

WE ARE CHRONICALLY SLEEP DEPRIVED*

Michael H. Bonnet and Donna L. Arand

Dayton Veterans Affairs Medical Center, Wright State University, and Kettering
Medical Center

* This work was supported by a Merit Review Grant from the Department of
Veterans Affairs.

Running Head: Chronic Sleep Deprivation

Correspondence to:

Michael H. Bonnet, Ph.D. (151N)

VA Hospital

4100 W. Third Street

Dayton, OH 45428

(513) 267-3910

FAX (513) 267-3983

Abstract

Data from recent laboratory studies indicate that nocturnal sleep periods reduced

by as little as 1.3 to 1.5 hours for one night result in reduction of daytime alertness by as much as 32% as measured by the MSLT. Other data document that a) 17-57% of normal young adults have MSLT latencies of 5.5 min or less while 50% or fewer have MSLT values of 10 min or more; and b) 28-29% of young adults reported normally sleeping 6.5 hours or less on each week night. More extensive reduction of daily sleep amount is seen in night-shift workers. A minimum of 2-4% of middle age adults have hypersomnolence associated with sleep apnea. Together, these data show that significant sleep loss exists in 1/3 or more of normal adults, that the effects are large and replicable, and that similar effects can be produced in just one night in the laboratory. In light of the magnitude of this sleep debt, it is not surprising that fatigue is a factor in 57% of accidents leading to the death of a truck driver and in 10% of fatal car accidents and results in costs of up to 56 billion dollars per year. A recent sleep extension study suggests that the average underlying sleep tendency in young adults is about 8.5 hours per night. By comparison, the average reported sleep length of 7.2-7.4 hours is deficient and common sleep lengths of 6.5 hours or less can be disastrous. We must recognize the alertness function of sleep and the increasing consequences of sleepiness with the same vigor that we have come to recognize the societal impact of alcohol.

Keywords: Sleep Deprivation, Sleepiness, Sleep Disorders, Work Schedule Tolerance.

Introduction

We are in the midst of a golden age of discovery of the intricate interrelationship between our nocturnal sleep process and our level of daytime function. Fueled by the discovery of the tremendous incidence of sleep apnea and periodic leg movements in the population, the relationship between fragmented sleep and residual sleepiness, and the increased ability to measure the level of objective sleepiness with an objective test (the Multiple Sleep Latency Test) and in ambulatory environments, the pervasive role of excessive sleepiness in our society is becoming apparent.

A very large literature documents the negative effects of sleep deprivation on a wide range of psychomotor performance tasks and mood variables. For the sake of simplicity, the primary outcome measure reported in this paper will be MSLT. However, the MSLT findings reported are consistent with similar changes in a broad range of abilities including reaction time, short term memory, vigilance, and mood (1).

Many empirical and applied studies have provided evidence for our national sleep debt. The degree of our weekly sleep loss can be measured by how long we continue to sleep when our alarm does not go off either in the famous Webb and Agnew (2) paradigm or in our own bedroom on Saturday morning. The degree of this debt can also be measured in the propensity to fall asleep rapidly.

Much sleep loss is the product of reduced and/or fragmented nocturnal sleep. For example, in one study in which total nocturnal sleep was reduced from 7.1 to 5.8 hours per night for 1 week, a significant decrease in MSLT (from 15.5 to 12.6 min) was found after the first night (3). In a study of time in bed reduced to 6 hours (total sleep 336 min), subjects had a 32% reduction in MSLT compared with an 8 hour sleep period (total sleep 430 min) (4) after one night. The actual MSLT values, which differed significantly, were 12.4 and 8.4 min, and it should be noted that subjects chosen for this study were prescreened on the MSLT to have values greater than 8 min. Thus two independent studies have found that when total sleep

times in normal young adults were reduced 1-1.5 hours for one night, decreases of up to 1/3 were found on objective alertness. The data suggest that the normal young adults in these two studies were unusually alert compared to their peers, partially based on subject selection criteria. In a study which involved over 100 young adults screened for normal sleep habits, lack of shiftwork or sleep pathology, and infrequent naps, 32% were found to have daytime nap latency values of 5 min or less, and only 40% had nap latency values of greater than 10 min (data from 5). Data from a large study by Levine et al (6) indicated that 17% of their sample of normal young adults had average MSLT values of 5 min or less and only 50% had average MSLT latencies of 10 min or greater. In a study of 10th grade students who had a 7:20 school start time, 57% had MSLT latencies less than 5.5 min (7). As has been noted, MSLT values in the pathological range are uncomfortably common in groups of normal young adults (8), and have been systematically studied by Roehrs et al (9; 10). These important papers have concluded that short MSLT values in normal young adults are most likely a function of chronic partial sleep deprivation and not an indication of simple ability to fall asleep because increasing nocturnal sleep consistently increased the following MSLT latencies.

Partial sleep loss is a common condition in many segments of our society. A review of 50 random young adult normal sleeping applicants to participate in sleep studies revealed an average nocturnal sleep length of 7.4 hours (s.d. 1.3). This value is in close agreement with other questionnaire studies (11; 12). However, in the group, 28% reported normally sleeping 6.5 hours or less each week night (as compared with 29% reported by Browman et al (13)). As would be expected, these young adults with short sleep lengths attempted to deal with their chronic sleep loss by increasing their sleep length on the weekend (80% of weekend sleep changes were increases, binomial probability = .053). In contrast, only 4 applicants reported sleeping 9 hours or more on week nights, and all four of those individuals reported decreased sleep length on the weekend. These data indicate that 1) almost 1/3 of normal young adults sleep 6.5 hours or less each night; 2) almost 1/3 of normal young adults had laboratory sleep latencies of 5 min or less; and 3) significant

reductions of up to 32% on MSLT have been shown after one night in two lab studies in which total sleep was almost 6 hours. Together these results show that significant sleep loss is common in normal young adults, that the effects are large and replicable, and that these effects can be duplicated rapidly in the laboratory.

As these young adults graduate from college and assume the responsibilities of full-time jobs and families, pressure to sacrifice sleep only grows. In addition, with age, the incidence of fragmenting sleep disorders begins to increase. For example, 24% of a random sample of 65 year olds had sleep apnea and 45% had periodic leg movements (14; 15). Thirty-nine percent of this older population reported problems with excessive daytime sleepiness at least once per week (14). A recent study of employed men and women age 30-60 found that 24% of the men and 9% of the women had sleep apnea (16). A smaller group, 2% of women and 4% of men were judged to have both sleep apnea and daytime hypersomnolence based on subjective reports, but objective laboratory measurements would probably indicate much higher levels of sleepiness. A minority of patients with major depression (10-15%) and a group of patients with bipolar and seasonal affective disorder show hypersomnia and excessive daytime sleepiness. Based upon the prevalence of these disorders, these patients probably account for 1-2% of the population (17; 18).

Reduced and disturbed sleep has equally serious consequences for shift workers. Eighty-two percent of oil refinery shift workers stated that they suffered from lack of sleep (19). About 3.5% of workers in the United States work midnight shifts (20). Workers permanently assigned to the graveyard shift sleep on the average only 5.8-6.4 hours per day (21; 22; 23) when working that shift while rotating shift workers sleep only 5.25-5.5 hours per day when working the night shift (21; 24). Because this sleep also occurs at a poor circadian time, further decrease in alertness and performance would be expected (25). Regardless, current laboratory studies indicate that sleep lengths in the 5-6 hour range will result in the accumulation of significant sleepiness (3, 4). Many important segments of society including doctors, nurses, police, firemen, nuclear power plant operators, and cross country truck drivers are graveyard shiftworkers and are chronically sleep deprived

when they are working at the nadir of their circadian rhythms. For example, a review of 10 studies of physician sleep in hospitals during “on-call” nights found that these doctors slept an average of 2.8 hours on these nights (1). Eight of the studies found significant decrements on at least one task after this night of reduced sleep, as would be expected. Because the data indicate that alertness deficits begin to develop after single sleep periods of almost 6 hours in length, it is clear that rotating and many permanent night shift workers work and drive home under the pressure of a significant sleep debt. It is therefore not surprising that sleepiness plays a prominent role in driving fatalities. For fatal car accidents, the three most common attributions as determined by Traffic Safety Committees (26) are alcohol (18%), poor attention (15%), and sleepiness (10%). It is well known that all three of these factors interact.

In a group of train drivers, 70% reported that they had dozed off while driving a train. When the EEG of the drivers was actually recorded while driving, 36% reported falling asleep and one actually failed to act on train stop signals while his EEG indicated drowsiness (27). These examples from laboratory studies can be compared with statistics from the National Transportation Safety Board, which reports that fatigue was a factor in 57% of accidents leading to the death of a truck driver (28). When one considers that there are about 4800 fatal accidents involving trucks each year and that the cost associated with each fatal truck crash was at least \$2,700,000, it can be seen that the costs of sleepiness in society are enormous. In fact, recent estimates indicate that the cost of only sleep-related accidents in the United States ranges from 2 to 56 billion dollars each year (29; 30). These costs do not include lost productivity, medical illness, or shortened life span secondary to sleepiness or sleep pathology.

It has been argued that the actual sleep requirement may be less than the total amount of sleep that can be accumulated during a 24-hour period (31). Evidence exists that normal young adults can sleep for 14.4 hours in a 24-hour period (32). While some of this sleep may be “excess”, there is currently no way to determine which sleep may be excess and which may be required. In a study designed to

mimic the 14-hour “night” of the winter dark period, Wehr et al (33) found that young adults who had slept for about 7.2 hours on baseline nights slept consistently more for 28 days. Subjects slept in excess of 10 hours for three nights, probably as recovery from chronic partial sleep loss, and about 9.1 hours for the next 4 nights. Over the next three weeks, average total sleep was 8.8, 8.9, and 8.4 hours. This implies that the actual sleep need for these 7.2 hour sleepers was about 8.5 hours. Subjects did not report negative consequences of their “excess” sleep. In fact, they reported being significantly more energetic and happier with less fatigue. Once again, these results probably indicate the degree of chronic partial sleep loss existing in most normal adults and the significant benefit of normalizing that sleep amount. The difference between 7.2 and 8.5 hours implies a chronic sleep loss of over an hour per night. Further, only about 14% of young adults applying to participate in sleep studies reported weekday sleep lengths of 8.5 hours or more, and this also implies that chronic sleep loss is the norm and not the exception.

Perhaps a more profitable way of viewing sleep is as a logarithmic process where large benefits accrue in initial hours but increasingly small benefits continue to accrue as the length of the sleep period increases. Such a view would indicate that some loss of function would exist in any non-naturally terminated sleep period but that such deficits might be small after long sleep periods. In fact, other studies of increasing time in bed have shown that an increase of time in bed from 8 to 10 hours will lengthen the MSLT by 1-5 min (8; 9) and improve reaction time significantly (9). It is true that we exist in a society where we may chose to starve or become obese; to become impaired by alcohol; or to become impaired by sleep deprivation. We are normally guided by standards of behavior: normal weight/height tables as a standard of eating and blood alcohol levels as a standard of sobriety. In sleepiness, the only real standard is the MSLT, and values in excess of 10 min. are typically considered “normal.” Having no expressible propensity to fall asleep is not abnormal, just as having no measurable alcohol in the blood is not abnormal. On the contrary, it is important to stress that unnaturally terminated or shortened sleep is the potentially pathological process which violates the standards of alertness. There is strong

evidence that sufficient shortening or disturbance of the sleep process compromises mood, performance, and alertness which can result in injury or death. In this light, the most common sense “do no injury” medical advice would be to avoid sleep deprivation. The best means of achieving this goal is to determine an individual sleep need; i.e., going to bed in the evening when sleepy and awakening in the morning without an alarm and then observing the total time as the sleep period time.

To posit that any sleep length less than the empirically defined sleep need is sufficient, one must first show that there is no reduction in any function with a lower sleep quota in large groups of subjects and that decrements do not appear under chronic conditions. Finally, one must bear the responsibility for fatigue-related incidents following reduced-sleep regimens. Because studies have already shown significantly improved performance and alertness when time in bed is increased from 8 to 10 hours per night (8; 9), we are certainly not ready to accept either the assertion that reduced sleep of any degree is benign or the responsibility when such benign sleep loss turns out to have hidden consequences. As our tests become more sensitive, the degree of sleep loss required to produce deficits has continued to decrease, yet many forces in our society continue to operate with the philosophy that sleep loss is unimportant or can be overcome by force of will. The idea that sleep loss is unimportant has resulted in tremendous personal and societal costs and is a position that we should have gained the wisdom to have outgrown.

Questionnaire studies of the prevalence of sleepiness in our society report widely divergent estimates from 5 - 36% of the total population based upon individual definitions of sleepiness (34). The data reviewed in this paper suggest that up to 1/3 of young adults may be excessively sleepy secondary to chronic partial sleep deprivation; perhaps 7% of middle age individuals are excessively sleepy secondary to sleep disorders; and another 2% are sleepy secondary to shift work. Considered together, these numbers certainly support the estimate that 36% or more of the population are suffering from sleep loss (34). One must also realize that it is common for people who are sleepy to deny that they are sleepy. How many of us feel like “just resting our eyes” for a few moments right now? Considering the

magnitude of the problem of sleepiness, the best course of action must be to encourage all of our contemporaries to “sleep on it” a bit more.

References

1. Bonnet MH. Sleep deprivation. In: Kryger, M, Roth, T, Dement, WC, ed. *Principles and practice of sleep medicine*. 2nd. Ed. Philadelphia: Saunders, 1994:50-68.
2. Webb WB, Agnew HW. Are we chronically sleep deprived? *Bulletin of the Psychonomic Society* 1975;6:47-48.
3. Bonnet MH, Arand DL. The consequences of a week of insomnia. *Sleep Res* 1995;24:201.
4. Rosenthal L, Roehrs TA, Rosen A, Roth T. Level of sleepiness and total sleep time following various time in bed conditions. *Sleep* 1993;16:226-32.
5. Bonnet MH. The effect of varying prophylactic naps on performance, alertness and mood throughout a 52-hour continuous operation. *Sleep* 1991;14:307-15.
6. Levine B, Roehrs T, Zorick F, Roth T. Daytime sleepiness in young adults. *Sleep* 1988;11:39-46.
7. Carskadon MA, Wolfson AR, Tzischinsky O, Acebo C. Early school schedules modify adolescent sleepiness. *Sleep Res*. 1995;24:92.
8. Carskadon MA, Dement WC. Nocturnal determinants of daytime sleepiness. *Sleep* 1982;5 Suppl 2:S73-81:
9. Roehrs T, Timms V, Zwyghuizen-Doorenbos A, Roth T. Sleep extension in sleepy and alert normals. *Sleep* 1989;12:449-57.
10. Roehrs T, Timms V, Zwyghuizen-Doorenbos A, Buzenski R, Roth T. Polysomnographic, performance, and personality differences of sleepy and alert normals. *Sleep* 1990;13:395-402.
11. Webb WB. *Sleep the Gentle Tyrant* 2nd. Bolton, MA: Anker, 1992. 1-179.
12. White RM. *Sleep length and variability: Measurement and interrelationships* (Unpublished Dissertation: University of Florida, 1975).
13. Browman CP, Gordon GC, Tepas DI, Walsh JK. Reported sleep and drug use of workers: a preliminary report. *Sleep Res*. 1977;6:111.
14. Ancoli-Israel S, Kripke DF, Klauber MR, Mason WJ, Fell R, Kaplan O. Sleep-disordered breathing in community-dwelling elderly. *Sleep* 1991;14:486-95.

15. Ancoli-Israel S, Kripke DF, Klauber MR, Mason WJ, Fell R, Kaplan O. Periodic limb movements in community-dwelling elderly. *Sleep* 1991;14:496-500.
16. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. Occurrence of sleep disordered breathing among middle-aged adults. *NEJM* 1993;328:1230-35.
17. Benca RM. Mood Disorders. In: Kryger, MH, Roth, T, Dement, WC, ed. *Principles and Practice of Sleep Medicine*. 2nd Ed. Philadelphia: W.B. Saunders, 1994:899-913.
18. Reynolds CF. Sleep in affective disorders. In: Kryger, MH, Roth, T, Dement, WC, ed. *Principles and Practice of Sleep Medicine*. Philadelphia: W.B. Saunders, 1989:413-415.
19. Kogi K. Social aspects of shift work in Japan. *Int. Labour Rev* 1971;104:414-33.
20. Webb WB. Work/rest schedules: economic, health, and social implications. In: Johnson, LC, Tepas, DI, Colquhoun, WP, Colligan, MJ, ed. *The Twenty-four Hour Workday: Proceedings of a Symposium on Variations in Work-sleep Schedules*. DHHS (NIOSH) Publication No. 81-127, 1981:1-12.
21. Walsh JK, Tepas DI, Moss PD. The EEG sleep of night and rotating shift workers. In: Johnson, LC, Tepas, DI, Colquhoun, WP, Colligan, MJ, ed. *The Twenty-four Hour Workday: Proceedings of a Symposium on Variations in Work-sleep Schedules*. DHHS (NIOSH) Publication No. 81-127, 1981:451-465.
22. Tasto DL, Colligan MJ, Skjei EW, Polly SJ. *Health consequences of shift work* (NIOSH Document #78-154, 1978).
23. Tepas DI. Adaptation to shiftwork: fact or fallacy. *J Human Ergol* 1982;11:1-12.
24. Tepas DI, Walsh JK, Armstrong DR. Comprehensive study of the sleep of shift workers. In: Johnson, LC, Tepas, DI, Colquhoun, WP, Colligan, MJ, ed. *The Twenty-four Hour Workday: Proceedings of a Symposium on Variations in Work-sleep Schedules*. DHHS (NIOSH) Publication No. 81-127, 1981:419-34.
25. Bonnet MH, Arand DL. Metabolic rate and the restorative function of sleep.

Sleep Res. 1995;24:88.

26. Summala H, Mikkola T. Fatal accidents among car and truck drivers: Effects of fatigue, age, and alcohol consumption. *Hum. Factors.* 1994;36:315-26.

27. Torsvall L, Akerstedt T. Sleepiness on the job: continuously measured EEG changes in train drivers. *Electroencephalogr. Clin. Neurophysiol.* 1987;66:502-11.

28. Mitler MM, Dinges DF, Dement WC. Sleep medicine, public policy, and public health. In: Kryger, MH, Roth, T, Dement, WC, ed. *Principles and Practice of Sleep Medicine.* 2nd Philadelphia: W.B. Saunders, 1994:453-462.

29. Leger H. The cost of sleep related accidents: a report for the National Commission on Sleep Disorders Research. *Sleep* 1994;17:84-93.

30. Webb WB. The cost of sleep-related accidents: A reanalysis. *Sleep* 1995;18:276-80.

31. Horne J. *Why We Sleep* New York: Oxford University Press, 1987. 1-319.

32. Aserinsky E. The maximal capacity for sleep: Rapid eye movement density as an index of sleep satiety. *Biological Psychiatry* 1969;1:147-159.

33. Wehr TA, Moul DE, Barbato G, Giesen HA, Seidel JA, Barker C, Bender C. Conservation of photoperiod-responsive mechanisms in humans. *Am J Physiol* 1993;265:

34. Partinen M. Epidemiology of sleep disorders. In: Kryger, MH, Roth, T, Dement, WC, ed. *Principles and Practice of Sleep Medicine.* 2nd Philadelphia: W.B. Saunders, 1994:437-52.