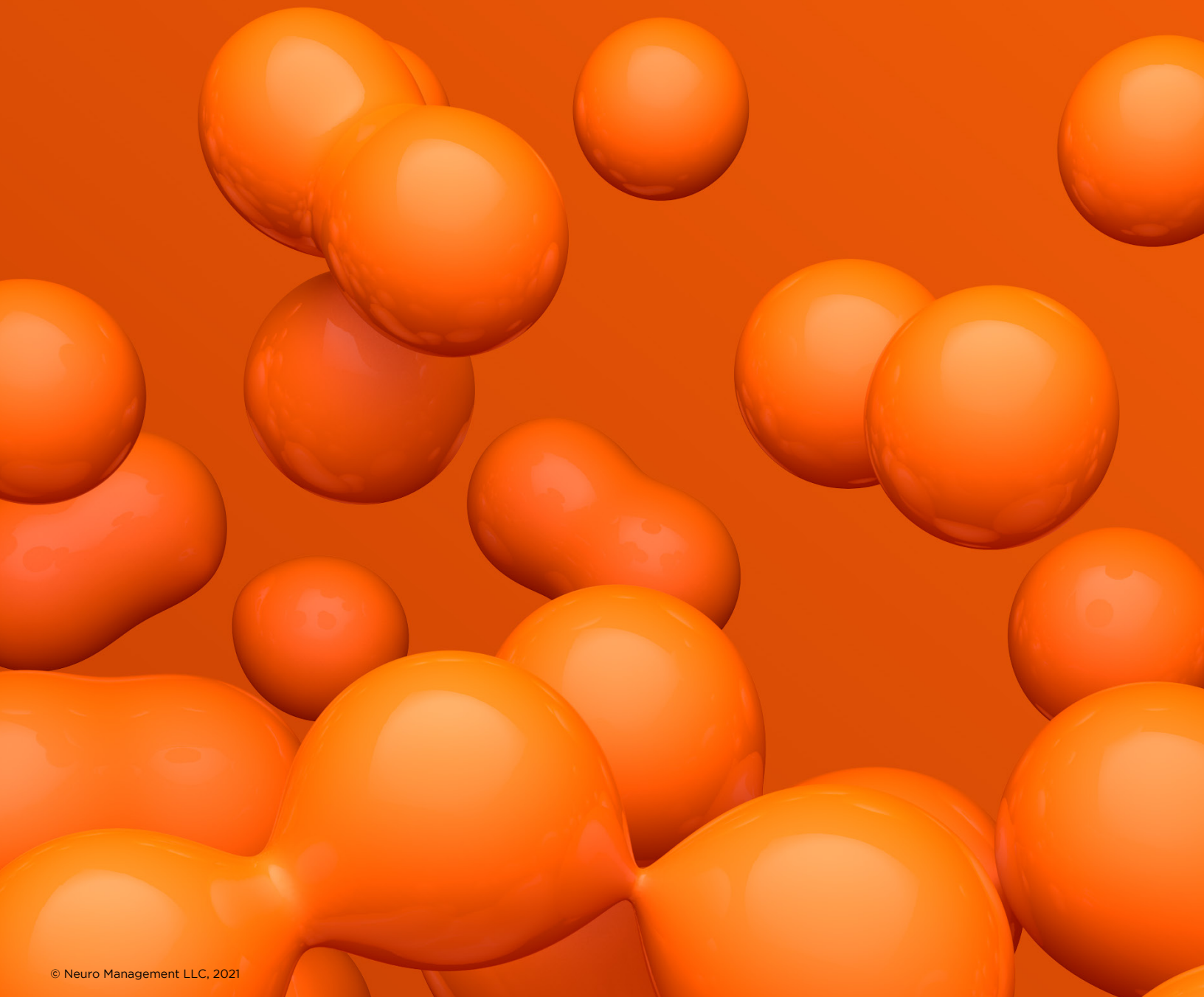
Neuro Management

Sleep Better, Live Better

Sarah Wyckoff[1], Noel Ford[1,2], Kara Waits[1], Heather Goulet[1], & Leslie Sherlin [1,2,3]

[1] Neuro Management LLC, Scottsdale, AZ, USA
[2] Department of Mind-Body Medicine, Southwest College of Naturopathic Medicine, Tempe, AZ, USA
[3] Department of Psychology, Ottawa University, Surprise, AZ, USA



Abstract

It is currently estimated that 50-70 million Americans suffer some form of sleep deprivation, costing the US economy billions of dollars due to loss of productivity, poor health, and accidents (Colten & Altevogt, 2006). The physiological underpinnings of sleep are well understood; however, formal methods of sleep monitoring for clinical diagnosis and treatment, though effective, are cumbersome, expensive, and often inaccessible for most.

Fortunately, there are more simple options for assessment and intervention demonstrated to be effective at improving sleep quality, such as heart rate monitoring, cardiorespiratory training, and neurofeedback. Recent technological advancements in mobile smart device technology (e.g., smartphones and tablets) provide even greater number of choices for assessing sleep-related parameters and for providing both passive and active interventions. These advances are already being integrated into health care settings (Luxton, McCann, Bush, Mishkind, & Reger, 2011) but the pervasiveness of smart devices support a growing interest in mobile applications for sleep improvement beyond formal health care settings. In fact, according to a recent report by Consumer Electronics Association (2015), 22% of US adults are already utilizing some form of sleep technology. Mobile sleep applications provide a more achievable avenue to address the growing public health concern of sleep deprivation.

Sleep and Quality of Life

Epidemiological studies indicate that 7-8 hours of sleep is associated with maintenance of good physical and mental health, as the body completes several important processes during sleep including hormone regulation, cardiovascular functions, and processing events from the day (Bixler, 2009). Despite this, over the last 50 years, the average reported hours of sleep per night have declined. Analysis of survey data collected by the American Cancer Society in 1960 found that the average total sleep time (TST) of adults was between 8.0 - 8.9 hours per night (Kripke, Simons, Garfinkel, & Hammond, 1979). The National Sleep Foundation 2015 poll indicates the current average adult TST is between 6.9 - 7.6 hours per night, with 34% of Americans endorsing at least one sleep disturbance, 49% reporting less than good or very good sleep quality, and 32% reporting moderate fatigue during the previous week (NSF, 2015). This reduction is predominantly noted in the employed and working-age populations and some posit this is likely due to rising stress levels among employees and changes in work habits (Bixler, 2009).

As expected with decreased TST in a working-age population, the frequency of reported insomnia symptoms and general sleep complaints such as difficulty falling asleep, waking during the night and daytime drowsiness have increased (Bixler, 2009). The aging population, 60 years and older, also experiences similar difficulties with sleep, which have been linked to prescribed medication side effects, depression, and other chronic diseases (Bixler, 2009; Cooke, 2008). Overall, the principal factors linked to poor sleep quality are lifestyle factors, socioeconomic factors, age, and stress (Bixler, 2009).

This trend of insufficient sleep is growing public health problem. It is estimated that between 50-70 million Americans chronically suffer from sleep deprivation, which negatively impacts daily functioning and overall health (review see, Colten & Altevogt, 2006). Specifically, sleep loss has been associated with a wide range of health problems including obesity (Knutson, Spiegel, Penev, & Van Cauter, 2007; Jean-Louis et al., 2014), hypertension, diabetes (Knutson et al., 2007), depression, weakening of the immune system, memory problems, heart attack, and stroke.

Beyond individual health, injuries associated with falling asleep while performing tasks such as driving are also of concern. For example, independent of alcohol related impairment, nearly 20 percent of all serious crash injuries in the general population are associated with driver sleepiness (Connor et al., 2002). On a grand scale, sleep deprivation has been identified as a contributing factor to several environmental disasters including the nuclear reactor meltdown at Three Mile Island and Chernobyl and the grounding of the Exxon Valdez oil tanker (Harrison & Horne, 2000; Colten & Altevogt, 2006).

Furthermore, the cost of this insufficient sleep trend is staggering. Each year hundreds of billions of dollars are spent each year on direct medical costs to alleviate sleep deprivation (Colten & Altevogt, 2006). Expenses from accidents related to sleep deprivation are also high. Each major incident, listed above, cost millions of dollars and had a devastating impact on the environment and local community (Colten & Altevogt, 2006; Harrison & Horne, 2000). While the total impact of sleep deprivation and poor sleep quality are too broad for the scope of the current paper, some of the staggering costs and ramifications are highlighted here for basic understanding. For a comprehensive review, see Colten and Altevogt (2006). Clearly, addressing this growing public health concern would have far-reaching implications, starting with increased safety, productivity and overall quality of life among average Americans struggling with sleep related problems.

Physiology of Sleep

The sleep-wake cycle is regulated by a complex system of neural pathways, transmitters, and receptors, as well as circadian and homeostatic processes (Fuller, Gooley, & Saper, 2006; Merica & Fortune, 2004; Tononi & Cirelli, 2006). In clinical and research settings, normal sleep processes and sleep-related disorders are investigated using polysomnography (PSG), or a sleep study. PSG involves the simultaneous monitoring of multiple physiological parameters including an 8-10 lead electroencephalography (EEG), 2 lead electrooculography (EOG), 3 lead electromyography (EMG), 2 lead electrocardiography (ECG), respiration sensor, blood oxygenation sensor, and accelerometer (American Electroencephalographic Society, 1994; Iber, Ancoli-Israel, & Quan, 2007).

While the physiological correlates of sleep have been investigated since the early part of the twentieth century, Loomis, Harvey, and Hobart (1937) were the first to publish their observations on the dynamic and cyclical patterns or “stages” of sleep, spurring the development of standardized classification systems and sleep metrics. Historically, researchers and clinicians have utilized the terminology, recording methodology, and scoring parameters defined by Rechtschaffen and Kales (1968) for the classification of PSG sleep phenomena. However, in 2007 the American Academy of Sleep Medicine (AASM) published a new manual that incorporated

arousal, cardiac, movement, and respiratory PSG scoring parameters along with revised nomenclature and recording procedures (Iber et al., 2007). The AASM scoring definitions and physiological characteristics of each stage of sleep observed in adults are presented below (Figure 1). When applicable, the AASM and corresponding Rechtschaffen and Kales nomenclature and abbreviations will be presented. Thereafter, only the AASM terminology will be referenced. This review will touch on each of the major components related to sleep, but will focus on how EEG and heart rate (HR), with an indirect focus on respiration, influence sleep quality. This is important as the larger, non-clinical population has easy access to track and train these variables to improve sleep quality.

According to the AASM Manual for the Scoring of Sleep and Associated Events (Iber et al., 2007), sleep is classified in two distinct states, non-rapid eye movement (N, NREM) and rapid eye movement (R, REM), and scored across five distinct stages: Stage W (wakefulness), Stage N1 (NREM 1, formerly Stage 1), Stage N2 (NREM 2, formerly Stage 2), Stage N3 (NREM 3, formerly Stage 3 and Stage 4), and Stage R (REM). The following generalizations can be made of sleep in a young adult maintaining a conventional sleep-wake schedule: (1) sleep stages cycle over a period of approximately 90 minutes, with varying concentrations and durations across the night, (2) wakefulness accounts for less than 5% of the night, (3) N stages constitute 75-80% of sleep, and (4) R sleep constituted 20-25% of sleep over four to six discrete episodes (Carskadon & Dement, 2011).

Stage W encompasses the transition from full alertness (eyes-open and eyes-closed) through early signs of drowsiness (eyes-closed) as the individual prepares for sleep. Dominant physiologic characteristics include waxing and waning occipital sinusoidal alpha (8-13 Hz) with eyes closure and opening (> 50% of each epoch), eye blinks and saccades associated with reading or scanning, and normal but maximal muscle tone and chin EMG (Iber et al., 2007). The crossover of wakefulness to sleep onset (Stage N1) is marked by decreased variability in respiration, decreased EMG amplitude and variability, decreased heart rate, and decrease in very low frequency (VLF) and low-frequency (LF), LF/high-frequency (HF) components of heart rate activity reflecting decreased sympathetic activity (Shinar, Akselrod, Dagan, & Baharav, 2006). Spontaneous electrodermal responses (EDRs) are uncommon during wakefulness and the transition into sleep (Broughton, Poiré, & Tassinari, 1965).

Stage N1 is marked by increased drowsiness, anteriorization and/or attenuation of occipital alpha (8-15 Hz), gradual increase (> 50% of each epoch) of low amplitude theta (4-7 Hz), possible presence of sharp waves over the central sites, sinusoidal eye movements (> 500 msec), variable but decreasing chin EMG (Iber et al., 2007), decreased ventilation, ventilatory responsiveness, an increase in airflow resistance (Trinder, Whitworth, Kay, & Wilkin, 1992).

Stage N2 is marked by the presence of K complexes (>500 msec, negative sharp followed by positive component) with maximal amplitude in the frontal sites and/or multiple trains of sleep spindles (>500 msec, 11-16 Hz, 12-14 Hz most common) over central sites in the absence of slow wave activity or an arousal event, limited eye movements, and variable but decreasing chin EMG (Iber et al., 2007).

Stage N3 is marked by the presence of slow wave activity (> 20% of each epoch, 0.5-2 Hz, p-p amplitude > 75 V) in the frontal sites, possible persistence of sleep spindles over central sites, limited or no eye movements, and variable but decreasing chin EMG (Iber et al., 2007). The HF power component of heart rate variability increases from stage N1-N3 sleep, while LF decreases during N stages (Mu-rali, Svantikova, & Somers, 2003). Heart rate, mean blood pressure, and the amplitude and frequency of sympathetic-nerve bursting (via microneurography) decrease with the descent into deeper N stage sleep, but show transient increases following arousal stimuli that produce K-complexes (Somers, Dyken, Mark, & Abboud, 1993). Trains of multiple galvanic skin response peaks or electrodermal "storms" increase with passage of stage N1-N3 sleep (Sano & Picard, 2011). In stage N2 EDRs are associated with K-complexes and may become continuous in stage 4 sleep (Broughton et al., 1965).

Stage R is marked by low amplitude mixed frequency EEG characterized by trains of triangular sawtooth waveforms (2-6 Hz) with maximal amplitudes over central sites that precede bursts of rapid eye movement with sharp irregular deflections (<500 msec; Iber et al., 2007). Ventilation and respiration become faster and more erratic (Hauri, Percy, Hellek-son, Hartmann, & Russ, 1982), muscles become atonic with short irregular bursts of chin or tibial EMG (<250 msec, REM twitches; Iber et al., 2007) accompanied by surges in blood pressure and cessation of sympathetic-nerve discharges (Somers et al., 1993) and the incidence of EDRs becomes infrequent or associated with bursts of REM activity (Broughton et al., 1965). These increases in cardiorespiratory variability are mirrored by increased LF power and an elevated LF/HF ratio in R stages compared to N stages (Baharav et al., 1995).

Quantitative Sleep Quality Assessment

While PSG is the "gold standard" for the assessment and diagnosis of sleep-related disorders, this procedure may be inaccessible, cost prohibitive, or unnecessary for many individuals experiencing sleep disturbances. Sleep health questionnaires are often employed as a first assessment step. These instruments are utilized as general measures of sleep health and daytime dysfunction, and are not specific for any single primary sleep disorder (Mon-dal, Gjevre, Taylor-Gjevre & Lim, 2013). Despite the utility of sleep health questionnaires, they may be inaccurate for some individuals due to poor observation, morning recall or lack of reporting of the previous night's sleep. Physiological measures are not susceptible to these same limitations and offer the individual the opportunity to objectively evaluate sleep history and efficiency. The following sections provides a review of commonly used sleep health questionnaires and physiological assessments that may be utilized outside of a sleep lab or clinical setting.

Self-Report Questionnaires

Self-report scales offer an alternative means of assessing subjective sleep parameters, specifically the feelings, behaviors, and experiences that accompany sleep. Although there are many online screeners that are easily accessible, reliable and validated questionnaires used by professionals

yield more accurate information. Some of the most widely used and scientifically validated questionnaires include the Epworth Sleepiness Scale, the Stanford Sleepiness Scale, and the Pittsburgh Sleep Quality Index (Spriggs, 2015).

Epworth Sleepiness Scale (ESS; Johns, 1991) - The ESS is an eight-item assessment of somnolence, with possible scores from 0 to 24. A score of over 10 is considered to be abnormal. The eight common situations are the respondent's self-reported probability of falling asleep. These everyday situations include while sitting and reading, watching television, sitting inactive in a public place, as a passenger in a car for an hour, while lying down in the afternoon, while sitting and talking to another, sitting quietly after lunch, and in a car while stopped in traffic (Monda et al., 2013).

Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman & Kupfer, 1989) - The PSQI is a seven domain (19 item) self-rated questionnaire evaluating usual sleep habits during the last month. The seven domain scores including: subjective sleep quality, sleep latency, sleep duration, sleep efficiency, daytime dysfunction, sleep fragmentation, and use of sleep aid medications; combine to provide a global sleep quality index score. The possible scores range from 0–21, with greater than five indicative of impaired sleep quality.

Stanford Sleepiness Scale (SSS; Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973) - The SSS has been used to assess sleepiness in sleep deprivation and sleep fragmentation and among shift workers (Pilcher, Schoeling & Prosansky, 2000). It uses a simple scale that assesses how alert you are feeling by recording your "degree of sleepiness" at different times throughout the day. Seven–point Likert-type scale has descriptors ranging from "feeling active, vital alert, or wide awake" (score = 1) to "no longer fighting sleep, sleep onset soon and having dream-like thoughts" (score = 7).

Insomnia Severity Index (ISI; Bastien, Valliéres, & Morin, 2001) - The ISI is a 7-item questionnaire, with 1 item for each of the following categories: 1) difficulty with sleep onset, 2) difficulty with sleep maintenance, 3) problem with early awakening, 4) satisfaction with sleep pattern, 5) interference with daily functioning as a result of sleep problems, 6) noticeability of sleep problem to others, and 7) degree of distress caused by sleep problem. The questionnaire was designed as a brief screening measure of insomnia and an outcome measure for use in treatment research. Content of the ISI corresponds in part to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) diagnostic criteria for insomnia and has been validated against both polysomnographic and prospective sleep diary measures and demonstrates convergence with clinical interview criteria (Omachi, 2011).

Electroencephalography (EEG)

EEG measures the electrical activity in the brain from electrodes placed on the scalp at standardized locations according to the International 10-20 system (Klem, Lüders, Jasper, & Elger, 1999). EEG signals are broken down into frequency bands (Hz) in the following ranges: delta (1.5-3.5 Hz), theta (4-7.5 Hz), alpha (8-12Hz), beta 1 (13–16 Hz), beta 2 (13–21 Hz), beta 3 (21–32 Hz),

and gamma (35–45 Hz)(Niedermeyer, 1999). Different stages of sleep can be determined by analyzing specific EEG details (see Table 1). The literature vastly describes the behavioral components related to each frequency band.

Several studies have presented how EEG changes from wakefulness to sleep and the differences between a good sleeper and a poor sleeper. A good sleeper exhibits higher levels of delta and theta during sleep, while poor sleepers have elevated levels of high frequency activity (Beta: 14-35 Hz and Gamma 35-45 Hz) which increase throughout the night starting at the onset of sleep and continuing through both non-REM and REM sleep (Acher-mann & Borbély, 2003; Perlis, Smith, Andrews, Orff, & Giles, 2001; Šušmáková, 2004). Increased beta activity during sleep has been shown to negatively correlate with self reported perception of sleep quality (Freedman, 1986; Jacobs, Benson, & Friedman, 1993; Lamarche & Ogilvie, 1997; Nofzinger et al., 1999; Perlis et al., 2001). This is important because as the amount of delta, also called sleep pressure, decreases and the amount of beta increases, the brain transitions into a state that is associated with wakefulness. This leads to shorter TST and sleep quality is reduced (Perlis et al., 2001). On the other hand, good sleepers are able to maintain more delta relative to beta, shown to be a healthy sleep pattern, for the entire night. Poor sleepers are only able to maintain that state for 3-4 hours and this likely accounts for an increased number and duration of awakenings (Perlis et al., 2001).

In the awake state, Buckelew, DeGood, Roberts, Butkovic, and MacKewn (2009) observed central (Cz) theta suppression in good sleepers from a resting eyes-open task to a sensory attentiveness task and theta enhancement across these tasks in poor sleepers. This research group also reported that good sleepers demonstrated greater “neuroflexibility”, control over the self-regulation of alpha enhancement and suppression, during bidirectional neurofeedback protocol when compared to poor sleepers (Buckelew et al., 2013).

Cardiorespiratory Activity

Cardiorespiratory activity has been demonstrated to be a reflection of the autonomic ner-vous system including sympathetic and parasympathetic responses. These vagal responses are related to sleep stages. Respiratory volume and frequency are more regular during NREM sleep than during REM sleep and wakefulness. Irregular respiration patterns occurring during wakefulness are usually caused by body movements or alteration of ventilation control manipulated by some external factors; during REM sleep they can be related to muscle atonia or subcortical structures with a possible involvement of the bizarre content of dreams (Long, Haakma, Leufkens, Fonseca & Aarts, 2015).

Heart Rate

Heart rate (HR) is defined as the number of contractions or heartbeats, per unit of time. HR is typically measured in beats per minute (bpm). At any given time, HR represents the net effect of the neural output of the parasympathetic nerves (slow HR) and sympathetic nerves (accelerated HR). The average resting heart rate for healthy adults in the US is 60-100 bpm (American Heart Association, 2015).

With regard to sleep, it has been shown that there is a significant correlation between time it takes to fall asleep (sleep latency) and heart rate. Studies have demonstrated both that nighttime and daytime resting heart rates are related to sleep quality. Johns, Thornton, and Doré (1976) demonstrated that a reduced resting heart rate at night is associated with shorter sleep onset and that as sleep onset occurs, there is a significant decrease in heart rate. Yuksel et al. (2012) demonstrated that self-reported poor sleep quality has been correlated with higher resting heart rate during daytime assessments (Yuksel et al., 2012). Although this study was focused on determining how sleep quality impacted cardiovascular measures during a physical stress test on a treadmill, the outcomes can be added to the large body of evidence solidifying a broader link between sleep quality and cardiovascular health.

Heart Rate Variability

Heart rate variability (HRV) measures oscillations in the interval between consecutive heartbeats (RR intervals) and is under the control of the autonomic nervous system (ANS). HRV can be analyzed in the frequency and time domain to provide insight into the physiological and pathological conditions and quantitative markers of parasympathetic and sympathetic activity in the ANS (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

In the frequency domain, three spectral components have been identified: very low frequency (VLF: 0.01-0.04 Hz), low frequency (LF: 0.04-0.15 Hz), and high frequency (HF: 0.15-0.40 Hz). VLF activity is assumed to reflect humonal factors, temperature, and slow components. LF activity reflects contributions of both the sympathetic and parasympathetic branches of the ANS, while HF activity is considered a marker of parasympathetic activity or vagal modulation.

Respiratory sinus arrhythmia (RSA), one of the physiologic interactions between respiration and circulation, is heart rate variability in synchrony with respiration, by which the R-R interval on an ECG is shortened during inspiration and prolonged during expiration. RSA or heart rate variability in synchrony with respiration is a biological phenomenon, which may have a positive influence on gas exchange at the level of the lung via efficient ventilation/ perfusion matching (Yasuma & Hayano, 2004). In the time domain, variations in heart rate or cycle length are analyzed over longer time periods (e.g., 24 hours), segmented into smaller recordings (e.g. 5 minute), and derived from the NN intervals (normal RR, direct HR) or variations between the NN intervals (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). The RMS-SD, square root of the mean differences in NN interval, and the pNN50, the proportion of interval differences of successive NN intervals greater than 50 ms divided by total number of NN intervals, are common metrics calculated from interval differences and are estimates of short-term, high frequency variations in HRV .

In an investigation of HRV and respiratory changes during sleep onset, Shinar, Akselrod, Dagan, and Baharav (2006) indicated that while VLF, LF, and LF/HF components of HRV decrease among normal controls and individuals with obstructive sleep apnea and sleep disorders, sympathovagal

balance (LF/HF) was highest among individuals with sleep disorders followed by those with sleep apnea and normal sleep quality. Gouin, Wenzel, Deschenes, and Dang-Vu (2013) evaluated the correlational relationship and predictive power of HRV measures in assessing sleep efficiency (time asleep/time in bed) over a 7-day sleep diary period.

The results indicated that HRV parameters (rMSSD, pNN50, and RSA) had a medium to large positive correlation with sleep efficiency (greater parasympathetic dominance, greater sleep efficiency) and were a better predictor of sleep efficiency than individual demographics or psychological distress. Burton, Rahman, Kadota, Lloyd, and Vollmer-Conna (2010) demonstrated similar findings reporting a significant predictive relationship between reduced RMSSD and increased general sleep problems and repeated awakenings, and increased HF power and increased subjective sleep quality. A reciprocal predictive relationship was observed between the report of situational insomnia (Bonnet & Arand, 2003) or sleep deprivation (Dettoni et al., 2012; Glos, Fietze, Blau, Baumann, & Penzel, 2014; Zhong et al., 2005) and decreased HF-HRV or rMSSD (Jackowska, Dockray, Endrighi, Hendrix, & Steptoe, 2012) during morning wakefulness. Finally, in a stress manipulation task, Hall et al. (2004) reported that the typical parasympathetic modulation increase observed in N stage of sleep in non-stressed individuals was blunted in stressed individuals and associated with poorer sleep maintenance and lower delta activity.

Actigraphy/Physical Activity

Actigraphy is the continuous recording of body movement, often by means of a body-worn device that detects movement through acceleration. These devices typically sample movement several times per second and are capable of storing this information, along with the measurement date and time, for days, weeks, or months, until it is downloaded for analysis (Ancoli-Israel et al., 2003). Actigraphy has become a valuable tool in tracking and assessing sleep. It is based off the principle that body movement is reduced during sleep and increased during waking periods (Littner et al., 2003). After data is collected, specialized algorithms are used to estimate common sleep variables such as TST, sleep percentage, and number of awakenings after sleep onset (Martin & Hakim, 2011). Several reviews have demonstrated the parallel between the sleep-wake and rest-activity rhythms which suggest that actigraphy can be cost effective substitution for the standard, polysomnographic techniques (Pollak, Tryon, Nagaraja, & Dzwonczyk, 2001; Martin & Hakim, 2011).

Interestingly, daytime movement may also be another valuable tool for assessing sleep quality. Loprinzi and Cardinal (2011) analyzed data from the National Health and Nutrition Examination Survey (NHANES) 2005-2006 to determine the relationship between sleep quality and daytime physical activity. Participants were interviewed about sleep quality and wore an actigraph for 7 days. The results indicated that after controlling for age, bmi, health status, smoking status, and depression, the relative risk of often feeling overly sleepy during the day, compared to never feeling overly sleepy during the day, decreased by a factor of 0.65 for participants meeting physical activity guidelines compared to those not meeting guidelines. Additionally, participants that met physical activity guidelines reported that they were less likely to inform their doctor that they had trouble sleeping, be informed by their doctor that they have a sleeping disorder,

wake up too early in the morning, feel unrested during the day, feel overly sleepy during the day and to take pills to fall asleep. Overall, greater daytime physical activity was associated with healthier self-reported sleep-related parameters. Furthermore, this research suggests that daytime actigraphy measurements can shed light on an individual's sleep quality.

Electromyography (EMG)

EMG monitoring is a noninvasive way to directly assess electrophysiological muscle activity and is used as a biofeedback method to teach patients to relax their muscles. When a muscle contracts an electrical signal is generated along the muscle fiber and an action potential spreads from the muscle to the skin where it can be recorded with electrodes. The total amount of recorded voltage is proportional to the number of muscle fibers contracting simultaneously (Pluess, Conrad, & Wilhelm, 2009). In sleep research, EMG is similar to EEG in terms of equipment setup, as several surface electrodes are connected to the subject (Lee & Lin, 2012). While direct support of elevated EMG and increased sleep latency or other sleep disturbances has not been observed (Good, 1975), the link between increased anxiety, elevated EMG, and sleep disturbances have been well established (Kahn, Baker, & Weiss, 1968; Zung, 1970; Baekeland, 1970; Gering & Mahrer, 1972).

Sleep Interventions

Efficient sleep has been shown to have many positive effects on mental and physical health. There are a variety of clinical interventions and pharmaceutical options that can be administered if an individual isn't sleeping as soundly or as long as desired. However, there are limitations and challenges to formal sleep interventions.

The most common test used to diagnose sleep disorders is the PSG, described above. This test is expensive (ranging from $700 to $2400), requires the individual to spend the night at a sleep center, and to schedule follow-up appointments for reviewing results and determining best treatment options. Home sleep testing is a version of PSG testing that can be completed outside of a lab at a reduced cost (Brunk, 2014). This is becoming increasingly popular because of the convenience to test at home. However, the results obtained by individuals at home are less reliable than tests performed at sleep clinics due to a high technical failure rate (El Shayeb, Topfer, Stafinski Pawluk, & Menon, 2014). It has been demonstrated that home sleep testing results were only in agreement with PSG results 76% of the time confirming that in-lab PSG remains the best option for accurate results (Hwang, 2013; El Shayeb et al., 2014). Both of these methods require many sensors be correctly attached to the body and the recording device(s) (Patel, Alexander, & Davidson, 2007).

Pharmaceuticals are aimed at improving sleep, reducing body movements during sleep or improving waking functions (Proctor & Bianchi, 2012). About 4% of adults in the U.S. use prescription sleep aids and nearly all of the prescribed medication have serious side effects (Chong, Fryer, & Gu, 2013; Proctor & Bianchi, 2012). If the level of sleep dysregulation has not reached a clinical level, individuals may opt to take over the counter sleep aids or other

alternative options such as caffeine, tracking sleep, breathing exercises, and/or neurofeedback. In 2008, Americans spent more than $600 million on OTC sleep aids which demonstrates the need to find new, effective options to improve sleep quality (Cassell, 2009). Technological advances, such as smart devices, have allowed home-based interventions which address many of the limitations of formal sleep interventions. These are capable of tracking sleep, guiding users through breathing exercises, monitoring (and training) heart rate/heart rate variability and completing neurofeedback training sessions.

Passive Interventions

Tracking Sleep Hygiene/Sleep Diaries. The most affordable and basic way to start to improve sleep is to track sleep. Keeping a sleep diary is an easy first step in working to improve sleep as it can be done at home, over many nights without the supervision of a trained professional. Overtime, the diary will help to recognize personal patterns associated with good and bad sleep. Common variables that are tracked include time to bed, wake up time, the conditions of the room, comfort of the bed, if the bed is being shared with a spouse, child, or pet, and the presence of any technology in the bedroom. It is also important to note if the time to bed and awakening are consistent. Other sleep hygiene contributions can be monitored as well, such as reports of the ambient room temperature and light level, as a cool, dark, quiet room often leads to better sleep. By tracking sleep patterns, it is possible to learn to create healthy habits and patterns that contribute to falling asleep easier, staying asleep longer and feeling more rested (Zoidis, 2006).

Movement/Actigraphy. Actigraphy has been used for over 20 years to track sleep-wake cycles measured by the amount of body movement recorded by a device worn on the wrist (Ancoli-Israel et al., 2003). This is a convenient, accurate, low cost, objective option for tracking TST, percent of time spent awake, and total number of awakenings over time. Additionally, actigraphy is more accurate than sleep diaries as it does not rely on the individual's memory to report how many awakenings occurred during the night. As referenced earlier, actigraphy monitoring can be used to not only assess, but also to help improve sleep quality through awareness and subsequent increase of physical activity.

Monitoring Heart Rate. Another basic, inexpensive and effective way to track sleep quality is to monitor HR. It has been shown that HR is significantly higher in the morning after a night of poor sleep and long term poor sleep is associated with overall elevated HR (Lusardi et al., 1999; Yuksel et al., 2014). HR patterns have also been identified in good sleepers, specifically at the onset of sleep HR decreases and remains lower to meet the reduced metabolic needs of the body during rest (Carrington, Walsh, Stambas, Kleiman, & Trinder, 2003; Penzel, Kantelhardt, Lo, Voigt, & Vogelmeier, 2003). Tracking heart rate in the morning, during the day and before bed will build a clear picture of sleep quality. A lower HR in the morning followed by a slightly increased (normal) daytime HR followed by a reduced HR before bed indicates good sleep quality. On the contrary, an elevated HR in the morning that extends throughout the day and into the night indicates poor sleep quality.

Active Interventions

EMG Biofeedback. As with HRV biofeedback and neurofeedback, EMG biofeedback involves the real-time assessment, feedback, and reward of muscle activity. The findings related to the efficacy of EMG biofeedback for the treatment of sleep-related disorder are limited and require further investigation.

Turner and Ascher (1979) investigated the efficacy of three treatment conditions (paradoxical intention, stimulus control, and progressive muscle relaxation) compared to a placebo and non-treatment control condition, indicating that all three treatments produced a significant reduction in sleep latency, number of awakenings, and difficulty falling asleep compared to the control conditions. They hypothesized that the lack of statistical significance among the treatment conditions was linked to individual differences in the type of sleep related disturbances and the randomized assignment of the appropriate treatment condition to address those issues. This hypothesis was supported by post-hoc findings of Hauri (1981), which indicated that application of EMG biofeedback or a combined neuro/biofeedback protocol rewarding the augmentation of EEG activity in the theta range along with the inhibition of EMG activity was more effective for individuals identified as "high-arousal" insomniacs with elevated tension levels at study baseline. The efficacy of EMG biofeedback was further tested indicating that the inhibition of EMG resulted in a significant decrease in sleep latency (Cortoos, De Valck, Arns, Brentler, & Cluydts, 2010; Freedman & Papsdorf, 1976), awakenings after sleep onset, and sleep efficiency (Cortoos et al., 2010).

CardioRespiratory Training. CardioRespiratory training (moderate-intensity aerobic physical activity) is one such alternative that is inexpensive and affects numerous health systems simultaneously. Exercise is associated with ease of falling asleep, deepness of sleep, a sense of well-being, and morning alertness (Buman & King, 2010). HRV, a noninvasive marker of parasympathetic activity is augmented after exercise (De Meersman, 1993). However, Browman and Tepas (1976) indicated that 45 minutes of moderate exercise within an hour of sleep increased sleep latency compared to a monotonous task or progressive muscle relaxation.

Respiratory Exercises. Guided breathing exercises are an active method to aid in preparing the body for sleep. Breathing exercise are proven to significantly reduce heart rate and decrease the effects of stress in a normal population (Pal & Velkumary, 2003; Turankar et al., 2013). Actively working to reduce HR before bed by performing 0.1Hz breathing exercises (6 breaths/min) has been shown to reduce sleep latency and improve overall reported sleep quality (Tsai, Kuo, Lee, & Yang 2015). In addition, performing slow paced breathing helps the individual enter deep sleep in less time and reduces time/number of awakenings throughout the night.

Heart Rate Variability. Applied HRV training or HRV biofeedback involves the real-time assessment, feedback, and conditioning of heart rhythms through reward conditioning of non-invasive earlobe and finger pulse information (McCraty & Shaffer, 2015). Sakakibara, Hayano, Oikawa, Katsamanis, and Lehrer (2013) evaluated the impact of HRV biofeedback before bed on HRV activity during

sleep compared to autogenic training and a non-treatment control. Results indicated that as few as two sessions of HRV training increased HF activity during sleep, an index of parasympathetic and restorative function, while it remained unchanged following autogenic training or usual sleep routine.

Neurofeedback. An emerging technology that is non-evasive and highly effective at improving sleep quality is neurofeedback (NFB). NFB is a process that involves analyzing EEG and providing feedback in real-time. This type of training requires an individual to learn how to alter specific aspects of cortical activity such as amplitude (uV) and frequency (Hz) through operant conditioning; specifically when the prescribed EEG activity is reached a reward is earned (Sherlin et al., 2011).

Several studies have demonstrated how NFB can be used to improve sleep quality. In animal research, Sterman, Howe, and MacDonald (1970) demonstrated that the conditioning of enhanced sensorimotor rhythm (SMR) during the awake state led to increased sleep spindle bursts, quiet sleep, and suppression of motor activity in cats. In humans, SMR enhancement training has been shown to reduce sleep latency, decrease number of awakenings, increase time spent in slow-wave (deep) sleep, and improve overall reported sleep quality (Arns, Feddema, & Kenemans, 2014; Cortoos, De Valck, Arns, Breteler, & Cluydts, 2010; Hammer, Colbert, Brown, & Ilioi, 2011; Hauri, 1981; Hauri et al., 1982; Hoedlmoser et al.. 2008, Schabus et al., 2011; 2014). Additionally, up-training theta has been shown to reduce sleep latency, increase TST, and improve subjective reports of sleep quality (Bell, 1979; Browne, 2000; Hauri, 1981; Hauri et al., 1982). Importantly, these positive results were shown to be maintained over time (Bell, 1979; Hauri et al., 1982). Although neurofeedback is typically thought of only as a clinical application, technology advancements in the last 5 years have allowed even greater access to EEG monitoring and even training outside the health care setting (e.g., Wyckoff, Sherlin, Ford, & Dalke, 2015).

Heart-Brain Connection

The preceding sections briefly described the relationship between specific physiological parameters and sleep. These patterns of activity do not exist in a vacuum, rather they reflect the coordinated actions of the central and peripheral nervous system. An extensive body of research supports the Neurovisceral Integration Model, detailing the direct and indirect connection of the heart and brain for emotion regulation, cognitive function, and executive performance (Thayer & Lane, 2000, 2009; Thayer & Ruiz-Padial, 2006; Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012; Thayer, Hansen, Saus-Rose, & Johnsen, 2009; Thay-er & Ruiz-Padial, 2006). One of the core principles of the Neurovisceral Integration Model is the idea that HRV activity is not only important for what it indicates about the heart, but also for what it tells us about the brain. This model has been used to investigate the heart-brain connection across sleep stages (Lechinger, Heib, Gruber, Schabus, & Klimesch, 2015) and is further supported by the bidirectional relationship observed in simultaneous EEG and HRV feedback studies.

Lechinger et al. (2015) investigated the modulatory effects of heartbeat on EEG activity across

sleep stages, reporting that heartbeat evoked frontocentral EEG amplitude and phase locking, occurring approximately 300-400 ms following the R peak, decreased with the deepening of N stage sleep and increased during R stage sleep. Heart rate was also correlated with the individual alpha peak frequency during wakefulness and decreased with increasing sleep depth. The QRS complex of heartbeat activity was further observed to have a modulatory effect on EEG spindle phase and frequency (12-15 Hz) during Stage N2 sleep.

Taken together these findings demonstrate that cardiac activity can modulate or be modulated by oscillatory brain activity. This reciprocal relationship has been further demonstrated in neuro/biofeedback research. Bazanova, Balioz, Muravleva, and Skoraya (2013) investigated the HRV (pNN50) effects of a 10-session eyes-closed EEG alpha power enhancement training protocol versus a sham-feedback control in a sample of healthy young males. The results revealed that alpha enhancement training in individuals with a low baseline alpha frequency led to improved cognitive performance, reduced stress, and increased HRV (pNN50), while training in individuals with a high baseline alpha frequency only led to a decrease in HRV.

Conversely, Sherlin, Muench, and Wyckoff (2 10), investigated QEEG and sLORETA changes following a single session of RSA biofeedback versus a sham-feedback control in a sample of adults reporting moderate to severe stress. The results indicated a significant eduction of scores on the State-Trait Anxiety Inventory (Spielberger, Gorsuch, Luschene, Vagg, & Jacobs, 1983) for the individuals in the RSA training group (Sherlin, Gevirtz, Wyckoff, & Muench, 2009) as well as increases in theta and alpha absolute power, and a dose response relationship for enhanced RSA regulation with decreased beta power (Sherlin et al., 2010). sLORETA analysis also revealed an increase in alpha relative power in Brodmann's area 24 and decreases in relative beta activity in Brodmann's areas 30 and 31, highlighting the modulatory effect of RSA training on cortical components of the limbic system associated with emotion processing and affective dimensions of pain (Williams, White, & Mace, 2005).

Collectively, these neuro/biofeedback techniques (EEG, HRV, EMG) have been classified by the American Academy of Sleep Medicine at the "Guideline" level, indicating a moderate degree of clinical certainty supported by Level II evidence (randomized control trials with high-beta error probabilities; Chesson et al, 1999). However, applied research is required to investigate the heart-brain connection in sleep, as well as the efficacy, specificity, and combined effects of neurofeedback and HRV biofeedback.

Discussion

In this review the consequences of sleep-related issues and the physiological characteristics of normal and pathological sleep were described. Subjective and objective assessment and non-pharmacological intervention strategies accessible to the general population were identified Recent advancements in smart device technology are becoming more effectively integrated into health care settings, as they gain credibility and reliability with professional health care providers (Luxton, McCann, Bush, Mishkind, & Reger, 2011). Despite this growing utility, several interventions have been omitted (prescription medication, CPAP, stimulus control, paradoxical intention, sleep restriction therapy, cognitive-behavioral therapy, etc.) or reported only the briefest information

(EMG Biofeedback) in this report, as we wanted to restrict our scope to those metrics and actions that are more easily accessible without any necessary mediation by a healthcare professional.

Today, accessibility of these interventions is growing as portable and mobile platforms are developed and released to general consumers (Consumer Electronics Association [CEA], 2015). For example, neurofeedback and biofeedback treatments, which were once only available in a clinical and research setting under the supervision of a licensed or certified provider, are now becoming increasingly available to broader consumer markets.

New technology advancements in physiological and health behavior monitoring designed for consumer electronics, like smartphones and tablets, now allow many users to begin to engage in lifestyle monitoring and behavior change with greater ease and interest. Sleep monitoring and interventions are of particular interest, as the prevalence and cost of sleep deprivation creates increasing demand for addressing this growing problem. Consumer Electronics Association (2015) recently published survey data aimed at identifying consumer demand for technology advancements for sleep monitoring and interventions for smart devices. Results suggest that almost a quarter of adults in the United States currently use technology to monitor or aid their sleep quality. Additionally that the mobile technology category of "health and fitness", under which sleep monitoring and interventions fall, is projected to produce $1.8 billion in wholesale revenue in 2015 (CEA, 2015).

Thus the consumer market is primed for improving sleep patterns with the use of mobile technology. It should be noted that additional research is needed to further investigate the efficacy, mechanisms of action and long-term or combined effects of sleep interventions, like those reviewed, as they are rendered into mobile applications.

References

Achermann, P., & Borbély, A. A. (2003). Mathematical models of sleep regulation. Frontiers in Bioscience, 8(1-3), S683-S693. doi: 10.2741/1064

American Electroencephalographic Society. (1994). Guideline fif een: Guidelines for polygraphic assessment of sleep-related disorders (polysomnography). Journal of Clinical Neurophysiology, 11(1), 116-124. doi: 10.1097/00004691-199201000-00010

American Heart Association. Target heart rates. 1 Jan 2015. Retrieved 30 October 2015 from: http://www.heart.org/HEARTORG/GettingHealthy/PhysicalActivity/FitnessBasics/Target-Heart-Rates_UCM_434341_Article.jsp#.Vj0N5rerTmE

Ancoli-Israel, S., Cole, R., Alessi, C., Chambers, M., Moorcroft, W., & Pollak, C. (2003). The role of actigraphy in the study of sleep and circadian rhythms. Sleep, 26(3), 342-392.

Arns, M., Feddema, I., & Kenemans, J. L. (2014). Differential effects of theta/beta and SMR neurofeedback in ADHD on sleep onset latency. Frontiers in Human Neuroscience, 8, 1019. doi: 10.3389/fnhum.2014.01019

Baekeland, F. (1970). Effects of the day's events on sleep. In E. Hartmann (Ed.), Sleep and dreaming. (pp. 49-58). Boston, MA: Little Brown and Company.

Bastien, C. H., Vallières, A., & Morin, C. M. (2001). Validation of the Insomnia Severity Index as an outcome measure for insomnia research. Sleep Medicine, 2(4), 297-307. doi: 10.1016/s1389-9457(00)00065-4

Bazanova, O. M., Balioz, N. V., Muravleva, K. B., & Skoraya, M. V. (2013). Effect of voluntary EEG power increase training on heart rate variability. Human Physiology, 39(1), 86-97. doi: 10.1134/s0362119712060035

Bell, J. S. (1979). The use of EEG theta biofeedback in the treatment of a patient with sleep-onset insomnia. Biofeedback and Self-regulation, 4(3), 229-236. doi: 10.1007/bf00998824

Bixler, E. (2009). Sleep and society: An epidemiological perspective. Sleep Medicine, 10, S3-S6. doi: 10.1016/j.sleep.2009.07.005

Bonnet, M. H., & Arand, D. L. (2003). Situational insomnia: Consistency, predictors, and out-comes. Sleep, 26(8), 1029-1036. doi:10.1007/s11251-013-9273-6

Brunk, D. (2014, June 15). Home testing for apnea beats sleep lab on cost. Family Practice News. Retrieved from: http://www.highbeam.com/doc/1G1-374627967.html

Broughton, R. J., Poiré, R., & Tassinari, C. A. (1965). The electrodermogram (Tarchanoff ef-fect) during sleep. Electroencephalography and Clinical Neurophysiology, 18(7), 691-708. doi: 10.1016/0013-4694(65)90113-6

Browman, C. P., & Tepas, D. I. (1976). The effects of presleep activity on all-night sleep. Psychophysiology, 13 (6), 536-540. doi: 10.1111/j.1469-8986.

Browne, T. G. (2000). EEG theta enhancement and heart rate variability biofeedback on interactional stress in a clinical population (Doctoral dissertation). Retrieved from: ProQuest Dissertations & Theses Full Text. Retrieved from: http://search.proquest.com/docview/304650752?accountid=458

Buckelew, S. P., De Good, D. E., Taylor, J., Cunningham, N. B., Thornton, J., & MacKewn, A. (2013). Neurofl xibility and sleep onset insomnia among college students: Implications for neurotherapy. Journal of Neurotherapy, 17(2), 106-115. doi: 10.1080/10874208.2013.784681

Buman, M. P., & King, A. C. (2010). Exercise as a treatment to enhance sleep. American Journal of Lifestyle Medicine, 4(6), 500-514. doi: 10.1177/1559827610375532

Burton, A. R., Rahman, K., Kadota, Y., Lloyd, A., Vollmer-Conna, U. (2010). Reduced heart rate variability predicts poor sleep quality in a case-control study of chronic fatigue syn-drome. Experimental Brain Research, 204, 71-78. doi:10.1007/s00221-010-2296-1

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. Psychiatry Research, 28(2), 193-213. doi: 10.1016/0165-178(89)90047-4

Carskadon, M.A., & Dement, W.C. (2011). Monitoring and staging human sleep. In M.H. Kry-ger, T. Roth, & W.C. Dement (Eds.), Principles and practice of sleep medicine (5th ed.), (pp 16-26). St. Louis: Elsevier Saunders.

Carrington, M., Walsh, M., Stambas, T., Kleiman, J., & Trinder, J. (2003). The influen e of sleep onset on the diurnal variation in cardiac activity and cardiac control. Journal of Sleep research, 12(3), 213-221. doi: 10.1046/j.1365-2869.2003.00364.x

Cassell, D. K. (2009). You snooze, you win with OTC sleep aids. Drug Topics, 153(12), 70.

Chesson, A. L., McDowell Anderson, W., Littner, M., Davilla, D., Hartse, K., Johnson, S., ...Rafe-cas, J. (1999). Practice parameters for the nonpharmacologic treatment of chronic insom-nia. An American Academy of Sleep Medicine report. Standards of practice committee of the American Academy of Sleep Medicine. Sleep, 22(8), 1128-1133.

Chong, Y., Fryer, C. D., Gu, Q. (2013). Prescription sleep aid use among adults: United States,2005–2010. NCHS Data Brief, 127, 1-8. Retrieved from: http://www.cdc.gov/nchs/data/databriefs/db127.pdf

Colten, H. R., & Altevogt, B. M. (Eds). (2006). Sleep disorders and sleep deprivation: An unmet public health problem. Washington (DC): National Academies Press. Available from: http://www.ncbi.nlm.nih.gov/books/NBK19960/

Connor, J., Norton, R., Ameratunga, S., Robinson, E., Civil, I., Dunn, R.,...Jackson, R. (2002). Driver sleepiness and risk of serious injury to car occupants: Population based case control study. BMJ, 324(7346), 1125. doi:10.1136/bmj.324.7346.1125

Consumer Electronics Association (2015). Consumer awareness and perception of sleep technology. October, 2015.

Cooke, J. R. (2008). Sleep disorders in the elderly. Psychiatric Times, 25(4), 76-79.

Cortoos, A., De Valck, E., Arns, M., Breteler, M. H. M., & Cluydts, R. (2010). An exploratory study on the effects of tele-neurofeedback and tele-biofeedback on objective and sub-jective sleep in patients with primary insomnia. Applied Psychophysiology and Biofeed-back,35(2), 125-134 doi: 10.1007/s10484-009-9116-z

De Meersman, R. E. (1993). Heart rate variability and aerobic fitne s. American Heart Jour-nal, 125(3), 726-731. doi: 10.1016/0002-8703(93)90164-5

Dettoni, J. L., Consolim-Colombo, F. M., Drager, L. F., Rubira, M. C., Cavasin de Souza, S. B. P., Irigoyen, M. C., ...Lorenzi-Filho, G. (2012). Cardiovascular effects of partial sleep depriva-tion in healthy volunteers. Journal of Applied Physiology, 113(2), 232-236. doi: 10.1152/jap-plphysiol.01604.2011

El Shayeb, M., Topfer, L.A., Stafinski T., Pawluk, L., & Menon, D. (2014). Diagnostic accuracy of level 3 portable sleep test versus level 1 polysomnography for sleep-disordered breath-ing: A systematic review and meta-analysis. Canadian Medical Association Journal, 186(1), E25-51. doi: 10.1503/cmaj.130952

Freedman, R. R. (1986). EEG power spectra in sleep-onset insomnia. Electroencephalogra-phy and Clinical Neurophysiology, 63(5), 408-413. doi: 10.1016/0013-4694(86)90122-7

Freedman, R., & Papsdorf, J. D. (1976). Biofeedback and progressive relaxation treatment of sleep-onset insomnia: A controlled, all-night investigation. Biofeedback and Self-Regula-tion, 1(3), 253-271. doi: 10.1007/bf01001167

Fuller, P. M., Gooley, J. J., & Saper, C. B. (2006). Neurobiology of the sleep-wake cycle: Sleep architecture, circadian regulation, and regulatory feedback. Journal of Biological Rhythms, 21(6), 482-493. doi:10.1177/0748730406294627

Gering, R., & Mahrer, A. (1972). Difficulty alling asleep. Psychological Reports, 31(2), 523-528. doi: 10.2466/pr0.1972.31.2.523

Glos, M., Fietze, L, Blau, A., Baumann, G., & Penzel, T. (2014). Cardiac autonomic modulation and sleepiness: Physiological consequences of sleep deprivation due to 40 h of prolonged wakefulness. Physiology & Behavior, 125, 45-53. doi: 10.1016/j.physbeh.2013.11.011

Good, R. (1975). Frontalis muscle tension and sleep latency. Psychophysiology, 12(4), 465-467. doi: 10.1111/j.1469-8986.1975.tb00025.x

Gouin, J., Wenzel, K., Deschenes, S., & Dang-Vu, T. (2013). Heart rate variability predicts sleep efficie y. Sleep Medicine, 14, e142. doi: 10.1016/j.sleep.2013.11.321

Hall, M., Vasko, R., Buysse, D., Ombao, H., Chen, Q., Cashmere, J. D.,...Thayer, J. F. (2004). Acute stress affects heart rate variability during sleep. Psychosomatic Medicine, 66(1), 56-62. doi: 10.1097/01.psy.0000106884.58744.09

Hammer, B. U., Colbert, A. P., Brown, K. A., & Ilioi, E. C. (2011). Neurofeedback for insomnia: A pilot study of Z-score SMR and individualized protocols. Applied Psychophysiology and Biofeedback, 36(4), 251-264. doi: 10.1007/s10484-011-9165-y

Harrison, Y., & Horne, J. A. (2000). The impact of sleep deprivation on decision making: A review. Journal of Experimental Psychology: Applied, 6(3), 236. doi: 10.1037//1076-898x.6.3.236

Hauri, P. (1981). Treating psychophysiologic insomnia with biofeedback. Archives of General Psychiatry, 38(7), 752-758. doi:10.1001/arcphsyc.1981.01780320032002

Hauri, P. J., Percy, L., Hellekson, C., Hartmann, E., & Russ, D. (1982). The treatment of psycho-physiologic insomnia with biofeedback: A replication study. Biofeedback and Self-Regula-tion, 7(2), 223-235. doi: 10.1007/bf00998785

Hoddes, E., Zarcone, V., Smythe, H., Phillips, R., & Dement, W. C. (1973). Quantific tion of sleepiness: A new approach. Psychophysiology, 10(4), 431-436. doi: 10.1111/j.1469-8986.1973. tb00801.x

Hoedlmoser, K., Pecherstorfer, T., Gruber, G., Anderer, P., Doppelmayr, M., Klimesch, W., & Schabus, M. (2008). Instrumental conditioning of human sensorimotor rhythm (12–15 Hz) and its impact on sleep as well as declarative learning. Sleep, 31(10), 1401.

Hwang, D. (2013). Home sleep testing versus in-lab polysomnography. Sleep Review, 14(1), 16.

Iber, C., Ancoli-Israel, S., Quan, S. F. (2007). The AASM manual for the scoring of sleep and associated events: Rules, terminology, and technical specific tions for the American Acad-emy of Sleep Medicine (1st ed.). Westchester, IL: American Academy of Sleep Medicine.

Jackowska, M., Dockary, S., Endrighi, R., Hendrix, H., & Steptoe, A. (2012). Sleep problems and heart rate variability over the working day. Journal of Sleep Research, 21, 434-440. doi: 10.1111/ j.1365-2869.21012.00996.

Jacobs, G. D., Benson, H., & Friedman, R. (1994). Home-based central nervous system as-sessment of a multifactor behavioral intervention for chronic sleep-onset insomnia. Behav-ior Therapy, 24(1), 159-174. doi: 10.1016/S0005-7894(05)80261-8

Jean-Louis, G., Williams, N. J., Sarpong, D., Pandey, A., Youngstedt, S., Zizi, F., & Ogedegbe, G. (2014). Associations between inadequate sleep and obesity in the US adult population: Analysis of the national health interview survey (1977–2009). BMC Public Health, 14(1), 290. doi: 10.1186/1471-2458-14-290

Johns, M. W. (1991). A new method for measuring daytime sleepiness: The Epworth sleepi ness scale. Sleep, 14(6), 540-545.

Johns, M. W., Thornton, C., & Doré, C. (1976). Heart rate and sleep latency in young men. Journal of Psychosomatic Research, 20(6), 549-553. doi: 10.1016/0022-3999(76)90056-8

Kahn, M., Baker, B. L,, & Weiss, J. M. (1968). Treatment of insomnia by relaxation training. Journal of Abnormal Psychology, 73(6), 556-558. doi: 10.1037/h0026599

Klem, G. H., Lüders, H. O., Jasper, H. H., & Elger, C. (1999). The ten-twenty electrode system of the International Federation. Electroencephalography & Clinical Neurophysiology, 52, 3-6.

Knutson, K. L., Spiegel, K., Penev, P., & Van Cauter, E. (2007). The metabolic consequences of sleep deprivation. Sleep Medicine Reviews, 11(3), 163-178. doi: 10.1016/j.smrv.2007.01.002

Kripke, D. F., Simons, R. N., Garfin el, L., & Hammond, E. C. (1979). Short and long sleep and sleeping pills: Is increased mortality associated? Archives of General Psychiatry, 36(1), 103-116. doi: 10.1001/archpsyc.1979.01780010109014

Lamarche, C. H., & Ogilvie, R. D. (1997). Electrophysiological changes during the sleep on-set period of psychophysiological insomniacs, psychiatric insomniacs, and normal sleepers. Sleep, 20(9), 724-733.

Lechinger, J., Heib, D. P. J., Gruber, W., Schabus, M., & Klimesch, W. (2015). Heartbeat-related EEG amplitude and phase modulations from wakefulness to deep sleep: Interactions with sleep spindles and slow oscillations. Psychophysiology, 52, 1441-1450. doi: 10.111/psyp.12508

Lee, J-K., & Lin, C-T. (2012). Biosensors for sleep technology. In R. P-Y. Chiang & S-C. Kang (Eds.). Introduction to modern sleep technology (pp. 201-217). Netherlands: Springer.

Littner, M., Kushida, C. A., Anderson, W. M., Bailey, D., Berry, R. B., Davila, D. G., ...Johnson, S. F. Standards of Practice Committee of the American Academy of Sleep Medicine. (2003). Practice parameters for the role of actigraphy in the study of sleep and circadian rhythms: An update for 2002. Sleep, 26(3), 337-341.

Loomis, A. L., Harvey, E. N., & Hobart, G. A. (1937). Cerebral states during sleep, as studied by human brain potentials. Journal of Experimental Psychology, 21(2), 127. doi: 10.1037/h0057431

Long, X., Haakma, R., Leufkens, T. R., Fonseca, P., & Aarts, R. M. (2015). Effects of be-tween-and within-subject variability on autonomic cardiorespiratory activity during sleep and their limitations on sleep staging: a multilevel analysis. Computational Intelligence and Neuroscience, 2015, 1-17. doi: 10.1155/2015/583620

Loprinzi, P. D., & Cardinal, B. J. (2011). Association between objectively-measured physical activity and sleep, NHANES 2005–2006. Mental Health and Physical Activity, 4(2), 65-69. doi: 10.1016/j.mhpa.2011.08.001

Lusardi, P., Zoppi, A., Preti, P., Pesce, R. M., Piazza, E., & Fogari, R. (1999). Effects of insuffi-cient sleep on blood pressure in hypertensive patients A 24-h study. American Journal of Hypertension, 12(1),63-68. doi: 10.1016/s0895-7061(98)00200-3

Luxton, D.D., McCann, R.A., Bush, N.E., Mishkind, M.C., & Reger, G.M. (2011). mHealth for mental health: Integrating smartphone technology in behavioral healthcare. Professional psychology: Research and Practice, 42(6), 505-512.

Martin, J. L., & Hakim, A. D. (2011). Wrist actigraphy. Chest Journal, 139(6), 1514-1527. doi: 10.1378/chest.10-1872

McCraty, R., & Shaffer, F. (2015). Heart rate variability: New perspectives on physiological mechanisms, assessment of self-regulatory capacity, and health risk. Global Advances in Health and Medicine, 4(1), 46-61. doi: 10.7453/gahmj.2014.073

Merica, H., & Fortune, R. D. (2004). State transitions between wake and sleep, and with-in the ultradian cycle, with focus on the link to neuronal activity. Sleep Medicine Reviews, 8(6), 473-485. doi: 10.1016/j.smrv.2004.06.006

Mondal, P., Gjevre, J. A., Taylor-Gjevre, R. M., & Lim, H. J. (2013). Relationship between the Pittsburgh Sleep Quality Index and the Epworth Sleepiness Scale in a sleep laboratory re-ferral population. Nature and Science of Sleep, 5, 15-21. doi: 10.2147/nss.s40608

Murali, N. S., Svatikova, A., & Somers, V. K. (2003). Cardiovascular physiology and sleep. Frontiers in Bioscience, 8(1-3), s636-52. doi: 10.2741/1105

National Sleep Foundation. (2015). 2015 sleep in America poll: Sleep and pain. Washington (DC): The Foundation. doi:10.1016/j.sleh.2015.02.005. Retrieved from: http://sleepfounda-tion.org/sleep-polls-data/2015-sleep-and-pain

Niedermeyer, E. (1999). The normal EEG of the waking adult. In E. Niedermeyer & F. Lopes da Silva (Eds.), Electroencephalography: Basic principles, clinical applications and related fields (pp. 149-173). Baltimore, MD: Lippincott William & Wilkins.

Nofzinger, E. A., Nowell, P.D., Buysse, D. J., Vasco, R. C., Thase, M. E., Frank, E., & Reynolds, C. F. (1999). Towards a neurobiology of sleep disturbance in primary insomnia and depres-sion: A comparison of subjective, visually scored, period amplitude, and power spectral density sleep measures. Sleep, 22(Suppl 1), S99-S99.

Omachi, T. A. (2011). Measures of sleep in rheumatologic diseases: Epworth Sleepiness Scale (ESS), Functional Outcome of Sleep Questionnaire (FOSQ), Insomnia Severity In-dex (ISI), and Pittsburgh Sleep Quality Index (PSQI). Arthritis Care & Research, 63(S11), S287-S296. doi: 10.1002/acr.20544

Pal, G. K., & Velkumary, S. (2004). Effect of short-term practice of breathing exercises on autonomic functions in normal human volunteers. Indian Journal of Medical Research, 120(2), 115-121. doi:

Penzel, T., Kantelhardt, J. W., Lo, C. C., Voigt, K., & Vogelmeier, C. (2003). Dynamics of heart rate and sleep stages in normals and patients with sleep apnea. Neuropsychopharmacolo-gy, 28(S1), S48-S53. doi: 10.1038/sj.npp.1300146

Patel, M. R., Alexander, T. H., & Davidson, T. M. (2007). Home sleep testing. Opera-tive Techniques in Otolaryngology-Head and Neck Surgery, 18(1), 33-51. doi:10.1016/j.otot.2007.01.005

Perlis, M. L., Smith, M. T., Andrews, P. J., Orff, H., & Giles, D. E. (2001). Beta/gamma EEG ac-tivity in patients with primary and secondary insomnia and good sleeper controls. Sleep, 24(1), 110-117.

Pilcher, J. J., Schoeling, S. E., & Prosansky, C. M. (2000). Self-report sleep hab-its as predictors of subjective sleepiness. Behavioral Medicine, 25(4), 161-168. doi: 10.1080/08964280009595745

Pluess, M., Conrad, A., & Wilhelm, F. H. (2009). Muscle tension in generalized anxiety disor-der: A critical review of the literature. Journal of Anxiety Disorders,23(1), 1-11. doi: 10.1016/j.janxdis.2008.03.016

Pollak, C. P., Tryon, W. W., Nagaraja, H., & Dzwonczyk, R. (2001). How accurately does wrist actigraphy identify the states of sleep and wakefulness? Sleep, 24(8), 957-965.

Proctor, A., & Bianchi, M. T. (2012). Clinical pharmacology in sleep medicine. ISRN Pharma-cology, 2012,914168. doi:10.5402/2012/914168

Rechtschaffen, A., & Kales, A. (1968). A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects.

Sakakibara, M., Hayano, J., Oikawa, L. O., Katsamanis, M., & Lehrer, P. (2013). Heart rate vari-ability biofeedback improves cardiorespiratory resting function during sleep. Applied Psy-chophysiology & Biofeedback, 38, 265-271. doi: 10.1007/s10484-013-9232-7

Sano, A., & Picard, R. W. (2011, August). Toward a taxonomy of autonomic sleep patterns with electrodermal activity. In Engineering in Medicine and Biology Society, EMBC, 2011 Annual International Conference of the IEEE (pp. 777-780). IEEE.

Schabus, M., Griessenberger, H., Hoedlmoser, K., Heib, D., Lechinger, J., Chwala-Schlegel, N., & Klimesch, W. (2011). Sensorimotor-rhythm neurofeedback and its effect on EEG and sleep quality. Frontiers in Human Neuroscience Conference Abstract: XI International Con-ference on Cognitive Neuroscience (ICON XI). doi: 10.3389/conf.fnhum.2011.207.00235

Schabus, M., Heib, D. P., Lechinger, J., Griessenberger, H., Klimesch, W., Pawlizki, A., ... & Hoedlmoser, K. (2014). Enhancing sleep quality and memory in insomnia using instrumen-tal sensorimotor rhythm conditioning. Biological Psychology, 95, 126-134. doi:10.1016/j.biopsycho.20133.02.020

Spriggs, W. H. (2015). Essentials of polysomnography: A training guide and reference for sleep technicians (2nd ed.). Burlington, MA: Jones & Bartlett Learning

Sherlin, L. H., Arns, M., Lubar, J., Heinrich, H., Kerson, C., Strehl, U., & Sterman, M. B. (2011). Neurofeedback and basic learning theory: Implications for research and practice. Journal of Neurotherapy, 15(4), 292-304. doi: 10.1080/10874208.2011.623089

Sherlin, L. H., Gevirtz, R., Wyckoff, S., & Muench, F. (2009). Effects of respiratory sinus ar-rhythmia biofeedback versus passive biofeedback control. International Journal of Stress Management, 16(3), 233–248. doi: 10.1037/a0016047

Sherlin, L. H., Muench, F., & Wyckoff, S. N. (2010). Respiratory sinus arrhythmia feedback in a stressed population exposed to a brief stressor demonstrated by quantitative EEG and sLORETA.

Shinar, Z., Akselrod, S., Dagan, Y., & Baharav, A. (2006). Autonomic changes during wake–sleep transition: A heart rate variability based approach. Autonomic Neuroscience, 130(1), 17-27. doi: 10.1016/j.autneu.2006.04.006

Somers, V. K., Dyken, M. E., Mark, L. M., Abboud, F. M. (1993). Sympathetic-nerve activity during sleep in normal subjects. New England Journal of Medicine, 328(5), 303-307. doi: 10.1056/nejrn199302043280502

Spielberger, C. D., Gorsuch, R. L., Luschene, R. E., Vagg, P. R., & Jacobs, G. A. (1983). Manual for the State–Trait Anxiety Inventory form Y. CA: Mind Garden Press.

Sterman, M. B., Howe, R. C., & MacDonald, L. R. (1970). Facilitation of spindle-burst sleep by conditioning of electroencephalographic activity while awake. Science, 167(3921), 1146–1148. doi: 10.1126/science.167.3291.1146

Šušmáková, K. (2004). Human sleep and sleep EEG. Measurement Science Review, 4(2), 59-74. Retrieved from: http://citeseerx.ist.psu.edu/viewdoc/download?-doi=10.1.1.188.8924&rep=rep1&type=pdf

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability. Standards of measurement, physiological interpretation and clinical use. European Heart Journal, 17(3), 354–381. doi: 10.1093/oxfordjournals.eurheartj.a01468

Thayer, J. F., Ahs, F., Fredrikson, M., Sollers, J. J., & Wager, T. D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. Neuroscience and Biobehavioral Reviews, 36, 747-756. doi: 10.1016/j.neubiorev.2011.11.009

Thayer, J. F., Hansen, A. L., Saus-Rose, E., & Johnsen, B. H. (2009). Heart rate variability, prefrontal neural function, and cognitive performance: The neurovisceral integration per-spective on self-regulation, adaptation, and health. Annals of Behavioral Medicine, 37(2), 141-153. doi: 10.1007/s12160-009-9101-z

Thayer, J.F., & Lane, R.D. (2000). A model of neurovisceral integration in emotion regu-lation and dysregulation. Journal of Affective Disorders 61, 201–216. doi: 10.1016/s0165-0327(00)00338-4

Thayer, J. F., & Lane, R. D. (2009). Claude Bernard and the heart-brain connection: Further elaboration of a model of neurovisceral integration. Neuroscience and Biobehavioral Re-views, 33, 81-88. doi: 10.1016/j.neubiorev.2008.08.004

Thayer, J. F., & Ruiz-Padial, E. (2006). Neurovisceral integration, emotions and health: An update. International Congress Series, 1287, 122-127. doi: 10.1016/j.ics.2005.12.018
Tononi G, Cirelli C. (2006). Sleep function and synaptic homeostasis. Sleep Medicine Re-views, 10(1), 49-62. doi: 10.1016/j.smrv.2005.09.002

Turankar, A. V., Jain, S., Patel, S. B., Sinha, S. R., Joshi, A. D., Vallish, B. N., ... & Turankar, S. A. (2013). Effects of slow breathing exercise on cardiovascular functions, pulmonary functions & galvanic skin resistance in healthy human volunteers-a pilot study. The Indian Journal of Medical Research,137(5), 916. Retrieved from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3734683/

Trinder, J, Whitworth, F, Kay, A , & Wilkin, P. (1992). Respiratory instability during sleep on-set. Journal of Applied Physiology, 73(6), 2462-2469. Retrieved from: http://jap.physiology. org/ content/73/6 /2462.long

Tsai, H. J., Kuo, T. B., Lee, G. S., & Yang, C. C. (2015). Effic y of paced breathing for insom-nia: Enhances vagal activity and improves sleep quality. Psychophysiology, 52(3), 388-396. doi: 10.1111/ psyp.12333

Turner, R. M., & Ascher, L. M. (1979). Controlled comparison of progressive relaxation, stim-ulus control, and paradoxical intention therapies for insomnia. Journal of Consulting and Clinical Psychology, 47(3), 500-508. doi:10.1037/0022-006x.47.3.500

Williams, S. M., White, L. E., & Mace, A. C. (2005). SylviusVG: Visual glossary of human neu-roanatomy. Sunderland, MA: Pyramis Studios, Inc.

Wyckoff, S. N., Sherlin, L. H., Ford, N. L., Dalke, D. (2015). Validation of a wireless dry elec-trode system for electroencephalography. Journal of NeuroEngineering, 12, 95. doi: 10.1186/ s12984-010089-2

Yasuma, F., & Hayano, J. I. (2004). Respiratory sinus arrhythmia: Why does the heart-beat synchronize with respiratory rhythm? Chest Journal, 125(2), 683-690. doi: 10.1378/chest.125.2.683

Yuksel, M., Yildiz, A., Demir, M., Bilik, M. Z., Ozaydogdu, N., Aktan, A., ...Toprak, N. (2014). Effect of sleep quality on hemodynamic response to exercise and heart rate recovery in apparently healthy individuals. Clinical and Investigative Medicine, 37(6), E352-E362.

Zhong, X. Hilton, H. J., Gates, G. J., Jelic, S., Stern, Y., Bartels, M. N.,...Basner, R. C. (2005). Increased sympathetic and decreased parasympathetic modulation in normal humans with acute sleep deprivation. Journal of Applied Physiology, 98(6), 2024-2032. doi: 10.1152/jap-plphysiol.00620.2004

Zoidis, J. D (2006). Tracking sleep movement. Retrieved from: www.sleepreviewmag.com. Zung, W. (1970). Insomnia and disordered sleep. In E. Hartmann (Ed.), Sleep and dreaming. (pp. 123-143). Boston, MA: Little Brown and Company.