The primary function of REM sleep

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In this paper, the physiological features associated with the different stages of REM sleep and with what information processing researchers have called "effort" and "arousal" are compared. It is suggested that tonic REM sleep and effort involve an increase in the metabolism of cerebral glycogen, and phasic REM sleep and arousal involve the transfer of glucose from the body to the brain. Both stages of REM sleep seem to elevate cerebral glucose levels and likely result in increased ATP generation in some part(s) of the brain. It is noted that the functioning of the hippocampus depends heavily on ATP, and that this part of the brain becomes especially active during REM sleep. From this, although many details remain to be clarified, it seems clear that the primary function of REM sleep is to re-energize the brain.

1. Introduction

Although many different theories have been suggested to explain the function of sleep, none has been widely accepted (Zepelin, Siegel, & Tobler, 2005). Common sense suggests that a person is somehow re-energized during sleep, and many scientists and members of the general public share this view. However, it has proved extremely difficult to determine what sort of energy reserve is replenished during sleep, where this reserve is located, when it is replenished, and how the process occurs (Rechtschaffen, 1998). Sleep has long remained "one of the major unanswered questions in biology" and "one of nature's greatest mysteries" (Bennington & Heller, 1995, p. 347; Frank, 2006, p. 47).

I propose that depleted ATP reserves in the hippocampus and associated brain structures are replenished during rapid eye movement (REM) sleep. A comparison of the physiological features associated with REM sleep and what information-processing

researchers have called “effort” and “arousal” strongly suggests that REM sleep facilitates the re-energization of the brain. Hippocampal functioning depends significantly on the presence of ATP (e.g., Inoue, 1998; Inoue, Koizumi, Ueno, Kita, & Tsuda, 1999), and the hippocampus is the part of the brain that becomes especially active during REM sleep (Lerma-Garcia-Austt, 1985; Rimbaud, Passouant, & Cadilhac, 1955).

In this paper, physiological features associated with tonic REM sleep, phasic REM sleep, effort, and arousal are reviewed. It is then noted that tonic REM sleep and effort share a number of significant physiological similarities, and phasic REM sleep and arousal also appear to share a common underlying biological process. It is argued that REM sleep replenishes depleted ATP reserves in the hippocampus and associated brain structures, such as the amygdala. A preliminary interpretation of the physiological features associated with REM sleep is offered.

2. REM sleep

REM sleep was initially discovered by researchers at the University of Chicago in the mid-twentieth century (Aserinsky & Kleitman, 1953), and it has become clear that it fulfills some critical biological function in nearly all mammals and birds (Zepelin, Siegel, & Tobler, 2005). REM sleep and non-REM (NREM) sleep alternate cyclically throughout the sleep cycle. Human infants typically enter REM sleep directly after the initial onset of sleep and spend approximately 50% of their total sleep time in REM sleep. Humans older than two years old typically enter NREM sleep before REM sleep and spend approximately 20% to 25% of their total sleep time in REM sleep “across

childhood, adolescence, adulthood, and into old age" (Carskadon & Dement, 2005, p. 13).

The pupils of the eyes generally appear constricted during REM sleep (Siegel, 2005), but they presumably dilate initially and do so periodically throughout this stage of the sleep cycle (Rechtschaffen, 1998). The heart rate periodically decelerates (Taylor, Moldofsky, & Furedy, 1985; Verrier, Harper, & Hobson, 2005; Verrier et al., 1998). REM sleep has been described as "activated" or "paradoxical" sleep because the brain appears active, and the EEG becomes desynchronized in a manner that resembles wakefulness (Siegel, 2005). It is also possible to observe rhythmic theta waves (Lerma & Garcia-Austt, 1985; Rowe et al., 1999; Sakai, Sano, & Iwahara, 1973; Sei & Morita, 1996), which are closely associated with activity in the hippocampus (Green & Arduini, 1954; Jung & Kornmueller, 1938; Walter & Walter, 1953; Winson, 1975). These are the tonic physiological features of REM sleep.

Each period of REM sleep is interrupted several times by phasic bursts of rapid eye movement (Aserinsky, 1965). In humans, a heart rate deceleration occurs about three seconds before each eye burst (Taylor, Moldofsky, & Furedy, 1985; Verrier, Harper, & Hobson, 2005). As eye activity begins, the heart rate surges, arterial blood pressure increases, and then the heart rate abruptly decelerates (Verrier et al., 2005). The pupils dilate (Kemp & Kaada, 1975; Rechtschaffen, 1998), the eyes jerk frantically back and forth (Aserinsky & Kleitman, 1953), and then the pupils constrict (Rechtschaffen, 1998; Siegel, 2005). Respiratory activity fluctuates (Aserinsky, 1965; Aserinsky & Kleitman, 1953; Sullivan, 1980), and electrodermal activity momentarily increases (Broughton, Poire, & Tassinari, 1965; Kushniruk, Rustenburg, & Ogilvie, 1985). Cerebral blood flow

increases (Franzini, 2005; Franzini, Zoccoli, Cianci, & Lenzi, 1996; Herman & Bassetti, 2002), particularly to the amygdala, anterior cortex, pontine tegumentum, left thalamus, and right parietal operculum (Maquet, 1995; Maquet et al., 1996). These areas as well as the hippocampal formation appear significantly activated on PET scans (Maquet, 2000).

3. Effort and arousal

Researchers have distinguished "three types of energetical supply or resources" that are involved in human information processing (Sanders, 1983, p. 74). These have become known as *energetic pools* (Sergeant, Oosterlaan, & van der Meere, 1999, p. 77). The first is *effort*, which is said to be "mobilized in response to the changing demands of the tasks in which one engages" (Kahneman, 1973, p. 26). It is "conceived of as the necessary energy to meet the demands of a task" (Sergeant et al., 1999, p. 77) and is responsible for controlling the other two pools (Pribram & McGuiness, 1975; Sanders, 1983). *Arousal* is associated with phasic readiness to act and refers to "energy mobilization of the organism" (Sanders, 1983, p. 116). *Activation* is described as "tonic physiological readiness to respond" (Pribram & McGuinness, 1975). The effort and arousal pools are most relevant here.

The concept of effort was developed to explain the fact that increased motivation can improve an individual's performance on a task (Kahneman, 1973). People exert more effort when they are motivated by factors such as time constraints, increased task difficulty, or the promise of incentives or rewards (Lohr, 1999). The pupils dilate during the exertion of effort (Kahneman, 1973; cf. Hess, 1965; Hess, 1964), but only to a point. Then they actually constrict (Granholm, Asarnow, Sarkin & Dykes, 1996; cf. Miller,

1956). The exertion of effort is associated with significant heart rate deceleration (Coles, 1972; Kramer & Spinks, 1991; cf. Kagan, 1965; Lewis, Kagan, Campbell, & Kalafat, 1966). The EEG becomes desynchronized (Boiten, Sergeant, & Geuze, 1992; Sergeant et al., 1999; van Winsum, Sergeant, & Geuze, 1984), and rhythmic theta waves associated with hippocampal activity can be observed (Pribram & McGuinness, 1975).

Effort is closely linked to arousal. Kahneman (1973) sometimes equated the two processes, but further research has suggested that they should be regarded as intimately related rather than inseparable (Pribram & McGuiness, 1975; Sanders, 1983). During an episode of physiological arousal, the heart rate surges (Obrist, Wood, & Perez-Reyes, 1965), arterial blood pressure increases (Abrahams & Hilton, 1964), and then the heart rate abruptly decelerates (Pribram & McGuinness, 1975). The eyes may dart back and forth briefly, as has been frequently noted in arousals associated with deceptive behavior (Ekman, 2001; Moore, Petrie, & Braga, 2003). Respiratory activity fluctuates, and electrodermal activity momentarily increases (Tursky, Shapiro, Crider, & Kahneman, 1969). Blood flow is altered (Abrahams & Hilton, 1964), and the amygdala becomes active (Pribram & McGuiness, 1975).

4. Analysis

Table 1 summarizes the physiological similiarities between tonic REM sleep and effort and between phasic REM sleep and arousal. Although it is currently challenging to make an exhaustive comparison because of the piecemeal and often imprecise nature of

Table 1

Physiological similarities between the different stages of REM sleep, effort, and arousal

Tonic REM/Effort	Phasic REM/Arousal
• Pupil dilation	• Heart rate acceleration
• Pupil constriction	• Increased arterial blood pressure
• Heart rate deceleration	• Heart rate deceleration
• EEG desynchronization	• Pupil dilation
• Rhythmic theta waves	• Rapid eye movement
• Intense activity in the hippocampus	• Pupil constriction
	• Respiratory fluctuations
	• Increased electrodermal activity
	• Altered blood flow
	• Intense activity in the amygdala

available data, the physiological similarities between the different stages of REM sleep, effort, and arousal are striking. Tonic REM appears to be the unconscious equivalent of effort, and phasic REM appears to involve the same kind of physiological arousal that occurs during wakefulness.

Given the many physiological similarities, it seems reasonable to suggest that REM sleep facilitates the "mobilization of energy," just as effort and arousal do. More specifically, the different stages of REM sleep, effort, and arousal probably increase the amount of energy available in the brain or some part of it. Such an idea is certainly supported by the fact that sleep, effort, and arousal have the same effect on a person: they elevate alertness. A person can improve his or her ability to focus on performing a task by getting a good night's sleep with adequate REM sleep or by exerting effort and becoming physiologically aroused. Any differences are merely a matter of degree.

The assertion that REM sleep replenishes depleted ATP reserves in the hippocampus and associated brain structures rest on four primary assumptions. First, the hippocampus becomes especially active during REM sleep (Lerma-Garcia-Austt, 1985; Rimbaud, Passouant, & Cadilhac, 1955). Second, the hippocampus requires ATP to function (e.g., Inoue, 1998; Inoue et al., 1999). Third, ATP in the brain comes almost exclusively from the metabolism of glucose (Peters et al., 2004). Fourth, glucose in the brain comes from two primary sources: cerebral glycogen and blood glucose (Benington & Heller, 1995).

A preliminary interpretation of the physiological features associated with REM sleep may now be offered. Preparations for replenishing depleted ATP reserves in the hippocampus appear to begin prior to the onset of REM sleep. It seems that a sequence of events ensures that an adequate amount of glucose will be available for significant ATP generation during REM sleep. Hormonal changes rhythmically increase blood glucose levels in the hours before nocturnal sleep in healthy young adults, and blood glucose levels remain elevated throughout sleep (Van Cauter, 2005). During NREM sleep, cerebral glycogen levels become elevated (Anchors & Burrows, 1983; Benington & Heller, 1995). “Glycogen represents the largest store of glucose equivalents in the brain” (Gruetter, 2003).

Tonic REM sleep begins with the complete immobilization of the body (Rama, Cho, & Kushida, 2006). Glycogen is metabolized, increasing the amount of glucose available in the brain (Rechtschaffen, 1998). Increased cerebral glycogen metabolism has also been observed in organisms exerting effort (Gruetter, 2003). However, this is “an unlikely end point of sleep’s functional role in brain energy homeostasis” (Franken, Gip,

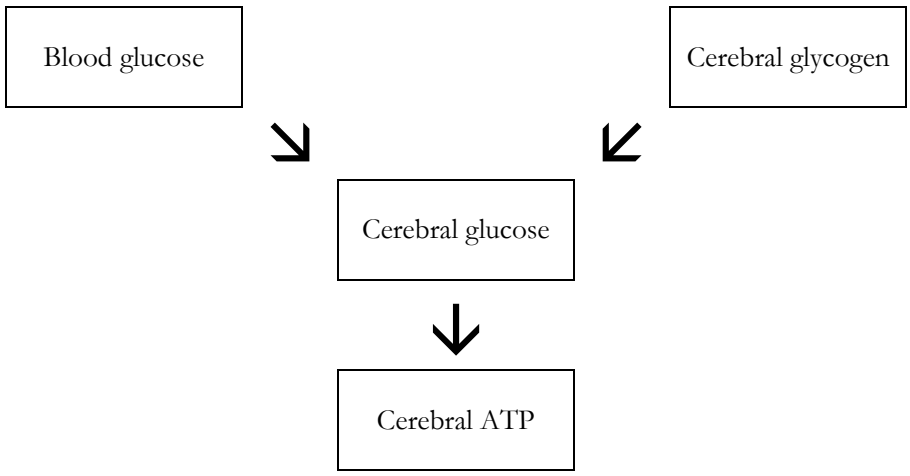
Hagiwara, Ruby, & Heller, 2003). Glucose still must be metabolized to generate ATP for the hippocampus. As this happens, brain temperature increases due to the intense metabolic activity (Heller, 2005). Homeostatic thermoregulation has been suspended (Orem & Keeling, 1980), perhaps to allow this increase in cerebral metabolic activity.

As ATP is generated, the hippocampus becomes active and rhythmic theta waves appear on the EEG. The pupils dilate (Rechtschaffen, 1998), perhaps indicating a change in hippocampal energy availability. Then, the pupils become constricted (Siegel, 2005), perhaps indicating that the hippocampus has more than enough energy to function. The EEG becomes desynchronized as it does during wakefulness. Dreams occur (Aserinsky & Kleitman, 1953). However, all of this brain activity is probably incidental. It likely occurs only because of the sudden availability of ATP, and it probably uses only a small percentage of the ATP that is generated. Thus, *most of the ATP generated during REM sleep is conserved for use during wakefulness*.

Phasic REM sleep likely occurs when additional cerebral glucose is required to meet the elevated metabolic requirements of REM sleep. The heart rate surges, arterial blood pressure increases, respiratory activity fluctuates, and there is a momentary increase in electrodermal activity. Blood glucose levels have already been elevated, and glucose is transported from the body to the brain via the bloodstream. Cerebral blood flow to the amygdala and parts of the pons, thalamus, cingulate cortex, and parietal operculum increases significantly (Maquet, 1995; Maquet et al., 1996). These areas and the hippocampal formation become significantly activated (Maquet, 2000). The pupils dilate maximally (Kemp & Kaada, 1975), perhaps indicating a massive increase in ATP

Figure 2

The restoration of depleted cerebral ATP reserves during REM sleep



Blood glucose levels are elevated prior to the onset of sleep and remain elevated throughout the sleep cycle. Cerebral glycogen levels are elevated during NREM sleep. Glycogen is metabolized to glucose during tonic REM sleep. Extra blood glucose is transported to brain during phasic REM sleep. Glucose is metabolized to generate ATP. The hippocampus requires ATP to function properly, and it becomes especially active during REM sleep.

levels in the hippocampus. The eyes move back and forth frantically (Aserinsky & Kleitman, 1953), perhaps as a result of the sudden energy surge. Then, the pupils become constricted and the heart rate slows again. It is probably fortunate that a person is unconscious and effectively immobilized during the energy surge of phasic REM sleep. People who are awakened from phasic REM sleep report quite bizarre mentation (Kushniruk, Rustenburg, & Ogilvie, 1985).

5. Conclusion

The details of the theory of REM sleep proposed here will undoubtedly require much clarification, and its potentially staggering implications for our understanding of

numerous medical conditions (e.g., type-II diabetes, Attention-Deficit/Hyperactivity Disorder, depression) and other biological phenomena (e.g., exercise, infant sleep, sleep deprivation) will have to be worked out thoroughly in the future. For the present, it only needs to be noted that this theory is testable. It should not be difficult to make an exhaustive comparison of the physiological features associated with tonic REM sleep, phasic REM sleep, effort, and arousal in human beings. The technology needed to monitor cerebral metabolism is now available, and it is also being rapidly improved.

I predict that two central tenets of this theory will withstand all future scrutiny. First, REM sleep involves the replenishment of a depleted ATP reserve somewhere in the brain. Present evidence suggests that the hippocampus is mostly likely the part of the brain that is re-energized during REM sleep. However, there are several other areas of the brain that may also be re-energized during this stage of the sleep cycle. Second, glucose is transferred from the body to the brain via the bloodstream during phasic REM sleep and during increases in physiological arousal that occur while a person is awake.

Many questions about sleep still remain to be answered, but it now seems clear that *the primary function of REM sleep is to re-energize the brain.*

6. References

Abrahams, V. C., & Hilton, S. M. (1964). The Role of Active Muscle Vasodilatation in the Alerting Stage of the Defence Reaction. *J Physiol*, 171, 189-202.

Anchors, J. M., & Burrows, B. L. (1983). Changes in brain glycogen during slow-wave sleep in the rat. *J Neurochem*, 41(5), 1498-1501.

Aserinsky, E. (1965). Periodic respiratory pattern occurring in conjunction with eye movements during sleep. *Science*, 150(697), 763-766.

Aserinsky, E., & Kleitman, N. (1953). Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. *Science*, 118(3062), 273-274.

Benington, J. H., & Heller, H. C. (1995). Restoration of brain energy metabolism as the function of sleep. *Prog Neurobiol*, 45(4), 347-360.

Boiten, F., Sergeant, J., & Geuze, R. (1992). Event-related desynchronization: the effects of energetic and computational demands. *Electroencephalogr Clin Neurophysiol*, 82(4), 302-309.

Broughton, R. J., Poire, R., & Tassinari, C. A. (1965). The Electrodermogram (Tarchanoff Effect) During Sleep. *Electroencephalogr Clin Neurophysiol*, 18, 691-708.

Carskadon, M. A., & Dement, W. C. (2005). Normal Human Sleep Overview. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (4th ed., pp. 13-23). Philadelphia, PA: Elsevier/Saunders.

Coles, M. G. (1972). Cardiac and respiratory activity during visual search. *J Exp Psychol*, 96(2), 371-379.

Ekman, P. (2001). *Telling lies : clues to deceit in the marketplace, politics, and marriage* (3rd ed.). New York: W.W. Norton.

Frank, M. G. (2006). The Function of Sleep. In T. L. Lee-Chiong (Ed.), *Sleep: A comprehensive handbook* (pp. 45-48). Hoboken, N.J.: Wiley.

Franken, P., Gip, P., Hagiwara, G., Ruby, N. F., & Heller, H. C. (2003). Changes in brain glycogen after sleep deprivation vary with genotype. *Am J Physiol Regul Integr Comp Physiol*, 285(2), R413-419.

Franzini, C. (2005). Cardiovascular Physiology: The Peripheral Circulation. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (4th ed., pp. 203-212). Philadelphia, PA: Elsevier/Saunders.

Franzini, C., Zoccoli, G., Cianci, T., & Lenzi, P. (1996). Sleep-Dependent Changes in Regional Circulations. *News Physiol. Sci.*, 11, 274-280.

Granholm, E., Asarnow, R. F., Sarkin, A. J., & Dykes, K. L. (1996). Pupillary responses index cognitive resource limitations. *Psychophysiology*, 33(4), 457-461.

Green, J. D., & Arduini, A. A. (1954). Hippocampal electrical activity in arousal. *J Neurophysiol*, 17(6), 533-557.

Gruetter, R. (2003). Glycogen: the forgotten cerebral energy store. *J Neurosci Res*, 74(2), 179-183.

Heller, H. C. (2005). Temperature, Thermoregulation, and Sleep. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (4th ed., pp. 292-304). Philadelphia, PA: Elsevier/Saunders.

Herman, D. M., & Bassetti, C. L. (2002). Cerebral Circulation and Sleep. *Sleep Med Rev*, 6(6), 425-427.

Hess, E. H. (1965). Attitude and Pupil Size. *Sci Am*, 212, 46-54.

Hess, E. H., & Polt, J. M. (1964). Pupil Size in Relation to Mental Activity during Simple Problem-Solving. *Science*, 140, 1190-1192.

Inoue, K. (1998). ATP receptors for the protection of hippocampal functions. *Jpn J Pharmacol*, 78(4), 405-410.

Inoue, K., Koizumi, S., Ueno, S., Kita, A., & Tsuda, M. (1999). The functions of ATP receptors in the synaptic transmission in the hippocampus. *Prog Brain Res*, 120, 193-206.

Jung, R., & Kornmueller, A. E. (1938). Eine methodik der Abkitung lokalisierter Potentialschwankungen aus subcorticalen Hirngebieten. *Arch für Psychiatrie und Nervenkrankheiten*, 109, 1-30.

Kagan, J. (1965). Studies of Attention in the Human Infant. *Merrill-Palmer Quarterly*, 11, 95-122.

Kahneman, D. (1973). *Attention and effort*. Englewood Cliffs, N.J.,: Prentice-Hall.

Kemp, I. R., & Kaada, B. R. (1975). The relation of hippocampal theta activity to arousal, attentive behaviour and somato-motor movements in unrestrained cats. *Brain Res*, 95(2-3), 323-342.

Kramer, A., & Spinks, J. (1991). Capacity views of human information processing. In J. R. Jennings & M. G. H. Coles (Eds.), *Handbook of cognitive psychophysiology: Central and autonomic nervous system approaches* (pp. 179-250). New York: John Wiley & Sons.

Kushniruk, A., Rustenburg, J., & Ogilvie, R. (1985). Psychological correlates of electrodermal activity during REM sleep. *Sleep*, 8(2), 146-154.

Lerma, J., & Garcia-Austt, E. (1985). Hippocampal theta rhythm during paradoxical sleep. Effects of afferent stimuli and phase relationships with phasic events. *Electroencephalogr Clin Neurophysiol*, 60(1), 46-54.

Lewis, M., Kagan, J., Campbell, H., & Kalafat, J. (1966). Child Development. *Child Development*, 37, 63-71.

Lohr, I. (1999). The influence of effort on impairments of attention associated with major affective disorders. Parkland, FL: Universal Publishers.

Maquet, P. (1995). Sleep function(s) and cerebral metabolism. *Behav Brain Res*, 69(1-2), 75-83.

Maquet, P. (2000). Functional neuroimaging of normal human sleep by positron emission tomography. *J Sleep Res*, 9(3), 207-231.

Maquet, P., Peters, J., Aerts, J., Delfiore, G., Degueldre, C., Luxen, A., et al. (1996). Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. *Nature*, 383(6596), 163-166.

Miller, G. A. (1956). The magical number seven plus or minus two: some limits on our capacity for processing information. *Psychol Rev*, 63(2), 81-97.

Moore, M. H., Petrie, C. V., & Braga, A. A. (2003). *The polygraph and lie detection*. Washington, D.C.: National Academies Press.

Obrist, P. A., Wood, D. M., & Perez-Reyes, M. (1965). Heart Rate During Conditioning in Humans: Effects of Ucs Intensity, Vagal Blockade, and Adrenergic Block of Vasomotor Activity. *J Exp Psychol*, 70, 32-42.

Orem, J., & Keeling, J. (1980). A Compendium of Physiology in Sleep. In J. Orem & C. D. Barnes (Eds.), *Physiology in Sleep* (pp. 315-336). New York: Academic Press.

Peters, A., Schweiger, U., Pellerin, L., Hubold, C., Oltmanns, K. M., Conrad, M., et al. (2004). The selfish brain: competition for energy resources. *Neurosci Biobehav Rev*, 28(2), 143-180.

Pribram, K. H., & McGuinness, D. (1975). Arousal, activation, and effort in the control of attention. *Psychol Rev*, 82(2), 116-149.

Rama, A. N., Cho, S. C., & Kushida, C. A. (2006). Normal Human Sleep. In T. L. Lee-Chiong (Ed.), *Sleep: A comprehensive handbook*. Hoboken, N.J.: Wiley.

Rechtschaffen, A. (1998). Current perspectives on the function of sleep. *Perspect Biol Med*, 41(3), 359-390.

Rimbaud, L., Passouant, P., & Cadilhac, J. (1955). Participation de l'hippocampe à la régulation des états de veille et de sommeil. *Rev Neurol* (Paris), 93(1), 303-308.

Rowe, K., Moreno, R., Lau, T. R., Wallooppillai, U., Nearing, B. D., Kocsis, B., et al. (1999). Heart rate surges during REM sleep are associated with theta rhythm and PGO activity in cats. *Am J Physiol*, 277(3 Pt 2), R843-849.

Sakai, K., Sano, K., & Iwahara, S. (1973). Eye movements and hipocampal theta activity in cats. *Electroencephalogr Clin Neurophysiol*, 34(5), 547-549.

Sanders, A. F. (1983). Towards a model of stress and human performance. *Acta Psychol* (Amst), 53(1), 61-97.

Sei, H., & Morita, Y. (1996). Acceleration of EEG theta wave precedes the phasic surge of arterial pressure during REM sleep in the rat. *Neuroreport*, 7(18), 3059-3062.

Sergeant, J. A., Oosterlaan, J., & van der Meere, J. (1999). Information processing and energetic factors in Attention-Deficit/Hyperactivity Disorder. In H. C. Quay & A.

E. Hogan (Eds.), *Handbook of disruptive behavior disorders* (pp. 75-104). New York: Plenum Publishers.

Siegel, J. M. (2005). REM Sleep. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (4th ed., pp. 120-134). Philadelphia, PA: Elsevier/Saunders.

Sullivan, C. E. (1980). Breathing in Sleep. In J. Orem & C. D. Barnes (Eds.), *Physiology in sleep* (pp. 212-271). New York: Academic Press.

Taylor, W. B., Moldofsky, H., & Furedy, J. J. (1985). Heart rate deceleration in REM sleep: an orienting reaction interpretation. *Psychophysiology*, 22(1), 110-115.

Tursky, B., Shapiro, D., Crider, A., & Kahneman, D. (1969). Pupillary, heart rate, and skin resistance changes during a mental task. *J Exp Psychol*, 79(1), 164-167.

Van Cauter, E., & Spiegel, K. (1999). Sleep as a mediator of the relationship between socioeconomic status and health: a hypothesis. *Ann NY Acad Sci*, 896, 254-261.

Van Winsum, W., Sergeant, J., & Geuze, R. (1984). The functional significance of event-related desynchronization of alpha rhythm in attentional and activating tasks. *Electroencephalogr Clin Neurophysiol*, 58(6), 519-524.

Verrier, R. L., Harper, R. M., & Hobson, J. A. (2005). Cardiovascular Physiology: Central and Autonomic Regulation. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (4th ed., pp. 192-202). Philadelphia, PA: Elsevier/Saunders.

Verrier, R. L., Lau, T. R., Wallooppillai, U., Quattrochi, J., Nearing, B. D., Moreno, R., et al. (1998). Primary vagally mediated decelerations in heart rate during tonic rapid eye movement sleep in cats. *Am J Physiol*, 274(4 Pt 2), R1136-1141.

Walter, V. J., & Walter, W. G. (1953). The central effects of rhythmic sensory stimulation. *Electroencephalogr Clin Neurophysiol*, 35, 215-217.

Winson, J. (1975). The Theta Mode of Hippocampal Function. In R. L. Isaacson & K. H. Pribram (Eds.), *The Hippocampus* (Vol. 2, pp. 169-184). New York: Plenum Press.

Zepelin, H., Siegel, J. M., & Tobler, I. (2005). Mammalian Sleep. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (4th ed., pp. 91-100). Philadelphia, PA: Elsevier/Saunders.