., 2019; Simor et al., 2018). Several of these studies were conducted in sleep overnight (Grimaldi et al., 2019; Huwiler et al., 2022; Ngo et al., 2013; Papalambros et al., 2019). The studies that implemented AS during a daytime nap employed different methods for the delivery of the stimulation (Koo-Poeggel et al., 2022; Ladenbauer et al., 2016; Ong et al., 2016; Simor et al., 2018). Closed-loop AS via a personalized algorithm (Koo-Poeggel et al., 2022; Ngo et al., 2013; Ong et al., 2016), transcranial brain stimulation through electrodes placed on the frontal area of the head (Ladenbauer et al., 2016),

and pink-noise through headphones (Simor et al., 2018) were all implemented and resulted in augmented slow wave activity. A potential cause for the lack of significant findings in sleep architecture in the current study with the AS condition could be that half of the participants experienced an arousal when the AS was initiated. Additionally, of the 13 participants that had an arousal with the AS, 12 of the participants shifted into a lighter stage (N1) or were awoken. The shift into the lighter stage of sleep or wakefulness after turning the sound on could have kept the participants from getting a more restorative afternoon nap, and likely impacted the ability of those individuals to enter deeper stages of sleep.

In addition to the impact of arousal on sleep stage and depth, arousals from sleep are also associated with increases in heart rate, potentially through parasympathetic withdrawal (Blasi et al., 2003). Research from our own laboratory has shown this same response, but also that the recovery of heart rate following an arousal is prolonged in habitually short sleepers (Bigalke et al., 2021). This is relevant to the current study, as the average actigraphy defined TST fell below 7 hours, indicating that the current dataset of habitually short sleepers may be susceptible to prolonged heart rate responsiveness to nocturnal arousals. Arousal caused by the application of AS may similarly cause elevated heart rate, and PNS withdrawal, impacting our assessment of sleep HRV within the current dataset.

Anxiety is an increasing concern within the United States, with around 26% of adults experiencing some type of anxiety disorder in their lifetime (Kessler et al., 2005). Further, a large scale study highlighted that individuals that experienced anxiety napped significantly more when compared to healthy counterparts (Léger et al., 2019). The current study observed a decrease in state anxiety after an afternoon nap in both experimental conditions. This finding

informs a potential role for the use of napping to alleviate levels of anxiety in those who have anxiety. However, whether napping harbors similar benefits in individuals with chronic anxiety and anxiety disorders remains to be elucidated. Indeed, cognitive hyperarousal, which likely underscores many anxiety disorders, impairs sleep's beneficial impacts and can lead to further chronic sleep disturbance (Bonnet & Arand, 2010; Kalmbach et al., 2018; Riemann et al., 2010). Hyperarousal increases brain wake activity during sleep (Fernandez-Mendoza et al., 2016; Spiegelhalder et al., 2012), which may potentially reduce the beneficial impact of daytime napping observed in the present study in populations who experience chronic anxiety and hyperarousal. While our study indicates that napping may acutely improve symptoms of anxiety, the frequency and timing of naps has also been associated with impairments in nighttime sleep (M. Mograss et al., 2022; Monk et al., 2001), which have the potential to worsen anxiety symptoms. Assessment of nap timing, duration, and strategic application is necessary to fully explain the potential benefits of napping in individuals with anxiety disorders while at the same time mitigating negative outcomes in nighttime sleep.

Limitations

There are limitations to acknowledge in the current study. First, there was no familiarization nap completed prior to the experimental napping conditions. The absence of a familiarization nap could have led to a more disrupted first nap in the sleep laboratory. However, the randomized, crossover design helps mitigate this concern. Second, this study was only conducted in young healthy adults, with most of the participants volunteering from the university student population. It would be beneficial to conduct this experiment in a larger more diverse sample size. Third, there was no individualized volume control utilized for the decibel level of

the AS device. Allowing each participant to have an adjusted volume for the AS could have resulted in much smaller amount of the participants to experience arousal when the AS was initiated. Lastly, there was no HRV measurements recorded following the 90-minute afternoon nap. This limitation does not allow for the current study to assess if wake HRV measures were positively impacted following the daytime napping period.

Future Directions

Future work is necessary to fully understand the interaction between AS, HRV, and daytime sleep. In the current study, nearly half of the participants were disturbed by the AS turning on. It has previously been shown that with a spontaneous nocturnal arousal there is an increase in heart rate (Bigalke et al., 2021), an effect that is prolonged in short sleeping adults, such as those recruited in the current study. With the current method of AS provoking spontaneous arousals in approximately half of the participants, this could potentially cause a decrease in PNS activity and poorer overall sleep quality during the nap. Future research may be necessary to assess individualized AS application strategies which do not result in wakefulness or arousals.

Previous studies which have shown beneficial effects from AS on SWS often use closed-loop stimulation methodologies (Grimaldi et al., 2019; Koo-Poeggel et al., 2022; Ngo et al., 2013). Additionally, intermittent stimulation as opposed to continuous stimulation has been shown to have a greater benefit on SWS architecture (Lustenberger et al., 2022). Future studies would benefit from comparing closed-loop stimulation verses external entrainment stimulation, as well as differing stimulation frequencies, intensities, among other factors which may reduce habituation.

Next, an assessment of time of day application strategies might help to disentangle potential circadian influences on the efficacy of AS in improving both sleep quality and HRV. AS improves HRV and slow wave activity when applied at night (Grimaldi et al., 2019), but not when applied during a daytime nap as in the current study. This may be partially due to effects of sleep timing on sleep-related changes in HRV measures and depth of sleep (Boudreau et al., 2013; Whitehurst et al., 2018). Future assessment of circadian effects on AS efficacy appears warranted.

Future studies should consider investigating a wider population range to include individuals with sleep disorders and/or comorbidities such as hypertension. AS and napping could have significant beneficial effects in individuals that are within an unhealthy population that have impaired vagal control such as hypertension (Singh et al., 1998) or anxiety disorders (Chalmers et al., 2014). Additionally, AS may benefit populations with chronic hyperarousal, in whom napping alone may not offer the autonomic benefits that healthy adults experience due to elevated arousal throughout the sleep period (Fernandez-Mendoza et al., 2016; Spiegelhalder et al., 2012).

Lastly, alternative means of sleep modulation are warranted. For instance, administration of differing odors has been associated with increased delta activity during NREM sleep in healthy adults (Perl et al., 2016). This modality of administration did not cause arousal within the subjects tested (Perl et al., 2016). Other sensory stimuli apart from AS may offer beneficial impacts on both sleep and HRV.

Conclusion

In conclusion, the current study aimed to assess the impact of AS on autonomic function and sleep architecture. Contrary to the initial hypothesis, AS did not significantly impact sleep architecture or HRV measures. However, this may be secondary to unintended increases in arousals during AS administration in approximately 50% of individuals. Although AS did not have an impact on autonomic function or sleep architecture, afternoon napping improved overall sleepiness and state anxiety levels. Further, napping offered a reprieve in cardiac activity as evidenced by an increase in RRI in both experimental conditions. These findings support a beneficial effect of napping on psychological wellbeing and sleepiness, although the potential impacts of AS on napping quality remain equivocal.

APPENDICES

APPENDIX A

DEMOGRAPHICS

Subject ID	Age	BMI	Sex
1	20	22.20	F
2	21	22.63	F
3	25	25.83	M
4	26	25.06	M
5	19	22.50	F
6	21	21.98	M
7	25	29.11	M
8	27	22.89	M
9	27	33.85	F
10	24	22.74	F
11	23	30.59	F
12	29	21.37	M
13	22	26.86	M
14	22	24.28	M
15	28	27.22	F
16	20	25.59	F
17	18	26.93	M
18	23	23.49	M
19	22	24.58	F
20	22	18.00	F
21	20	18.40	F
22	19	23.38	F
23	25	20.16	M
24	18	23.80	M
25	22	20.31	F

Table A1: Demographics: Participant demographics within the current study.

APPENDIX B

ACTIGRAPHY

Subject ID	TST (mins)	SE (%)	WASO (mins)
1	418	79	43
2	431	87	27
3	562	84	39
4	432	82	48
5	403	79	57
6	391	71	58
7	447	85	30
8	431	90	27
9	383	81	29
10	395	80	38
11	436	91	27
12	463	86	34
13	379	85	25
14	407	81	26
15	376	84	32
16	412	68	44
17	325	83	21
18	409	73	97
19	416	88	28
20	394	85	40
21	447	86	38
22	396	81	42
23	515	85	46
24	402	90	29
25	382	77	34

Table B1: Actigraphy: Objective actigraphy for each participant within the current study.

APPENDIX C

POLYSOMNOGRAPHY SLEEP MEASUREMENTS

	TST (mins)		SE (%)		WASO (mins)		SOL (mins)	
Subject ID	**Control**	**AS**	**Control**	**AS**	**Control**	**AS**	**Control**	**AS**
1	81	75	83	80	9	15	7.7	4.6
2	90	89.5	95	97	0.5	0.5	4	1.9
3	76	66.5	81	72	14	23.5	4.1	2.5
4	81	86	89	92	9	4	1.6	3.3
5	79	71	71	65	10.5	19.5	22.4	19.7
6	88	43	94	41	2.5	47.5	2.9	15.4
7	87	69	93	72	4.5	20.5	2	5.9
8	20.5	74.5	19	69	69.5	18.5	15.4	15.8
9	59.5	74	61	80	30.5	16	7.3	3.0
10	83.5	84	90	92	6.5	6.5	3.3	0.9
11	71	52.5	74	54	19	37.5	6	7.4
12	83	89	90	96	6.5	2	2.7	1.9
13	86	89.5	92	98	4.5	0.5	3.4	1.4
14	41	10	44	11	49	80.5	4.2	2.4
15	85	83.5	85	83	4.5	6	10.5	11.0
16	88	75	95	80	2	14.5	3	4.2
17	82.5	85	89	93	8	5	1.7	1.6
18	77.5	79.5	82	81	13	10.5	4.7	8.6
19	85	48.5	93	51	3.5	41.5	3.8	5.8
20	18.5	35.5	19	37	75.5	54.5	4.3	5.8
21	91.5	92.5	99	99	0.5	0.5	0.6	0.4
22	33	68	29	60	55.5	21	23.6	23.2
23	56	87	53	75	35	3	15.4	26.1
24	88.5	85.5	89	91	2.5	4.5	8.7	4.9
25	88.5	86.5	98	94	1	3.5	0.6	1.9

Table 1C: Polysomnography Measurements: Objective PSG sleep measurements for participants.

APPENDIX D

SLEEP STAGES: PERCENT

	N1 (%)		N2 (%)		N3 (%)		REM (%)	
Subject ID	**Control**	**AS**	**Control**	**AS**	**Control**	**AS**	**Control**	**AS**
1	42.6	25.3	13	12.7	44.4	62	0	0
2	8.3	25.1	27.8	49.7	57.2	17.3	6.7	7.8
3	19.7	21.1	57.2	56.4	0	10.5	23	12
4	15.4	16.9	64.2	61.6	20.4	21.5	0	0
5	16.5	13.4	48.1	13.4	35.4	73.2	0	0
6	20.5	27.9	53.4	29.1	26.1	43	0	0
7	48.9	55.1	45.4	44.9	5.7	0	0	0
8	34.1	19.5	65.9	36.2	0	44.3	0	0
9	44.5	39.2	50.4	50	5	0	0	10.8
10	13.2	9.5	39.5	46.4	38.9	38.1	8.4	6
11	100	56.2	0	34.3	0	9.5	0	0
12	17.5	12.9	68.1	43.3	3	1.1	11.4	42.7
13	12.2	3.9	43.6	40.2	25	35.8	19.2	20.1
14	25.6	45	74.4	55	0	0	0	0
15	12.9	9	27.1	32.9	48.8	34.1	11.2	24
16	19.3	57.3	44.9	42.7	14.8	0	21	0
17	15.8	8.8	30.3	39.4	45.5	37.6	8.5	14.1
18	39.4	58.5	29.7	22	0	0	31	19.5
19	14.1	36.1	24.1	51.5	61.8	12.4	0	0
20	100	59.2	0	38	0	2.8	0	0
21	7.7	5.9	41.5	35.7	41	50.8	9.8	7.6
22	39.4	19.9	40.9	8.1	19.7	72.1	0	0
23	27.7	14.4	58.9	35.1	11.6	46	1.8	4.6
24	12.4	11.1	32.2	23.4	36.7	42.1	18.6	23.4
25	4.5	11.6	40.7	37	29.9	14.5	24.9	37

Table 1D: Sleep Stages: Percent: Percentage of time spent in each stage of sleep for all participants within the current study.

APPENDIX E

SLEEP STAGES: TOTAL MINUTES

	N1 (mins)		N2 (mins)		N3 (mins)		REM (mins)	
Subject ID	**Control**	**AS**	**Control**	**AS**	**Control**	**AS**	**Control**	**AS**
1	34.5	19	10.5	9.5	36	46.5	0	0
2	7.5	22.5	24	44.5	51.5	15.5	6	7
3	15	14	43.5	37.5	0	7	17.5	8
4	12.5	14.5	52	53	16.5	18.5	0	0
5	13	9.5	38	9.5	28	52	0	0
6	18	12	47	12.5	23	18.5	0	0
7	42.5	38	39.5	31	5	0	0	0
8	7	14.5	13.5	27	0	33	0	0
9	26.5	29	30	37	3	0	0	8
10	11	8	33	39	32.5	32	7	5
11	71	29.5	0	18	0	5	0	0
12	14.5	11.5	56.5	38.5	2.5	1	9.5	38
13	10.5	3.5	37.5	36	21.5	32	16.5	18
14	10.5	4.5	30.5	5.5	0	0	0	0
15	11	7.5	23	27.5	41.5	28.5	9.5	20
16	17	43	39.5	32	13	0	18.5	0
17	13	7.5	25	33.5	37.5	32	7	12
18	30.5	46.5	23	17.5	0	0	24	15.5
19	12	17.5	20.5	25	52.5	6	0	0
20	18.5	21	0	13.5	0	1	0	0
21	7	5.5	38	33	37.5	47	9	7
22	13	13.5	13.5	5.5	6.5	49	0	0
23	15.5	12.5	33	30.5	6.5	41	1	4
24	11	9.5	28.5	20	32.5	36	16.5	20
25	4	10	36	32	26.5	12.5	22	32

Table 1E: Sleep Stages: Total Minutes: Amount of time in minutes spent in each stage of sleep for all participants within the current study.

APPENDIX F

EEG SPECTRAL POWER: DELTA POWER AVERAGES

	Central Leads		Frontal Leads	
Subject ID	**Control**	**AS**	**Control**	**AS**
1	147.21	444.02	758.74	1613.41
2	763.26	311.84	119.80	128.94
3				
4	627.41	632.87	1780.24	1406.78
5	275.50	572.69	887.23	2163.47
6	482.53	510.15	1085.12	1208.48
7				
8				
9				
10	179.45	230.55	725.17	915.92
11				
12				
13	231.50	336.16	106.83	119.85
14				
15	851.71	479.25	357.64	1187.09
16				
17	300.68	460.46	1007.33	203.63
18				
19	389.16	261.94	1092.84	115.60
20				
21				
22	116.83	346.60	356.10	1495.58
23	110.30	249.90	59.57	121.28
24	274.59	213.70	191.82	82.73
25	124.03	120.92	519.69	368.71

Table 1F: EEG Spectral Power: Delta Power Averages for each participant that had stage 3 sleep in both napping conditions.

APPENDIX G

EPWORTH SLEEPINESS SCALE SCORES

	Control		Acoustic Stim.	
Subject ID	**Pre-Nap**	**Post-Nap**	**Pre-Nap**	**Post-Nap**
1	1	1	1	1
2	7	8	8	8
3	5	0	3	0
4	7	5	6	6
5	6	7	7	7
6	17	12	18	17
7	3	3	3	3
8	12	13	12	12
9	5	5	3	4
10	3	3	5	5
11	9	5	4	5
12	14	4	11	1
13	7	7	7	7
14	4	2	5	2
15	1	1	2	1
16	12	9	9	9
17	9	7	9	8
18	9	6	12	12
19	7	7	6	7
20	4	4	4	4
21	14	9	7	1
22	10	11	10	9
23	9		9	9
24	0	0	1	1
25	9	11	12	8

Table 1G: Epworth Sleepiness Scale Scores: Subjective sleepiness scores for each participant within the current study.

APPENDIX H

SPIELBERGER STATE ANXIETY INVENTORY SCORES

	Control		Acoustic Stim.	
Subject ID	**Pre-Nap**	**Post-Nap**	**Pre-Nap**	**Post-Nap**
1	20	21	20	20
2	28	30	32	28
3	22	20	22	20
4	33	33	33	30
5	23	21	23	21
6	25	20	23	20
7	20	23	20	20
8	20	23	20	20
9	32	22	26	25
10	39	29	44	39
11	32	30	35	31
12	26	28	30	27
13	25	23	26	25
14	24	25	23	22
15	36	36	37	31
16	47	20	37	29
17	22	21	23	22
18	29	25	29	22
19	31	31	36	33
20	27	23	24	22
21	31	31	38	28
22	20	24	24	22
23	35	28	36	33
24	20	20	20	21
25	30	34	29	35

Table 1H: Spielberger State Anxiety Inventory Scores: Subjective state anxiety scores for each participant within the current study.

APPENDIX I

HEART RATE VARIABILITY MEASUREMENTS

	AVG RRI		pNN50		RMSSD		HF	
Subject ID	**Wake**	**NREM**	**Wake**	**NREM**	**Wake**	**NREM**	**Wake**	**NREM**
1	1054.46	1214.89	33.40	39.88	55.94	54.74	1089.73	1071.83
2	788.00	912.61	28.56	54.50	82.48	75.37	3681.52	2434.86
3	1228.12	1289.71	58.66	61.44	109.74	102.20	3551.07	3331.04
4	1359.37	1451.88	70.73	64.52	102.78	104.97	3652.47	4491.17
5	996.82	1065.42	13.45	10.40	34.63	35.16	611.03	562.28
6	1128.19	1211.90	41.15	60.02	104.20	115.26	3293.16	5981.91
7	943.81	1026.63	60.42	59.67	85.52	89.77	3061.85	3270.01
8	963.08	956.69	22.86	11.09	39.82	31.51	453.78	262.47
9	839.25	853.21	30.89	30.58	49.33	43.85	735.37	860.80
10	803.24	915.05	18.12	42.91	66.85	54.90	3519.42	1502.74
11								
12	879.46	973.32	23.41	42.24	45.81	118.92	936.91	4646.04
13	1059.20	1182.19	57.95	66.36	108.13	103.58	3153.58	4422.68
14	974.02	1220.06	46.66	65.98	97.75	96.84	6302.01	3945.39
15	933.72	989.97	46.48	59.87	61.92	77.16	1432.49	2003.93
16	674.20	698.49	26.94	26.13	55.54	48.90	1213.89	1457.37
17	968.88	898.14	65.45	35.79	132.87	67.25	5564.37	1518.02
18								
19	883.18	907.78	62.80	52.81	142.72	89.99	9271.47	3330.86
20								
21								
22	945.19	889.10	21.58	1.00	41.18	27.43	698.80	116.91
23	824.49	1035.17	13.28	50.82	73.67	72.91	539.88	2619.85
24	1008.85	1131.53	49.25	40.53	90.93	69.26	2540.42	2001.78
25	848.56	853.95	0.00	0.13	6.56	6.34	14.96	9.84

Table 1I: Heart Rate Variability Measurements: Control: Heart rate variability measurements for participants in the control condition. RRI, R-R interval; pNN50, proportion of RRIs differing by more than 50 ms; RMSSD, root mean square of successive differences; HF, high frequency. N = 20 with both wake and NREM HRV analysis.

	AVG RRI		pNN50		RMSSD		HF	
Subject ID	**Wake**	**NREM**	**Wake**	**NREM**	**Wake**	**NREM**	**Wake**	**NREM**
1	925.67	1089.01	25.52	51.71	46.43	65.74	884.96	1603.78
2	865.17	1033.65	50.54	74.17	70.15	138.75	3295.41	7390.45
3	1179.13	1317.25	48.73	58.93	97.54	95.16	6519.52	3199.73
4	1414.37	1505.06	66.67	61.46	113.04	96.87	5497.24	4204.22
5	895.92	991.99	16.41	6.34	38.70	27.50	582.65	205.19
6	903.40	1142.50	45.52	60.87	136.27	116.26	3859.56	6170.30
7	901.70	1027.71	44.19	60.93	67.22	94.86	1991.81	3204.68
8	989.40	1039.40	15.89	11.32	34.06	32.54	358.68	301.77
9	729.20	850.04	1.23	42.45	19.68	57.93	65.44	1249.12
10	865.78	999.47	29.59	71.54	43.51	121.85	943.91	7015.97
11	871.89	945.59	49.65	48.73	82.49	88.93	2721.06	2188.37
12	990.67	1044.29	67.36	66.71	141.68	154.53	8910.88	8228.76
13	1076.42	1055.06	51.59	54.46	82.62	72.40	1205.01	1813.07
14								
15	885.74	1000.58	33.55	64.27	55.30	86.98	1171.92	2775.22
16	736.66	729.37	47.20	25.81	72.13	49.72	2676.35	1408.29
17	1047.46	1056.80	68.87	51.08	121.95	77.64	5119.01	2168.87
18								
19	947.32	901.74	71.97	62.97	184.38	152.19	11107	7771.13
20	1221.63	1168.12	68.04	38.84	138.87	68.55	11052.6	2334.82
21								
22	933.52	897.58	22.07	0.90	42.69	16.04	708.54	56.29
23	884.06	997.53	33.58	60.02	83.43	78.63	4149.08	3091.66
24	1062.13	1120.02	49.61	44.43	116.57	78.42	3452.02	3049.91
25	972.61	979.78	5.80	5.43	43.81	26.45	1259.36	820.62

Table 2I: Heart Rate Variability Measurements: Acoustic Stimulation: Heart rate variability measurements for participants in the acoustic stimulation (AS) condition. RRI, R-R interval; pNN50, proportion of RRIs differing by more than 50 ms; RMSSD, root mean square of successive differences; HF, high frequency. N = 20 with both wake and NREM HRV analysis.

APPENDIX J

BLOOD PRESSURES

	SAP		DAP		MAP	
Subject ID	**Pre-Nap**	**Post-Nap**	**Pre-Nap**	**Post-Nap**	**Pre-Nap**	**Post-Nap**
1	128	126	65	64	86	85
2	106	112	69	74	81	87
3	106	115	61	56	76	76
4	119	121	74	81	89	94
5	117	119	63	69	81	86
6	114	124	66	72	82	89
7	112	116	66	69	81	85
8	106	121	67	73	80	89
9	103	119	69	79	80	92
10	101	96	60	59	74	71
11	101	97	64	70	76	79
12	121	124	78	77	92	93
13	124	108	46	53	72	71
14	146	143	83	84	104	104
15	99	87	69	60	79	69
16	116	120	71	70	86	87
17	110	116	61	68	77	84
18	113	118	62	63	79	81
19	113	122	82	82	92	95
20	119	120	75	76	90	91
21	108	111	69	77	82	88
22	135	129	67	67	90	88
23	117	120	72	74	87	89
24	125	123	70	74	88	90
25	124	121	80	78	95	92

Table 1J: Blood Pressures: Control: SAP, systolic arterial pressure; DAP, diastolic arterial pressure; MAP, mean arterial pressure. N=25 with pre and post nap blood pressures.

	SAP		DAP		MAP	
Subject ID	**Pre-Nap**	**Post-Nap**	**Pre-Nap**	**Post-Nap**	**Pre-Nap**	**Post-Nap**
1	119	125	63	76	82	92
2	111	115	75	78	87	90
3	108	106	53	58	71	74
4	117	125	68	72	84	90
5	125	107	71	65	89	79
6	128	120	74	72	92	88
7	114	109	62	64	79	79
8	113	116	71	78	85	91
9	117	114	76	82	90	93
10	96	103	60	61	72	75
11	95	99	57	66	70	77
12	121	128	70	78	87	95
13	108	118	58	60	75	79
14	157	147	93	86	114	106
15	95	91	65	61	75	71
16	116	112	71	67	86	82
17	122	128	73	76	89	93
18	116	120	65	71	82	87
19	111	119	82	89	92	99
20	113	110	63	67	80	81
21	100	101	65	73	77	82
22	129	131	63	66	85	88
23	121	128	76	76	91	93
24	115	125	65	62	82	83
25	120	121	84	94	96	103

Table 2J: Blood Pressures: Acoustic Stimulation: SAP, systolic arterial pressure; DAP, diastolic arterial pressure; MAP, mean arterial pressure. N=25 with pre and post nap blood pressures.

APPENDIX K

SEX DIFFERENCES-PARTICIPANT CHARACTERISTICS

Descriptive Statistics					
	Sex	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**
Age	M	12	23.4167	3.39675	0.98056
	F	13	22.0769	2.84199	0.78823
BMI	M	12	24.3126	2.58694	0.74679
	F	13	23.9991	4.50900	1.25057
Actigraphy TST (mins)	M	12	430.3172	62.62729	18.07894
	F	13	406.9082	22.06185	6.11885
Actigraphy SE (%)	M	12	83.0279	5.62181	1.62288
	F	13	82.1436	5.91381	1.64020
Actigraphy WASO (mins)	M	12	39.9409	21.03325	6.07178
	F	13	36.7972	8.64811	2.39855

Table 1K: Sex Differences – Participant Characteristics: Descriptive Statistics

Independent Samples T-Test – Sex Differences								
	F	**Sig.**	**t**	**df**	**P-Value**	**Std. Error Difference**	**Lower**	**Upper**
Age	0.788	0.384	1.073	23	0.294	1.24886	-1.24371	3.92320
BMI	2.064	0.164	0.211	23	0.835	1.48757	-2.76373	3.39080
Actigraphy TST (mins)	4.965	0.036	1.226	13.502	0.241	19.08634	-17.66920	64.48726
Actigraphy SE (%)	0.088	0.769	0.382	23	0.706	2.31225	-3.89899	5.66752
Actigraphy WASO (mins)	3.622	0.070	0.496	23	0.625	6.33724	-9.96583	16.25332

Table 2K: Sex Differences – Participant Characteristics: Independent Samples T-Test

APPENDIX L

PAIRED SAMPLES T-TEST: SLEEP CHARACTERISTICS

Group Statistics					
	Condition	**Mean**	**N**	**SD**	**SEM**
TST (mins)	AS	72.0000	25	20.07382	4.01476
	Control	72.8200	25	21.92168	4.38434
SE (%)	AS	74.3720	25	22.22661	4.44532
	Control	76.1960	25	24.52545	4.90509
SOL (mins)	AS	7.1840	25	7.26560	1.45312
	Control	6.5560	25	6.31078	1.26216
WASO (mins)	AS	18.2600	25	20.00267	4.00053
	Control	17.4600	25	22.15801	4.43160
Wake (mins)	AS	25.9200	25	21.89210	4.37842
	Control	24.4000	25	25.76941	5.15388
N1 (mins)	AS	16.9600	25	11.79859	2.35972
	Control	17.8600	25	14.27010	2.85402
N1 (%)	AS	26.5120	25	18.66673	3.73335
	Control	28.4880	25	24.85279	4.97056
N2 (mins)	AS	27.6957	23	12.76691	2.66209
	Control	31.9783	23	11.99003	2.50009
N2 (%)	AS	37.6826	23	14.38445	2.99937
	Control	44.4043	23	15.69257	3.27213
N3 (mins)	AS	31.1333	15	14.43021	3.72586
	Control	30.0000	15	13.85125	3.57638
N3 (%)	AS	40.0333	15	19.11255	4.93484
	Control	36.1600	15	14.22833	3.67374
SWS Latency (mins)	AS	27.7000	15	18.30866	4.72728
	Control	27.2333	15	17.77023	4.58825
REM (mins)	AS	15.5417	12	10.79027	3.11488
	Control	12.1250	12	7.02633	2.02833
REM (%)	AS	18.2333	12	12.17011	3.51321
	Control	14.5417	12	8.68368	2.50676
REM Latency (mins)	AS	65.29	12	13.236	3.821
	Control	66.79	12	17.740	5.121

Table 1L: Paired Samples T-Test: Sleep Characteristics: Group Statistics

Paired Samples T-Test						
	Lower 95%	**Upper 95%**	**t**	**df**	**One sided P-Value**	**Two-sided P-value**
TST (mins)	-9.66850	8.02850	-0.191	24	0.425	0.850
SE (%)	-10.82874	7.18074	-0.418	24	0.340	0.680
SOL (mins)	-1.00711	2.26311	0.793	24	0.218	0.436
WASO (mins)	-8.02201	9.62201	0.187	24	0.427	0.853
Wake (mins)	-7.71820	10.75820	0.340	24	0.369	0.737
N1 (mins)	-5.86123	4.06123	-0.374	24	0.356	0.711
N1 (%)	-9.44837	5.49637	-0.546	24	0.295	0.590
N2 (mins)	-9.90000	1.33479	-1.581	22	0.064	0.128
N2 (%)	-13.78772	0.34424	-1.973	22	0.031	0.061
N3 (mins)	-11.89965	14.16632	0.187	14	0.427	0.855
N3 (%)	-11.30704	19.05371	0.547	14	0.296	0.593
SWS Latency (mins)	-13.53108	14.46441	0.072	14	0.472	0.944
REM (mins)	-2.94401	9.77734	1.182	11	0.131	0.262
REM (%)	-3.61448	10.99781	1.112	11	0.145	0.290
REM Latency (mins)	-12.834	9.834	-0.291	11	0.388	0.776

Table 2L: Paired Samples T-Test: Sleep Characteristics

APPENDIX M

HEART RATE VARIABILITY STATISTICS

Group Statistics					
	Condition	**Mean**	**N**	**SD**	**SEM**
Wake HF	AS	3187.92	20	3004.55	671.84
	Control	2450.81	20	2209.94	494.16
Wake RMSSD	AS	80.56	20	43.48	9.72
	Control	74.53	20	35.14	7.86
Wake pNN50	AS	39.79	20	20.98	4.69
	Control	37.27	20	20.54	4.59
Wake Avg RRI	AS	960.32	20	150.98	33.76
	Control	956.50	20	158.41	35.42
NREM HF	AS	3286.45	20	2652.36	593.09
	Control	2294.82	20	1692.85	378.53
NREM RMSSD	AS	82.02	20	40.80	9.12
	Control	69.48	20	31.51	7.04
NREM pNN50	AS	46.79	20	23.55	5.27
	Control	40.53	20	21.22	4.75
NREM Avg RRI	AS	1038.94	20	161.58	36.13
	Control	1022.88	20	178.88	39.99

Table 1M: Heart Rate Variability Group Descriptive Statistics: Group Statistics

Multivariate Tests						
		Value	**F**	**Hypothesis df**	**Error df**	**Sig.**
DSB	Pillai's Trace	0.017	.331	1.000	19.000	0.572
	Wilks' Lambda	0.983	.331	1.000	19.000	0.572
	Hotelling's Trace	0.017	.331	1.000	19.000	0.572
	Roy's Largest Root	0.017	.331	1.000	19.000	0.572
Sleep	Pillai's Trace	0.575	25.68	1.000	19.000	0.000
	Wilks' Lambda	0.425	25.68	1.000	19.000	0.000
	Hotelling's Trace	1.352	25.683b	1.000	19.000	0.000
	Roy's Largest Root	1.352	25.68	1.000	19.000	0.000
DSB x Sleep	Pillai's Trace	0.029	.576	1.000	19.000	0.457
	Wilks' Lambda	0.971	.576	1.000	19.000	0.457
	Hotelling's Trace	0.030	.576	1.000	19.000	0.457
	Roy's Largest Root	0.030	.576	1.000	19.000	0.457

Table 2M: Average RRI ANOVA: Multivariate Tests

Mauchly's Test of Sphericity							
Within Subjects Effect	**Mauchly's W**	**Approx. Chi-Square**	**df**	**Sig.**	**Greenhouse-Geisser**	**Huynh-Feldt**	**Lower-bound**
DSB	1.000	0.000	0		1.000	1.000	1.000
Sleep	1.000	0.000	0		1.000	1.000	1.000
DSB x Sleep	1.000	0.000	0		1.000	1.000	1.000

Table 3M: Average RRI ANOVA: Mauchly's Test of Sphericity

Within Subject						
Source		**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
DSB	Sphericity Assumed	1974.175	1	1974.175	0.331	0.572
	Greenhouse-Geisser	1974.175	1.000	1974.175	0.331	0.572
	Huynh-Feldt	1974.175	1.000	1974.175	0.331	0.572
	Lower-bound	1974.175	1.000	1974.175	0.331	0.572
Error (DSB)	Sphericity Assumed	113264.573	19	5961.293		
	Greenhouse-Geisser	113264.573	19.000	5961.293		
	Huynh-Feldt	113264.573	19.000	5961.293		
	Lower-bound	113264.573	19.000	5961.293		
Sleep	Sphericity Assumed	105129.060	1	105129.060	25.683	0.000
	Greenhouse-Geisser	105129.060	1.000	105129.060	25.683	0.000
	Huynh-Feldt	105129.060	1.000	105129.060	25.683	0.000
	Lower-bound	105129.060	1.000	105129.060	25.683	0.000
Error (Sleep)	Sphericity Assumed	77772.756	19	4093.303		
	Greenhouse-Geisser	77772.756	19.000	4093.303		
	Huynh-Feldt	77772.756	19.000	4093.303		

	Lower-bound	77772.756	19.000	4093.303		
DSB x Sleep	Sphericity Assumed	749.825	1	749.825	0.576	0.457
	Greenhouse-Geisser	749.825	1.000	749.825	0.576	0.457
	Huynh-Feldt	749.825	1.000	749.825	0.576	0.457
	Lower-bound	749.825	1.000	749.825	0.576	0.457
Error (DSB x Sleep)	Sphericity Assumed	24732.316	19	1301.701		
	Greenhouse-Geisser	24732.316	19.000	1301.701		
	Huynh-Feldt	24732.316	19.000	1301.701		
	Lower-bound	24732.316	19.000	1301.701		

Table 4M: Average RRI ANOVA: Within Subject

Multivariate Tests						
		Value	**F**	**Hypothesis df**	**Error df**	**Sig.**
DSB	Pillai's Trace	0.135	2.974b	1.000	19.000	0.101
	Wilks' Lambda	0.865	2.974b	1.000	19.000	0.101
	Hotelling's Trace	0.157	2.974	1.000	19.000	0.101
	Roy's Largest Root	0.157	2.974	1.000	19.000	0.101
Sleep	Pillai's Trace	0.090	1.883	1.000	19.000	0.186
	Wilks' Lambda	0.910	1.883	1.000	19.000	0.186
	Hotelling's Trace	0.099	1.883	1.000	19.000	0.186
	Roy's Largest Root	0.099	1.883	1.000	19.000	0.186
DSB x Sleep	Pillai's Trace	0.065	1.315	1.000	19.000	0.266
	Wilks' Lambda	0.935	1.315	1.000	19.000	0.266
	Hotelling's Trace	0.069	1.315	1.000	19.000	0.266
	Roy's Largest Root	0.069	1.315	1.000	19.000	0.266

Table 5M: pNN50 ANOVA: Multivariate Tests

Mauchly's Test of Sphericity							
Within Subjects Effect	**Mauchly's W**	**Approx. Chi-Square**	**df**	**Sig.**	**Greenhouse-Geisser**	**Huynh-Feldt**	**Lower-bound**
DSB	1.000	0.000	0		1.000	1.000	1.000
Sleep	1.000	0.000	0		1.000	1.000	1.000
DSB x Sleep	1.000	0.000	0		1.000	1.000	1.000

Table 6M: pNN50 ANOVA: Mauchly’s Test of Sphericity

Within Subject						
Source		**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
DSB	Sphericity Assumed	385.615	1	385.615	2.974	0.101
	Greenhouse-Geisser	385.615	1.000	385.615	2.974	0.101
	Huynh-Feldt	385.615	1.000	385.615	2.974	0.101
	Lower-bound	385.615	1.000	385.615	2.974	0.101
Error (DSB)	Sphericity Assumed	2463.939	19	129.681		
	Greenhouse-Geisser	2463.939	19.000	129.681		
	Huynh-Feldt	2463.939	19.000	129.681		
	Lower-bound	2463.939	19.000	129.681		
Sleep	Sphericity Assumed	526.392	1	526.392	1.883	0.186
	Greenhouse-Geisser	526.392	1.000	526.392	1.883	0.186
	Huynh-Feldt	526.392	1.000	526.392	1.883	0.186
	Lower-bound	526.392	1.000	526.392	1.883	0.186
Error (Sleep)	Sphericity Assumed	5312.182	19	279.589		
	Greenhouse-Geisser	5312.182	19.000	279.589		
	Huynh-Feldt	5312.182	19.000	279.589		
	Lower-bound	5312.182	19.000	279.589		
DSB x Sleep	Sphericity Assumed	69.619	1	69.619	1.315	0.266
	Greenhouse-Geisser	69.619	1.000	69.619	1.315	0.266
	Huynh-Feldt	69.619	1.000	69.619	1.315	0.266
	Lower-bound	69.619	1.000	69.619	1.315	0.266
Error (DSB x Sleep)	Sphericity Assumed	1005.714	19	52.932		
	Greenhouse-Geisser	1005.714	19.000	52.932		
	Huynh-Feldt	1005.714	19.000	52.932		
	Lower-bound	1005.714	19.000	52.932		

Table 7M: pNN50 ANOVA: Within Subjects

Multivariate Test						
		Value	**F**	**Hypothesis df**	**Error df**	**Sig.**
DSB	Pillai's Trace	0.161	3.646	1.000	19.000	0.071
	Wilks' Lambda	0.839	3.646	1.000	19.000	0.071
	Hotelling's Trace	0.192	3.646	1.000	19.000	0.071
	Roy's Largest Root	0.192	3.646	1.000	19.000	0.071
Sleep	Pillai's Trace	0.006	.106	1.000	19.000	0.749
	Wilks' Lambda	0.994	.106	1.000	19.000	0.749
	Hotelling's Trace	0.006	.106	1.000	19.000	0.749
	Roy's Largest Root	0.006	.106	1.000	19.000	0.749
DSB x Sleep	Pillai's Trace	0.035	.695	1.000	19.000	0.415
	Wilks' Lambda	0.965	.695	1.000	19.000	0.415
	Hotelling's Trace	0.037	.695	1.000	19.000	0.415
	Roy's Largest Root	0.037	.695	1.000	19.000	0.415

Table 8M: RMSSD ANOVA: Multivariate Test

Mauchly's Test of Sphericity							
Within Subjects Effect	**Mauchly's W**	**Approx. Chi-Square**	**df**	**Sig.**	**Greenhouse-Geisser**	**Huynh-Feldt**	**Lower-bound**
DSB	1.000	0.000	0		1.000	1.000	1.000
Sleep	1.000	0.000	0		1.000	1.000	1.000
DSB x Sleep	1.000	0.000	0		1.000	1.000	1.000

Table 9M: RMSSD ANOVA: Mauchly’s Test of Sphericity

Within Subjects						
Source		**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
DSB	Sphericity Assumed	1725.213	1	1725.213	3.646	0.071
	Greenhouse-Geisser	1725.213	1.000	1725.213	3.646	0.071
	Huynh-Feldt	1725.213	1.000	1725.213	3.646	0.071
	Lower-bound	1725.213	1.000	1725.213	3.646	0.071
Error (DSB)	Sphericity Assumed	8991.207	19	473.221		
	Greenhouse-Geisser	8991.207	19.000	473.221		
	Huynh-Feldt	8991.207	19.000	473.221		
	Lower-bound	8991.207	19.000	473.221		
Sleep	Sphericity Assumed	64.473	1	64.473	0.106	0.749
	Greenhouse-Geisser	64.473	1.000	64.473	0.106	0.749
	Huynh-Feldt	64.473	1.000	64.473	0.106	0.749
	Lower-bound	64.473	1.000	64.473	0.106	0.749
Error (Sleep)	Sphericity Assumed	11608.088	19	610.952		
	Greenhouse-Geisser	11608.088	19.000	610.952		
	Huynh-Feldt	11608.088	19.000	610.952		
	Lower-bound	11608.088	19.000	610.952		
DSB x Sleep	Sphericity Assumed	212.573	1	212.573	0.695	0.415
	Greenhouse-Geisser	212.573	1.000	212.573	0.695	0.415
	Huynh-Feldt	212.573	1.000	212.573	0.695	0.415
	Lower-bound	212.573	1.000	212.573	0.695	0.415
Error (DSB x Sleep)	Sphericity Assumed	5808.047	19	305.687		
	Greenhouse-Geisser	5808.047	19.000	305.687		
	Huynh-Feldt	5808.047	19.000	305.687		
	Lower-bound	5808.047	19.000	305.687		

Table 10M: RMSSD ANOVA: Within Subjects

Multivariate Test						
		Value	**F**	**Hypothesis df**	**Error df**	**Sig.**
DSB	Pillai's Trace	0.227	5.590b	1.000	19.000	0.029
	Wilks' Lambda	0.773	5.590b	1.000	19.000	0.029
	Hotelling's Trace	0.294	5.590b	1.000	19.000	0.029
	Roy's Largest Root	0.294	5.590b	1.000	19.000	0.029
Sleep	Pillai's Trace	0.000	.006b	1.000	19.000	0.941
	Wilks' Lambda	1.000	.006b	1.000	19.000	0.941
	Hotelling's Trace	0.000	.006b	1.000	19.000	0.941
	Roy's Largest Root	0.000	.006b	1.000	19.000	0.941
DSB x Sleep	Pillai's Trace	0.009	.163b	1.000	19.000	0.691
	Wilks' Lambda	0.991	.163b	1.000	19.000	0.691
	Hotelling's Trace	0.009	.163b	1.000	19.000	0.691
	Roy's Largest Root	0.009	.163b	1.000	19.000	0.691

Table 11M: HF – HRV ANOVA: Multivariate Test

Mauchly's Test of Sphericity							
Within Subjects Effect	**Mauchly's W**	**Approx. Chi-Square**	**df**	**Sig.**	**Greenhouse-Geisser**	**Huynh-Feldt**	**Lower-bound**
DSB	1.000	0.000	0		1.000	1.000	1.000
Sleep	1.000	0.000	0		1.000	1.000	1.000
DSB x Sleep	1.000	0.000	0		1.000	1.000	1.000

Table 12M: HF- HRV ANOVA: Mauchly’s Test of Sphericity

Within Subjects						
Source		**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
DSB	Sphericity Assumed	14942764.859	1	14942764.859	5.590	0.029
	Greenhouse-Geisser	14942764.859	1.000	14942764.859	5.590	0.029
	Huynh-Feldt	14942764.859	1.000	14942764.859	5.590	0.029
	Lower-bound	14942764.859	1.000	14942764.859	5.590	0.029
Error (DSB)	Sphericity Assumed	50788763.923	19	2673092.838		
	Greenhouse-Geisser	50788763.923	19.000	2673092.838		
	Huynh-Feldt	50788763.923	19.000	2673092.838		
	Lower-bound	50788763.923	19.000	2673092.838		
Sleep	Sphericity Assumed	16505.756	1	16505.756	0.006	0.941
	Greenhouse-Geisser	16505.756	1.000	16505.756	0.006	0.941
	Huynh-Feldt	16505.756	1.000	16505.756	0.006	0.941
	Lower-bound	16505.756	1.000	16505.756	0.006	0.941
Error (Sleep)	Sphericity Assumed	55953507.338	19	2944921.439		
	Greenhouse-Geisser	55953507.338	19.000	2944921.439		
	Huynh-Feldt	55953507.338	19.000	2944921.439		
	Lower-bound	55953507.338	19.000	2944921.439		
DSB x Sleep	Sphericity Assumed	323906.077	1	323906.077	0.163	0.691
	Greenhouse-Geisser	323906.077	1.000	323906.077	0.163	0.691
	Huynh-Feldt	323906.077	1.000	323906.077	0.163	0.691
	Lower-bound	323906.077	1.000	323906.077	0.163	0.691
Error (DSB x Sleep)	Sphericity Assumed	37717548.134	19	1985134.112		
	Greenhouse-Geisser	37717548.134	19.000	1985134.112		
	Huynh-Feldt	37717548.134	19.000	1985134.112		
	Lower-bound	37717548.134	19.000	1985134.112		

Table 13 M: HF-HRV ANOVA: Within Subjects

APPENDIX N

BLOOD PRESSURE STATISTICS

Group Statistics					
	Condition	**Mean**	**N**	**SD**	**SEM**
Pre-Nap SAP	AS	115.48	25	12.78	2.56
	Control	115.32	25	11.17	2.23
Pre-Nap DAP	AS	68.92	25	9.09	1.82
	Control	68.36	25	7.96	1.59
Pre-Nap MAP	AS	84.44	25	9.42	1.88
	Control	84.01	25	7.38	1.48
Post-Nap SAP	AS	116.72	25	12.10	2.42
	Control	117.12	25	11.24	2.25
Post-Nap DAP	AS	75.92	25	23.24	4.65
	Control	70.76	25	8.15	1.63
Post-Nap MAP	AS	89.52	25	17.40	3.48
	Control	86.21	25	8.11	1.62

Table 1N: Blood Pressure Group Descriptive Statistics: Group Statistics

Multivariate Test						
		Value	**F**	**Hypothesis df**	**Error df**	**Sig.**
DSB	Pillai's Trace	0.001	.015	1.000	24.000	0.902
	Wilks' Lambda	0.999	.015	1.000	24.000	0.902
	Hotelling's Trace	0.001	.015	1.000	24.000	0.902
	Roy's Largest Root	0.001	.015	1.000	24.000	0.902
Sleep	Pillai's Trace	0.100	2.677	1.000	24.000	0.115
	Wilks' Lambda	0.900	2.677	1.000	24.000	0.115
	Hotelling's Trace	0.112	2.677	1.000	24.000	0.115
	Roy's Largest Root	0.112	2.677	1.000	24.000	0.115
DSB x Sleep	Pillai's Trace	0.003	.066	1.000	24.000	0.800
	Wilks' Lambda	0.997	.066	1.000	24.000	0.800
	Hotelling's Trace	0.003	.066	1.000	24.000	0.800
	Roy's Largest Root	0.003	.066	1.000	24.000	0.800

Table 2N: SAP ANOVA: Multivariate Test

Mauchly's Test of Sphericity							
Within Subjects Effect	**Mauchly's W**	**Approx. Chi-Square**	**df**	**Sig.**	**Greenhouse-Geisser**	**Huynh-Feldt**	**Lower-bound**
DSB	1.000	0.000	0		1.000	1.000	1.000
Sleep	1.000	0.000	0		1.000	1.000	1.000
DSB x Sleep	1.000	0.000	0		1.000	1.000	1.000

Tale 3N: SAP ANOVA: Mauchly’s Test of Sphericity

Within Subjects						
Source		**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
DSB	Sphericity Assumed	0.360	1	0.360	0.015	0.902
	Greenhouse-Geisser	0.360	1.000	0.360	0.015	0.902
	Huynh-Feldt	0.360	1.000	0.360	0.015	0.902
	Lower-bound	0.360	1.000	0.360	0.015	0.902
Error (DSB)	Sphericity Assumed	562.140	24	23.423		
	Greenhouse-Geisser	562.140	24.000	23.423		
	Huynh-Feldt	562.140	24.000	23.423		
	Lower-bound	562.140	24.000	23.423		
Sleep	Sphericity Assumed	57.760	1	57.760	2.677	0.115
	Greenhouse-Geisser	57.760	1.000	57.760	2.677	0.115
	Huynh-Feldt	57.760	1.000	57.760	2.677	0.115
	Lower-bound	57.760	1.000	57.760	2.677	0.115
Error (Sleep)	Sphericity Assumed	517.740	24	21.573		
	Greenhouse-Geisser	517.740	24.000	21.573		
	Huynh-Feldt	517.740	24.000	21.573		
	Lower-bound	517.740	24.000	21.573		
DSB x Sleep	Sphericity Assumed	1.960	1	1.960	0.066	0.800
	Greenhouse-Geisser	1.960	1.000	1.960	0.066	0.800
	Huynh-Feldt	1.960	1.000	1.960	0.066	0.800
	Lower-bound	1.960	1.000	1.960	0.066	0.800
Error (DSB x Sleep)	Sphericity Assumed	716.540	24	29.856		
	Greenhouse-Geisser	716.540	24.000	29.856		
	Huynh-Feldt	716.540	24.000	29.856		
	Lower-bound	716.540	24.000	29.856		

Table 4N: SAP ANOVA: Within Subjects

Multivariate Test						
		Value	**F**	**Hypothesis df**	**Error df**	**Sig.**
DSB	Pillai's Trace	0.022	.546	1.000	24.000	0.467
	Wilks' Lambda	0.978	.546	1.000	24.000	0.467
	Hotelling's Trace	0.023	.546	1.000	24.000	0.467
	Roy's Largest Root	0.023	.546	1.000	24.000	0.467
Sleep	Pillai's Trace	0.376	14.483	1.000	24.000	0.001
	Wilks' Lambda	0.624	14.483	1.000	24.000	0.001
	Hotelling's Trace	0.603	14.483	1.000	24.000	0.001
	Roy's Largest Root	0.603	14.483	1.000	24.000	0.001
DSB x Sleep	Pillai's Trace	0.009	.206	1.000	24.000	0.654
	Wilks' Lambda	0.991	.206	1.000	24.000	0.654
	Hotelling's Trace	0.009	.206	1.000	24.000	0.654
	Roy's Largest Root	0.009	.206	1.000	24.000	0.654

Table 5N: DAP ANOVA: Multivariate Test

Mauchly's Test of Sphericity							
Within Subjects Effect	**Mauchly's W**	**Approx. Chi-Square**	**df**	**Sig.**	**Greenhouse-Geisser**	**Huynh-Feldt**	**Lower-bound**
DSB	1.000	0.000	0		1.000	1.000	1.000
Sleep	1.000	0.000	0		1.000	1.000	1.000
DSB x Sleep	1.000	0.000	0		1.000	1.000	1.000

Table 6N: DAP ANOVA: Mauchly’s Test of Sphericity

Within Subjects						
Source		**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
DSB	Sphericity Assumed	18.490	1	18.490	0.546	0.467
	Greenhouse-Geisser	18.490	1.000	18.490	0.546	0.467
	Huynh-Feldt	18.490	1.000	18.490	0.546	0.467
	Lower-bound	18.490	1.000	18.490	0.546	0.467
Error (DSB)	Sphericity Assumed	812.760	24	33.865		
	Greenhouse-Geisser	812.760	24.000	33.865		
	Huynh-Feldt	812.760	24.000	33.865		
	Lower-bound	812.760	24.000	33.865		
Sleep	Sphericity Assumed	182.250	1	182.250	14.483	0.001
	Greenhouse-Geisser	182.250	1.000	182.250	14.483	0.001
	Huynh-Feldt	182.250	1.000	182.250	14.483	0.001
	Lower-bound	182.250	1.000	182.250	14.483	0.001
Error (Sleep)	Sphericity Assumed	302.000	24	12.583		
	Greenhouse-Geisser	302.000	24.000	12.583		
	Huynh-Feldt	302.000	24.000	12.583		
	Lower-bound	302.000	24.000	12.583		
DSB x Sleep	Sphericity Assumed	2.250	1	2.250	0.206	0.654
	Greenhouse-Geisser	2.250	1.000	2.250	0.206	0.654
	Huynh-Feldt	2.250	1.000	2.250	0.206	0.654
	Lower-bound	2.250	1.000	2.250	0.206	0.654
Error (DSB x Sleep)	Sphericity Assumed	262.000	24	10.917		
	Greenhouse-Geisser	262.000	24.000	10.917		
	Huynh-Feldt	262.000	24.000	10.917		
	Lower-bound	262.000	24.000	10.917		

Table 7N: DAP ANOVA: Within Subjects

Multivariate Test						
		Value	**F**	**Hypothesis df**	**Error df**	**Sig.**
DSB	Pillai's Trace	0.012	.299	1.000	24.000	0.589
	Wilks' Lambda	0.988	.299	1.000	24.000	0.589
	Hotelling's Trace	0.012	.299	1.000	24.000	0.589
	Roy's Largest Root	0.012	.299	1.000	24.000	0.589
Sleep	Pillai's Trace	0.311	10.857	1.000	24.000	0.003
	Wilks' Lambda	0.689	10.857	1.000	24.000	0.003
	Hotelling's Trace	0.452	10.857	1.000	24.000	0.003
	Roy's Largest Root	0.452	10.857	1.000	24.000	0.003
DSB x Sleep	Pillai's Trace	0.001	.031	1.000	24.000	0.862
	Wilks' Lambda	0.999	.031	1.000	24.000	0.862
	Hotelling's Trace	0.001	.031	1.000	24.000	0.862
	Roy's Largest Root	0.001	.031	1.000	24.000	0.862

Table 8N: MAP ANOVA: Multivariate Test

Mauchly's Test of Sphericity							
Within Subjects Effect	**Mauchly's W**	**Approx. Chi-Square**	**df**	**Sig.**	**Greenhouse-Geisser**	**Huynh-Feldt**	**Lower-bound**
DSB	1.000	0.000	0		1.000	1.000	1.000
Sleep	1.000	0.000	0		1.000	1.000	1.000
DSB x Sleep	1.000	0.000	0		1.000	1.000	1.000

Table 9N: MAP ANOVA: Mauchly’s Test of Sphericity

Within Subjects						
Source		**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
DSB	Sphericity Assumed	7.290	1	7.290	0.299	0.589
	Greenhouse-Geisser	7.290	1.000	7.290	0.299	0.589
	Huynh-Feldt	7.290	1.000	7.290	0.299	0.589
	Lower-bound	7.290	1.000	7.290	0.299	0.589
Error (DSB)	Sphericity Assumed	584.904	24	24.371		
	Greenhouse-Geisser	584.904	24.000	24.371		
	Huynh-Feldt	584.904	24.000	24.371		
	Lower-bound	584.904	24.000	24.371		
Sleep	Sphericity Assumed	133.788	1	133.788	10.857	0.003
	Greenhouse-Geisser	133.788	1.000	133.788	10.857	0.003
	Huynh-Feldt	133.788	1.000	133.788	10.857	0.003
	Lower-bound	133.788	1.000	133.788	10.857	0.003
Error (Sleep)	Sphericity Assumed	295.740	24	12.322		
	Greenhouse-Geisser	295.740	24.000	12.322		
	Huynh-Feldt	295.740	24.000	12.322		
	Lower-bound	295.740	24.000	12.322		
DSB x Sleep	Sphericity Assumed	0.321	1	0.321	0.031	0.862
	Greenhouse-Geisser	0.321	1.000	0.321	0.031	0.862
	Huynh-Feldt	0.321	1.000	0.321	0.031	0.862
	Lower-bound	0.321	1.000	0.321	0.031	0.862
Error (DSB x Sleep)	Sphericity Assumed	250.984	24	10.458		
	Greenhouse-Geisser	250.984	24.000	10.458		
	Huynh-Feldt	250.984	24.000	10.458		
	Lower-bound	250.984	24.000	10.458		

Table 10N: MAP ANOVA: Within Subjects

APPENDIX O

SUBJECTIVE QUESTIONNAIRES STATISTICS

Group Statistics					
	Condition	**Mean**	**N**	**SD**	**SEM**
Pre-Nap STAI	AS	28.4000	25	6.98212	1.39642
	Control	27.88	25	6.86367	1.37273
Post-Nap STAI	AS	25.84	25	5.56537	1.11307
	Control	25.64	25	4.94031	0.98806

Table 1O: STAI ANOVA: Group Statistics

Multivariate Test						
		Value	**F**	**Hypothesis df**	**Error df**	**Sig.**
DSB	Pillai's Trace	0.027	.678	1.000	24.000	0.419
	Wilks' Lambda	0.973	.678	1.000	24.000	0.419
	Hotelling's Trace	0.028	.678	1.000	24.000	0.419
	Roy's Largest Root	0.028	.678	1.000	24.000	0.419
Sleep	Pillai's Trace	0.255	8.229	1.000	24.000	0.008
	Wilks' Lambda	0.745	8.229	1.000	24.000	0.008
	Hotelling's Trace	0.343	8.229	1.000	24.000	0.008
	Roy's Largest Root	0.343	8.229	1.000	24.000	0.008
DSB x Sleep	Pillai's Trace	0.003	.079	1.000	24.000	0.782
	Wilks' Lambda	0.997	.079	1.000	24.000	0.782
	Hotelling's Trace	0.003	.079	1.000	24.000	0.782
	Roy's Largest Root	0.003	.079	1.000	24.000	0.782

Table 2O: STAI ANOVA: Multivariate Test

Mauchly's Test of Sphericity							
Within Subjects Effect	**Mauchly's W**	**Approx. Chi-Square**	**df**	**Sig.**	**Greenhouse-Geisser**	**Huynh-Feldt**	**Lower-bound**
DSB	1.000	0.000	0		1.000	1.000	1.000
Sleep	1.000	0.000	0		1.000	1.000	1.000
DSB x Sleep	1.000	0.000	0		1.000	1.000	1.000

Table 3O: STAI ANOVA: Mauchly’s Test of Sphericity

Within Subjects						
Source		**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
DSB	Sphericity Assumed	3.240	1	3.240	0.678	0.419
	Greenhouse-Geisser	3.240	1.000	3.240	0.678	0.419
	Huynh-Feldt	3.240	1.000	3.240	0.678	0.419
	Lower-bound	3.240	1.000	3.240	0.678	0.419
Error (DSB)	Sphericity Assumed	114.760	24	4.782		
	Greenhouse-Geisser	114.760	24.000	4.782		
	Huynh-Feldt	114.760	24.000	4.782		
	Lower-bound	114.760	24.000	4.782		
Sleep	Sphericity Assumed	144.000	1	144.000	8.229	0.008
	Greenhouse-Geisser	144.000	1.000	144.000	8.229	0.008
	Huynh-Feldt	144.000	1.000	144.000	8.229	0.008
	Lower-bound	144.000	1.000	144.000	8.229	0.008
Error (Sleep)	Sphericity Assumed	420.000	24	17.500		
	Greenhouse-Geisser	420.000	24.000	17.500		
	Huynh-Feldt	420.000	24.000	17.500		
	Lower-bound	420.000	24.000	17.500		
DSB x Sleep	Sphericity Assumed	0.640	1	0.640	0.079	0.782
	Greenhouse-Geisser	0.640	1.000	0.640	0.079	0.782
	Huynh-Feldt	0.640	1.000	0.640	0.079	0.782
	Lower-bound	0.640	1.000	0.640	0.079	0.782
Error (DSB x Sleep)	Sphericity Assumed	195.360	24	8.140		
	Greenhouse-Geisser	195.360	24.000	8.140		
	Huynh-Feldt	195.360	24.000	8.140		
	Lower-bound	195.360	24.000	8.140		

Table 4O: STAI ANOVA: Within Subjects

Group Statistics					
	Condition	**Mean**	**N**	**SD**	**SEM**
Pre-Nap ESS	AS	6.9600	25	4.11785	0.82357
	Control	7.36	25	4.34818	0.86964
Post-Nap ESS	AS	5.88	25	4.23596	0.84719
	Control	6.04	25	3.83493	0.76699

Table 5O: ESS ANOVA: Group Statistics

Multivariate Test						
		Value	**F**	**Hypothesis df**	**Error df**	**Sig.**
DSB	Pillai's Trace	0.017	.416b	1.000	24.000	0.525
	Wilks' Lambda	0.983	.416b	1.000	24.000	0.525
	Hotelling's Trace	0.017	.416b	1.000	24.000	0.525
	Roy's Largest Root	0.017	.416b	1.000	24.000	0.525
Sleep	Pillai's Trace	0.206	6.216b	1.000	24.000	0.020
	Wilks' Lambda	0.794	6.216b	1.000	24.000	0.020
	Hotelling's Trace	0.259	6.216b	1.000	24.000	0.020
	Roy's Largest Root	0.259	6.216b	1.000	24.000	0.020
DSB x Sleep	Pillai's Trace	0.012	.291b	1.000	24.000	0.594
	Wilks' Lambda	0.988	.291b	1.000	24.000	0.594
	Hotelling's Trace	0.012	.291b	1.000	24.000	0.594
	Roy's Largest Root	0.012	.291b	1.000	24.000	0.594

Table 6O: ESS ANOVA: Multivariate Test

Mauchly's Test of Sphericity							
Within Subjects Effect	**Mauchly's W**	**Approx. Chi-Square**	**df**	**Sig.**	**Greenhouse-Geisser**	**Huynh-Feldt**	**Lower-bound**
DSB	1.000	0.000	0		1.000	1.000	1.000
Sleep	1.000	0.000	0		1.000	1.000	1.000
DSB x Sleep	1.000	0.000	0		1.000	1.000	1.000

Table 7O: ESS ANOVA: Mauchly’s Test of Sphericity

Within Subjects						
Source		**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
DSB	Sphericity Assumed	1.960	1	1.960	0.416	0.525
	Greenhouse-Geisser	1.960	1.000	1.960	0.416	0.525
	Huynh-Feldt	1.960	1.000	1.960	0.416	0.525
	Lower-bound	1.960	1.000	1.960	0.416	0.525
Error (DSB)	Sphericity Assumed	113.040	24	4.710		
	Greenhouse-Geisser	113.040	24.000	4.710		
	Huynh-Feldt	113.040	24.000	4.710		
	Lower-bound	113.040	24.000	4.710		
Sleep	Sphericity Assumed	36.000	1	36.000	6.216	0.020
	Greenhouse-Geisser	36.000	1.000	36.000	6.216	0.020
	Huynh-Feldt	36.000	1.000	36.000	6.216	0.020
	Lower-bound	36.000	1.000	36.000	6.216	0.020
Error (Sleep)	Sphericity Assumed	139.000	24	5.792		
	Greenhouse-Geisser	139.000	24.000	5.792		
	Huynh-Feldt	139.000	24.000	5.792		
	Lower-bound	139.000	24.000	5.792		
DSB x Sleep	Sphericity Assumed	0.360	1	0.360	0.291	0.594
	Greenhouse-Geisser	0.360	1.000	0.360	0.291	0.594
	Huynh-Feldt	0.360	1.000	0.360	0.291	0.594
	Lower-bound	0.360	1.000	0.360	0.291	0.594
Error (DSB x Sleep)	Sphericity Assumed	29.640	24	1.235		
	Greenhouse-Geisser	29.640	24.000	1.235		
	Huynh-Feldt	29.640	24.000	1.235		
	Lower-bound	29.640	24.000	1.235		

Table 8O: ESS ANOVA: Within Subjects

APPENDIX P

DELTA POWER STATISTICS

Group Statistics					
	Condition	**Mean**	**N**	**SD**	**SEM**
Delta Power – C leads	AS	369.3600	14	150.00416	40.09030
	Control	348.1544	14	244.84221	65.43683
Delta Power – F leads	AS	795.1050	14	712.55799	190.43913
	Control	646.2951	14	495.17857	132.34204

Table 1P: Paired Samples T-Test: Delta Power: Group Statistics

Paired Samples T-Test						
	Lower 95%	**Upper 95%**	**t**	**df**	**One sided P-Value**	**Two-sided P-value**
Delta Power – C leads	-107.37506	149.78627	0.356	13	0.364	0.727
Delta Power – F leads	-239.05159	536.67145	0.829	13	0.211	0.422

Table 2P: Paired Samples T-Test: Delta Power

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