

FAMILIARIZATION WITH AMBULATORY SLEEP AND BLOOD PRESSURE
MONITORING

A Thesis
by
KASEY JORDAN KLEIBER

Submitted to the Graduate School
at Appalachian State University
in partial fulfillment of the requirements for the degree of
MASTER OF SCIENCE

May 2019
Department of Health and Exercise Science

FAMILIARIZATION WITH AMBULATORY SLEEP AND BLOOD PRESSURE
MONITORING

A Thesis
by
KASEY JORDAN KLEIBER
May 2019

APPROVED BY:

Scott R. Collier, Ph.D.
Chairperson, Thesis Committee

Caroline J. Smith, Ph.D.
Member, Thesis Committee

Adam Hege, Ph.D.
Member, Thesis Committee

Kelly J. Cole, Ph.D.
Chairperson, Department of Health and Exercise Science

Mike McKenzie, Ph.D.
Dean, Cratis D. Williams School of Graduate Studies

Copyright by Kasey Jordan Kleiber 2019
All Rights Reserved

Abstract

FAMILIARIZATION WITH AMBULATORY SLEEP AND BLOOD PRESSURE MONITORING

Kasey Jordan Kleiber, B.S., Appalachian State University
M.S., Appalachian State University

Chairperson: Scott R. Collier

Objectives: Sleep is a life-sustaining action that has implications in aspects of physical, mental, and emotional well-being. One necessary event that occurs during sleep is nocturnal blood pressure dipping. Measurement of ambulatory sleep and blood pressure are gaining popularity as these can be completed in an individual's home. However, little is known regarding the reliability of data and the time it takes oneself to familiarize with the equipment. Therefore, the purpose of this study was to determine how many nights of wearing the monitoring equipment were required to restore sleep architecture and blood pressure data to baseline. **Methods:** Eight male and female subjects completed all 3 nights of both sleep and blood pressure readings. Visit 1 consisted of anthropometric and resting blood pressure measurements. The subjects were also familiarized with the equipment and instructed to wear the Sleep Profiler™ and SunTech Medical Oscar2 ambulatory blood pressure cuff simultaneously for 3 consecutive nights. Visit 2 consisted of the subjects returning the equipment and the data being downloaded to a laboratory computer. **Results:** The percent of time spent in N1, N2, N3, and REM were not statistically different between nights 1, 2, and 3. Time for wake after sleep onset was not statistically different between nights 1, 2, and 3. Time for sleep latency was statistically greater from night 2 to night 3

($p = 0.042$). Percent nocturnal systolic and diastolic blood pressure dips were not statistically different between nights 1, 2, and 3. Cortical and autonomic arousals were not statistically different between nights 1, 2, and 3. **Conclusions:** Ambulatory sleep monitoring takes 3 nights before the data is reliable and the person is familiarized with the mode of measurement.

Acknowledgments

This research was completed with an Office of Student Research Grant through Appalachian State University.

I would like to thank my mentor, Dr. Scott Collier, for your guidance these last two years. You have extended your mentorship beyond education and have taught me valuable life lessons. Thank you for always believing in me and never failing to push me out of my comfort zone just to show me how successful I can be.

I would also like to thank my thesis committee, Dr. Caroline Smith and Dr. Adam Hege, for your support. Your guidance and help throughout this entire process has been greatly appreciated.

I would also like to thank my parents. Thank you for being there through the laughs, tears, tough times and happy times. I also want to thank you for supporting me in all my crazy career changes. Your continual love and support have never gone unnoticed.

Lastly, I would like to thank my friends. The memories I have made here with you all will be cherished forever.

Table of Contents

| | |
|-----------------------|------|
| Abstract | iv |
| Acknowledgments | vi |
| Foreword | viii |
| Introduction | 1 |
| References | 12 |
| Vita | 18 |

Foreword

This thesis will be submitted to *Sleep Health: Journal of the National Sleep Foundation*, an international peer-review journal owned by the National Sleep Foundation and published by Elsevier; it has been formatted according to the instructions to authors for that journal.

Introduction

Sleep is an imperative, mandatory process that has been shown by both human and rodent studies to have implications in psychological and physiological health.^{1,2} Sleep can be divided into two main phases that are accompanied by physiological changes: non-rapid eye movement (NREM) and rapid eye movement (REM).³ Non-rapid eye movement is further composed of three stages: N1, N2, and N3.³ Marked by a state of drowsiness, stage N1 represents the transition from wake to sleep and is considered light sleep.³ Following a decrease in body temperature, stage N2 (sleep onset) is entered.³ This stage exerts influence on respiration and heart rate (beats per minute).³ Stage N3, also known as slow wave sleep (SWS) or deep sleep, is the most restorative of all stages.^{3,4} During this stage, growth and repair occur, hormones are secreted, and nocturnal blood pressure (BP) dipping occurs due to enhanced relaxation during this stage, amongst other physiological processes.³ The amount of time spent in SWS is important to allow for recovery from the previous day's events as well as allowing for central nervous system adjustments to obtain the drop in nocturnal BP.⁵ Following all NREM stages is REM sleep, which is characterized by darting eye movements, increased brain activity and dreaming, memory storage and paralysis.^{3,5} Furthermore, BP and heart rate rise as sleep transitions from NREM to REM.⁵ Typically, NREM and REM cycle amongst one another approximately every 90 minutes throughout the night.^{5,6} This pattern persists for a total of 3 to 6 cycles each night; and as the sleep period progresses, more time is spent within REM and less time is spent within SWS.⁵

The recommended amount of sleep varies across a lifetime but nonetheless is just as important as the quality of sleep obtained each night. Short (less than 6 hours) and prolonged (greater than 9 hours) sleep durations have both been associated with increased risk of

adverse health outcomes including, but not limited to, impaired immune function, stroke, diabetes, obesity, and hypertension.^{7, 8, 9, 10, 11} Further, research has shown that insufficient sleep has been linked to a variety of mood and emotional disorders, such as stress, depression, and anger.^{12,13,14} In accordance with the aforementioned research, the National Sleep Foundation recommends 7-9 hours of sleep for adults.⁶ Sleep durations outside of this range can have detrimental effects on an individual's health.

As previously noted, one important physiological change that occurs during sleep is the reduction of BP. This decline in pressures is known as nocturnal BP dipping and is classified by a 10-20% decrease.¹⁵ The mechanism behind nocturnal BP dipping involves baroreflex sensitivity and resetting to a lower set point in conjunction with reductions in sympathetic nervous system activity.^{16, 17, 18} The absence of nocturnal dipping, or less than a 10% reduction in pressure, is coined "non-dipping" and is linked to a multitude of physiological ailments including increased risk of mortality, cardiovascular disease, and stroke.^{19, 20, 21} Sleep can affect BP measurements, with frequent arousals, fragmented sleep, and more time spent in stage N1 resulting in blunted BP dipping.^{18, 22, 23, 24} Sleep continuity in conjunction with a greater amount of time spent in deep, restorative sleep is needed to mitigate non-dipping status.

Currently, there are both subjective and objective ways of measuring sleep. Subjectively, sleep questionnaires are employed as user-friendly, quick screening tools that aid in identifying habits and behaviors that could translate into sleep disorders.²⁵ However, research has shown that individuals tend to unintentionally fabricate sleeping habits.²⁶ Polysomnography is an objective measurement that is considered the cornerstone for sleep architecture assessment.²⁷ Polysomnography utilizes EEG signals, electrooculogram (EOG)

signals, electromyography (EMG) signals, and electrocardiogram (ECG) signals, amongst other parameters such as pulse oximetry and respiration.²⁸ Due to the invasive and costly nature of the test, PSG is typically performed only over one to two nights in a sleep lab.²⁹ A less invasive and more economically feasible option to PSG is actigraphy, which can be employed in ambulatory settings. Actigraphs are worn on the wrist and rely on movement to record sleep parameters.^{27, 30} While less invasive, relying solely on movement allows for the potential to misclassify measured data, especially in the absence of brain wave activity.²⁷ The creation of new technology, like the Sleep Profiler™, has allowed for more accurate data collection. The Sleep Profiler™ gathers electroencephalogram (EEG) data to determine sleep architecture.³¹ When compared to laboratory PSG, the Sleep Profiler™ had strong agreement for all sleep architecture variables and wakefulness, indicating congruency between these two methods of data collection.³¹ Portable sleep monitors allow for data collection in a similar fashion as PSG but provide ecological and economic benefits as these can be used in the home setting.

The concurrent measurement of BP and sleep raises the concern of collecting accurate data, as some studies have shown that BP monitoring does affect sleep architecture.²⁴ Despite this concern, early research by Dimsdale and colleagues³² concluded that BP cuff inflation showed an increase in arousals and disturbed sleep. However, two consecutive nights of data collection showed a familiarization with ambulatory blood pressure monitoring, as demonstrated by significant decreases in arousals in the subjects.³² Therefore, the purpose of this study is to determine how many nights of wearing the Sleep Profiler™ paired with an ambulatory BP monitor are required to restore BP and sleep architecture data to baseline in normotensive, college-aged students. Previous pilot data in our lab has shown no sleep

architecture changes from night 3 to night 7 when measured for 7 consecutive days.

Therefore, we hypothesize that the third night of continual ambulatory sleep and BP measurement would be representative sleep architecture for each subject.

Methods

Subjects

Eighteen normotensive (< 130 mmHg systolic blood pressure (SBP) and < 80 mmHg diastolic blood pressure (DBP)) male and female subjects between the ages of 18 and 25 years old were recruited at Appalachian State University for this study. Subjects were excluded from the study if their resting BP in visit 1 was above 130/80 mmHg or if they could not wear the equipment all 3 nights.

Procedure

Eight subjects completed both sleep architecture and BP measurements on all 3 nights of data collection. An additional 2 subjects completed all but one variable of data and were included in the sample. The first visit consisted of anthropometric and resting BP measurements. Prior to visit 1, subjects were advised to refrain from eating and drinking caffeinated beverages at least 2 hours prior. After providing their written informed consent, height and weight were measured without shoes and the subject was instructed to rest seated for 5 minutes. After 5 minutes, BP was measured, the subject was given a 1-minute rest and a subsequent measure was taken. If the measurements were not congruent, a third BP measurement was ascertained after a 1-minute rest and averaged. Blood pressure measurements were completed using an automated BP detection system (GE DinaMap, Pro 400v2, USA). Following BP measurements, the subject was familiarized with the Sleep Profiler™ (Advanced Brain Monitoring, Inc., Carlsbad, CA, USA) and the SunTech Medical

Oscar2 (SunTech Medical, Inc., Morrisville, NC, USA) ambulatory BP device prior to departing the laboratory. Blood pressure measurements were recorded every 40 minutes during sleep. Subjects were instructed to wear both devices simultaneously for 3 consecutive nights and were also advised to avoid alcohol and caffeine late in the afternoon. Data were stored within both devices and downloaded to a laboratory computer following night 3 of data collection.

Treatment of the data

All data were analyzed for outliers via visual inspection, and descriptive statistics were determined for each category (SPSS, v.24, Chicago, IL, USA). Sleep and BP data were analyzed using a repeated measures ANOVA (group x time) to determine any differences in the outcome variables over successive nights. If significance was observed, an LSD correction factor was used to determine where any differences were detected between nights. Significance was set at $p \leq 0.05$, and all data are reported as mean \pm standard error (SE).

Results

Eight subjects completed all 3 nights of data collection. Eight subjects were excluded for the aforementioned reasons. See Table 1 for subject characteristics.

Table 1

Descriptive Characteristics of Subjects

| Age (years) | Height (cm) | Weight (kg) | Resting SBP (mmHg) | Resting DPB (mmHg) |
|----------------|-----------------|----------------|--------------------|--------------------|
| 21.6 \pm 0.5 | 163.9 \pm 2.1 | 71.2 \pm 5.8 | 119 \pm 0.6 | 70 \pm 0.5 |

All data are reported as mean \pm SE.

Percent of time spent in N1, N2, and N3 sleep was not significantly different in any of the 3 measured nights (Table 2).

Table 2

Percent of time spent in N1, N2, and N3 for nights 1, 2, and 3

| Night | N1 (%) | N2 (%) | N3 (%) |
|-------|-----------|------------|------------|
| 1 | 7.6 ± 1.3 | 51.2 ± 2.8 | 24.9 ± 2.1 |
| 2 | 8.3 ± 1.0 | 49.3 ± 2.7 | 24.3 ± 3.3 |
| 3 | 7.1 ± 0.9 | 50.1 ± 2.4 | 23.2 ± 3.2 |

All data are reported as mean ± SE.

Percent of time spent in REM sleep for night 1, 2, and 3 was 16.4 ± 1.7%, 18.2 ± 1.9%, 19.6 ± 2.3%, respectively (Figure 1).

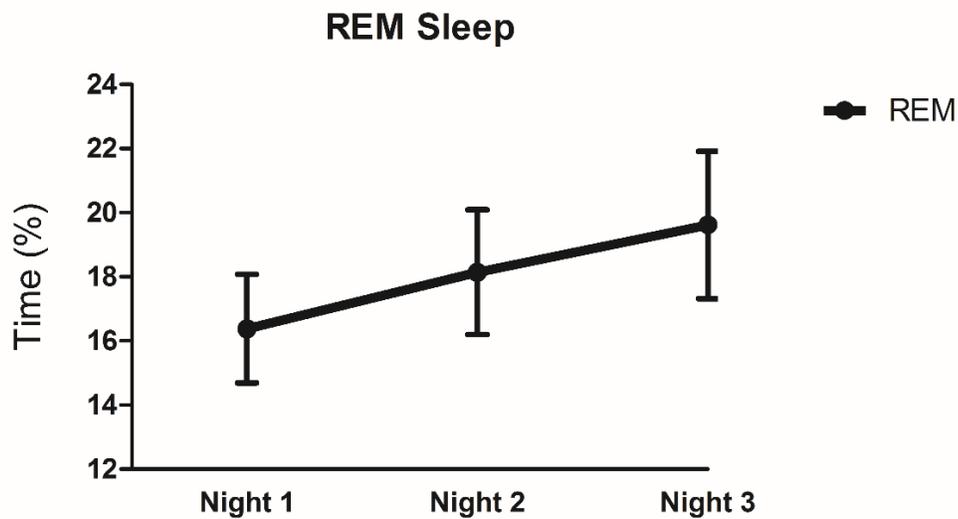


Fig. 1. Percent of time spent in REM sleep for nights 1, 2, and 3.

Percent nocturnal systolic BP dip for night 1, 2, and 3 was 16.9 ± 3.8%, 22.4 ± 2.9%, and 20.6 ± 2.4%, respectively. While not statistically different, the greatest difference in percent nocturnal systolic BP dip occurred between nights 1 and 2 (Figure 2). Percent nocturnal diastolic BP dip for night 1, 2, and 3 was 19.1 ± 3.9%, 18.2 ± 4.1%, and 15.5 ±

3.6%, respectively (Figure 2). While not statistically different, the greatest difference in percent nocturnal diastolic BP dip occurred between nights 1 and 3.

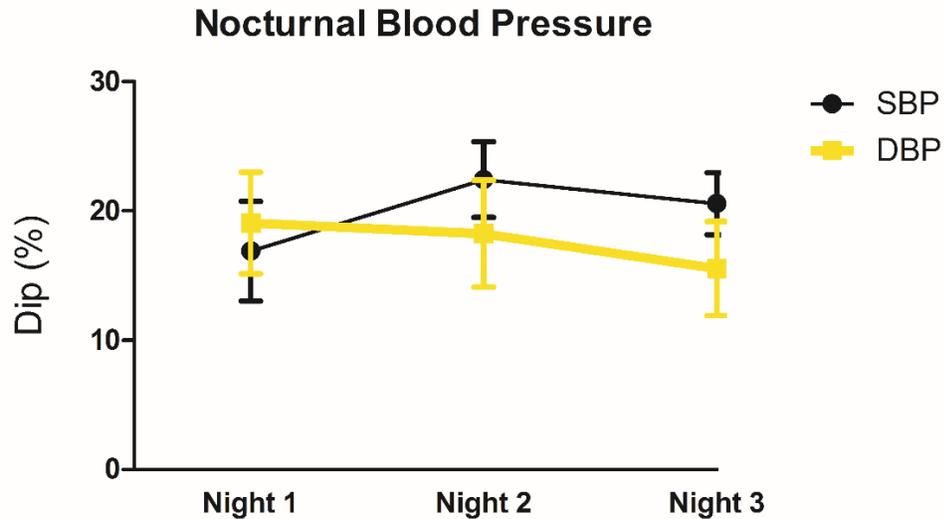


Fig. 2. Percent nocturnal systolic blood pressure dip and percent nocturnal diastolic blood pressure dip for nights 1, 2, and 3.

Wake after sleep onset (WASO) for night 1, 2, and 3 was 62.1 ± 16.6 minutes, 61.0 ± 15.4 minutes, and 67.0 ± 1.0 minutes, respectively (Figure 3). While not statistically different, the greatest difference in WASO occurred between nights 2 and 3. Sleep latency for night 1, 2, and 3 was 28.8 ± 7.6 minutes, 23.8 ± 8.4 minutes, and 36.9 ± 9.7 minutes, respectively (Figure 3). Sleep latency between nights 2 and 3 was significantly greater ($p = 0.042$).

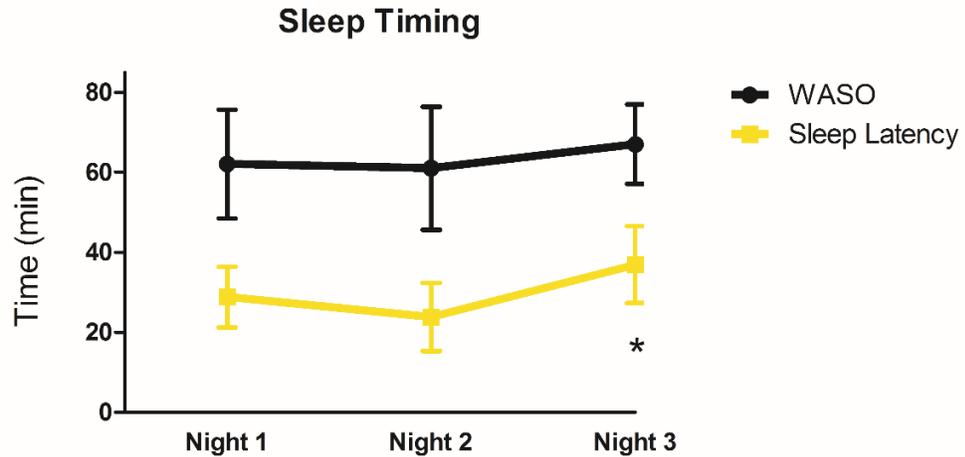


Fig. 3. Time in minutes for wake after sleep onset and sleep latency for nights 1, 2, and 3. * denotes significance ($p = 0.042$).

Autonomic arousals overall for night 1, 2, and 3 were $38.2 \pm 5.7/\text{hr}$, $36.0 \pm 4.7/\text{hr}$, and $38.2 \pm 5.7/\text{hr}$, respectively (Figure 4). Cortical arousals for night 1, 2, and 3 were $14.6 \pm 2.1/\text{hr}$, $16.7 \pm 3.9/\text{hr}$, and $15.4 \pm 3.2/\text{hr}$, respectively (Figure 4).

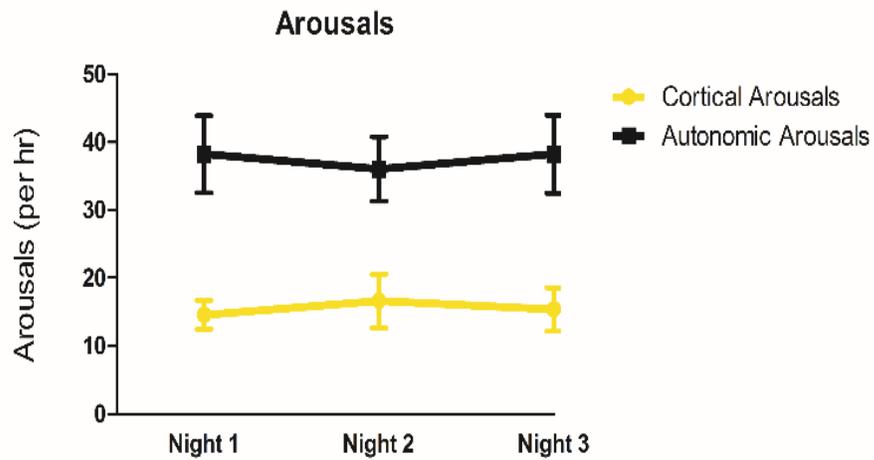


Fig. 4. Cortical and autonomic arousals per hour for nights 1, 2, and 3.

Discussion

To our knowledge, this is the first study to report sleep architecture combined with ambulatory BP monitoring within the subjects own bed. The main finding of this study was

the trend of most sleep architecture variables and percent nocturnal systolic BP dipping showing a regression towards the mean on the third night of data collection. As previously mentioned, BP follows a circadian pattern, with nighttime pressures dipping 10-20% in healthy individuals.¹⁵ In accordance with this measure, the BP dips, for both nocturnal systolic and diastolic BP pressures, fell within this range indicating that these 3 nights would be considered “normal” sleep nights.¹⁵

Slow wave sleep, or N3, is the reparative and restorative stage that is essential for proper functioning. Typically, stage N3 comprises 20-25% of a sleep episode.³³ The values for percent of time spent in stage N3 fell within this range indicating that SWS was not altered by cortical or autonomic arousals. Because SWS was not altered, nocturnal BP dipping could have occurred as it normally would, which might explain why nocturnal systolic BP dipping was not statistically different between nights. Sayk et al.³⁴ has shown that decreased time spent in SWS exhibits deleterious effects on cardiovascular regulation during nocturnal dipping. This is evidenced when individuals were shifted via sleep interventional response, from SWS to N1 or N2, and the nocturnal dip was attenuated and sleep quality was decreased.³⁴ Our data demonstrates that the nocturnal systolic BP dip was lowest on night 1, highest on night 2, and then rebounded on night 3 to a value between night 1 and night 2 ($16.9 \pm 3.8\%$, $22.4 \pm 2.9\%$, and $20.6 \pm 2.4\%$, respectively). This trend could have been attributed to excessive tiredness following sleep changes during the first night of habituation with the monitoring equipment. However, the data shows a return toward night 1 data following the third night of measurement, indicating a return towards “normal” sleep on night 3. We may be able to explain the changes in dipping between nights by the stress response from the subjects. Whereas, increased levels of cortisol, from the perceived stress of

wearing the equipment for the first time, may contribute to decreases in nocturnal dipping.³⁵ Anticipation of the unknown has also been shown to increase the stress response leading to a worsened sleep quality, as Mohammad H et al.³⁶ has recently demonstrated. Our subjects showed the greatest increase in autonomic arousals on night 1 and the lowest on night 2, congruent with our dipping responses. However, it is noted that we did not show any significant differences within nocturnal systolic dipping between nights; therefore, a minimal detrimental effect on sleep was determined from these data.

In contrast, sleep latency and WASO were both lowest on night 2 and highest on night 3. The difference in time for sleep latency was statistically different between nights 2 and 3, but time for WASO was not statistically different between nights. Stage N3 continued to decrease and was lowest on night 3; whereas, REM sleep continued to increase and was highest on night 3. Although, neither time spent in N3 nor time spent in REM were statistically different from night 1 to night 3. The increase in REM over the successive nights of measurement may be attributed to greater cortical integration.³⁷ Further, Ferreria et al.³⁸ has shown that decreases in the prior night's sleep lead to increases in melanin-concentrating hormone (MCH) which has been shown to directly increase REM sleep. Our subjects displayed increases in N1 sleep on night 2 and increases in N2 sleep on night 3. These shifts to lighter sleep stages could have been attributed to the decrements in time spent in SWS, adding to the aforementioned NREM stages over the course of the 3 nights. This has been further supported by Sayk et al.³⁴ who showed that SWS deprivation resulted in increases in NREM variables. This dataset also supports our findings of increases in WASO on day 3 in our subjects.³⁴ While it is detrimental to decrease time spent in SWS, our data showed that there were no significant differences between nights; therefore, we can assume wearing

sleeping monitoring equipment is not detrimental to sleep architecture and BP. This data revealed the third night of data collection was the most accurate depiction of the subjects' sleep.

References

1. Belenky G, Wesensten N, Thorne D, et al. Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: a sleep dose-response study. *JOURNAL OF SLEEP RESEARCH*. 12(1):1-12.
2. Allan Rechtschaffen author, Marcia A. Gilliland author, Bernard M. Bergmann author, Jacqueline B. Winter author. Physiological Correlates of Prolonged Sleep Deprivation in Rats. *Science*. 1983;(4606):182.
3. What Happens When You Sleep? <https://www.sleepfoundation.org/articles/what-happens-when-you-sleep>. Accessed March 21, 2018.
4. Dijk DJ. Regulation and functional correlates of slow wave sleep. *Journal of Clinical Sleep Medicine*. 2009;5(2 Suppl):S6-15.
5. Sleep Cycles [National Sleep Foundation Web site]. April 8, 2019. Available at: <http://sleepdisorders.sleepfoundation.org/chapter-1-normal-sleep/sleep-regulation/>
6. What Is Sleep? Latest Research & Treatments. *Am Sleep Assoc*. <https://www.sleepassociation.org/about-sleep/what-is-sleep/>. Accessed April 23, 2018.
7. Sleep and Disease Risk [Division of Sleep Medicine at Harvard Medical School Healthy Sleep Web site]. April 8, 2019. Available at: <http://healthysleep.med.harvard.edu/healthy/matters/consequences/sleep-and-disease-risk>
8. Aggarwal S, Loomba RS, Arora RR, Molnar J. Associations Between Sleep Duration and Prevalence of Cardiovascular Events. *CLINICAL CARDIOLOGY*. 36(11):671-676. doi:10.1002/clc.22160.

9. Gangwisch JE, Heymsfield SB, Boden-Albala B, et al. Sleep duration as a risk factor for diabetes incidence in a large US sample. *SLEEP*. 30(12):1667-1673.
10. Buxton OM, Marcelli E. Short and long sleep are positively associated with obesity, diabetes, hypertension, and cardiovascular disease among adults in the United States. *Social Science & Medicine*. 2010;71(5):1027-1036.
doi:10.1016/j.socscimed.2010.05.041.
11. Tochikubo O, Ikeda A, Miyajima E, Ishii M. Effects of insufficient sleep on blood pressure monitored by a new multibiomedical recorder. *Hypertension (Dallas, Tex: 1979)*. 1996;27(6):1318-1324.
12. Dinges DF, Pack F, Williams K, et al. Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4-5 hours per night. *Sleep*. 1997;20(4):267-277.
13. Riemann D, Berger M, Voderholzer U. Sleep and depression — results from psychobiological studies: an overview. *Biological Psychology*. 2001;57:67-103.
doi:10.1016/S0301-0511(01)00090-4.
14. Krizan Z, Hisler G. Sleepy anger: Restricted sleep amplifies angry feelings. *Journal of Experimental Psychology: General*. October 2018. doi:10.1037/xge0000522.
15. Yano Y, Kario K. Nocturnal blood pressure and cardiovascular disease: a review of recent advances. *HYPERTENSION RESEARCH*. 35(7):695-701.
doi:10.1038/hr.2012.26.
16. Conway J, Boon N, Jones JV, Sleight P. Involvement of the baroreceptor reflexes in the changes in blood pressure with sleep and mental arousal. *Hypertension (Dallas, Tex: 1979)*. 1983;5(5):746-748.

17. Sayk F, Becker C, Teckentrup C, et al. To dip or not to dip - On the physiology of blood pressure decrease during nocturnal sleep in healthy humans. *HYPERTENSION*. 49(5):1070-1076. doi:10.1161/HYPERTENSIONAHA.106.084343.
18. Sherwood A, Steffen P, Blumenthal J, Kuhn C, Hinderliter A. Nighttime blood pressure dipping: The role of the sympathetic nervous system. *AMERICAN JOURNAL OF HYPERTENSION*. 15(2):111-118.
19. Smolensky MH, Hermida RC, Castriotta RJ, Portaluppi F. Role of sleep-wake cycle on blood pressure circadian rhythms and hypertension. *Sleep Medicine*. 2007;8(6):668-680. doi:10.1016/j.sleep.2006.11.011.
20. Dolan E, Stanton A, Thijs L, et al. Superiority of ambulatory over clinic blood pressure measurement in predicting mortality - The Dublin Outcome Study. *HYPERTENSION*. 46(1):156-161. doi:10.1161/01.HYP.0000170138.56903.7a.
21. Fagard RH, Celis H, Thijs L, et al. Daytime and nighttime blood pressure as predictors of death and cause-specific cardiovascular events in hypertension. *Hypertension (0194911X)*. 2008;51(1):55-61.
22. Mansoor GA. Sleep actigraphy in hypertensive patients with the “non-dipper” blood pressure profile. *Journal of Human Hypertension*. 2002;16(4):237. doi:10.1038/sj.jhh.1001383.
23. Matthews KA, Kamarck TW, Hall MH, et al. Blood pressure dipping and sleep disturbance in African-American and Caucasian men and women. *AMERICAN JOURNAL OF HYPERTENSION*. 21(7):826-831. doi:10.1038/ajh.2008.183.

24. Ross AJ, Yang H, Larson RA, Carter JR. Sleep efficiency and nocturnal hemodynamic dipping in young, normotensive adults. *AMERICAN JOURNAL OF PHYSIOLOGY-REGULATORY INTEGRATIVE AND COMPARATIVE PHYSIOLOGY*. 307(7):R888-R892. doi:10.1152/ajpregu.00211.2014.
25. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research*. 1989;28(2):193-213.
26. Matthews KA, Patel SR, Pantesco EJ, et al. Similarities and differences in estimates of sleep duration by polysomnography, actigraphy, diary, and self-reported habitual sleep in a community sample. *Sleep Health*. 2018;4(1):96-103. doi:10.1016/j.sleh.2017.10.011.
27. Krystal AD, Edinger JD. Original article: Measuring sleep quality. *Sleep Medicine*. 2008;9(Supplement 1):S10-S17. doi:10.1016/S1389-9457(08)70011-X.
28. Sleep Technology: Technical Guideline. Standard Polysomnography - Updated July 2012 [American Association of Sleep Technologists Web site]. April 20, 2018. Available at: <https://www.aastweb.org/hubfs/Technical%20Guidelines/Updated%206.14.2017/StandardPSG.pdf?t=1522256354173>
29. Sleep Assessment & Evaluation: Sleep Center / Lab Tools [National Sleep Foundation Web site]. April 8, 2019. Available at: <http://sleepdisorders.sleepfoundation.org/chapter-1-normal-sleep/sleep-assessment-evaluation-sleep-center-lab-tools/>

30. Ancoli-Israel S, Cole R, Alessi C, Chambers M, Moorcroft W, Pollak CP. The Role of Actigraphy in the Study of Sleep and Circadian Rhythms. *Sleep*. 2003;26(3):342-392. doi:10.1093/sleep/26.3.342
31. Finan PH, Richards JM, Gamaldo CE, et al. Validation of a Wireless, Self-Application, Ambulatory Electroencephalographic Sleep Monitoring Device in Healthy Volunteers. *JOURNAL OF CLINICAL SLEEP MEDICINE*. 2016;12(11):1443-1451. doi:10.5664/jcsm.6262.
32. Dimsdale JE, Coy TV, Ancoli-Israel S, Clausen J, Berry CC. The effect of blood pressure cuff inflation on sleep. A polysomnographic examination. *American Journal Of Hypertension*. 1993;6(10):888-891.
33. Spriggs WH: Normal Sleep, in *Essentials of Polysomnography: A Training Guide and Reference for Sleep Technicians*. Edited by Smith M. Burlington, MA, Jones & Bartlett Learning, 2014, pp. 1-11.
34. Sayk F, Teckentrup C, Becker C, et al. Effects of selective slow-wave sleep deprivation on nocturnal blood pressure dipping and daytime blood pressure regulation. *AMERICAN JOURNAL OF PHYSIOLOGY-REGULATORY INTEGRATIVE AND COMPARATIVE PHYSIOLOGY*. 298(1):R191-R197. doi:10.1152/ajpregu.00368.2009.
35. Holt-Lunstad J, Steffen PR. Diurnal cortisol variation is associated with nocturnal blood pressure dipping. *PSYCHOSOMATIC MEDICINE*. 69(4):339-343. doi:10.1097/PSY.0b013e318050d6cc.

36. Mohammad H, Mohammad AI, Saba A. Sleeping pattern before thoracic surgery: A comparison of baseline and night before surgery. *Heliyon*. 2019;5(3):e01318. doi:10.1016/j.heliyon.2019.e01318.
37. Clawson BC, Durkin J, Suresh AK, Pickup EJ, Broussard CG, Aton SJ. Sleep Promotes, and Sleep Loss Inhibits, Selective Changes in Firing Rate, Response Properties and Functional Connectivity of Primary Visual Cortex Neurons. *Front Syst Neurosci*. 2018;12:40. Published 2018 Sep 7. doi:10.3389/fnsys.2018.00040
38. Ferreira JGP, Bittencourt JC, Adamantidis A. Melanin-concentrating hormone and sleep. *Current Opinion in Neurobiology*. 2017;44:152-158. doi:10.1016/j.conb.2017.04.008.

Vita

Kasey Jordan Kleiber was born in Charlotte, North Carolina to Michael and Amy Kleiber. She attended Lake Norman High School in Mooresville, North Carolina and graduated in May 2013. In the fall of 2013, she began attending Appalachian State University, where she majored in Exercise Science. After graduation in August 2017, she transitioned straight into a graduate assistantship in the Exercise Science department at Appalachian State University and began study toward a Master of Science degree. During her time in the master's degree, she was awarded one research grant and the Graduate Student Award for 2019 for the Exercise Science program. In May 2019, Kasey graduated with a Master of Science.