

**REVIEW ARTICLE**

**SLEEP AND PERFORMANCE – RECENT TRENDS**

**GIDUGU HIMASHREE\*, P. K. BANERJEE  
AND W. SELVAMURTHY**

*Defence Institute of Physiology and Allied Sciences,  
Lucknow Road, Timarpur,  
Delhi – 110 054*

( Received on July 26, 2001 )

**Abstract :** Sleep and sleep deprivation are intimately related to performance. Sleep management of people working in different sectors of the society like multi shift workers, nurses, doctors, students in professional schools and the armed forces has a great bearing on performance, health and safety of the subject population. The detrimental effects of sleep deprivation on psychological performance are indicated as increased lapsing, cognitive slowing, memory impairment, decrease in vigilance and sustained attention and shift in optimum response capability. Its effects on physical performance are manifested as decline in ability to perform maximal exercise, self-selected walking pace and increase in perceived exertion. Sleep deprivation appears to have no effect in respect of muscle contractile properties and maximum anaerobic power. At high altitude (HA), there is a reduction in NREM sleep with frequent awakening due to hypoxia as a physiological adaptive measure to prevent accentuation of hypoxemia due to sleep-hypoventilation. Total sleep deprivation for 48 hours at high altitude can affect the acclimatization status, thermoregulation efficiency and cognitive functions. The concept of 'sleepiness' has also been studied, as it is an emerging concept for better understanding of the effects of sleep deprivation and its effects on performance. A special mention of sustained operations in the armed forces has been made keeping in mind its uniqueness in challenging the normal sleep-work schedule and its deployment in extreme environment and operational condition. This article reviews in detail the functions of sleep, its requirement and the effects of sleep deprivation on human performance.

**Key words :** sleep      sleep deprivation      psychological performance  
sleepiness      physical performance      sustained operations

**INTRODUCTION**

Sleep has a significant role on the well being and performance of humans. Clinical

studies of sleep disorders like sleepiness and insomnia are well known. Also, sleep or lack of it has a lot to do with many occupations and professions demanding long or abnormal

---

\*Corresponding Author

waking hours or in particular groups of population who require to keep awake for more hours than others e.g. multi shift workers, students in professional schools and in armed forces during operations. The effect of altered sleep quantity and quality has a significant bearing on the psychological and physiological profile of an individual. Sleep and sleepiness are necessary deterrents in man's life today where his occupational demands are higher than ever before in terms of physical performance and mental alertness. A better understanding of sleep, sleepiness and performance would be tremendously useful to the society, both in ameliorating the clinical burden, and in improving human performance, safety, well being and productivity.

Numerous studies have been done and many more are on to evaluate the intricate relationship of sleep, sleepiness and performance (1-4) and also the ways and means of reducing the deleterious effects of sleepiness (5, 6). Efforts are also being made to incorporate sleep management as an integral part of functioning of sectors with irregular work schedule. An understanding into the minimum requirement of sleep for a human being without compromising on his performance efficiency is also being worked out (7-16). An attempt to review the intimate relationship between sleep and performance has been made with reference to studies in these fields in the last eight decades or so. A special mention is made to the military operations and their peculiarity of demands in terms of sleep deprivation and physical performance.

### Theories of sleep

The earliest thinking about sleep, which occurred to man, was a "retreat" of the spirit from the body, which would enable all the spirits to be in communion. In this state, the body was considered highly vulnerable to evil motives of spirits. Some thought that sleep is for nourishing the body. As logical thinking prevailed, this theory of retreat gave in to the 'Deafferentation theory' in which the opinion was that when there was no input to the brain it fell asleep (17, 18). O'Leary and Goldring (1976) designed a model in which sleep was interpreted as a state of unresponsiveness and turning off of 'wakefulness' (19). Rapid Eye Movement (REM) in sleep was first noted by Greissinger (20) in 1868 and further studied by Aserinsky and Kleitman in 1955 (21, 22). In 1920 Mac William studied the physiological changes in human being during sleep (23). von Economo capitalized on the pandemic of viral encephalitis in 1918-20 and pointed out the role of hypothalamus in wakefulness and sleep (24) which was later confirmed by Brenner in 1974 (25).

The advent of EEG by Hans Berger (1929) who demonstrated spontaneous oscillations of the electrical potentials of the brain (26), which was another important milestone. In 1949, Moruzzi et al highlighted the importance of Reticular Activating system (RAS) in keeping the animal awake by a constant volley of impulses to the cerebral cortex (27) and loss of this facilitation led to somnolence. This offered support to the 'Deafferentation theory'. However, in 1937, Loomis et al with

the help of EEG studies described the various stages of sleep and concluded that sleep is not mere deafferentation but is a complicated and integrated physiological event (1). Age and sex can significantly influence sleep and performance (28, 29). Drugs like Amphetamine, Methylphenidate and Pemoline decrease sleep and improve performance (30).

Currently the two acceptable theories are the restorative theory and the ethological/conservative theory. Fortunately these two are not mutually exclusive or contradictory. Conservative theory also encompasses the teleology of sleep. Sleep is supposed to have a protective value when it originated in life forms. Probably, in recent times sleep is considered more of an acquired habit down the evolution than a physiological necessity. The restorative theory has been supported and lauded by a number of workers (31-34).

#### Stages of sleep

The sleep scoring is done in many ways but the gold standard is that devised by Rechtschaffen and Kales based on EEG, EMG and EOG (35). Human sleep progresses through different stages very well characterized by the sleep scoring systems (36). Sleep is heralded by the appearance of diminution of the alpha rhythm which is the onset of Stage I non-REM (NREM) sleep. Slow rolling eye movements and a reduction in muscle tone are also salient features of this stage. This stage lasts for a few minutes. The appearance of sleep spindles marks the beginning of stage II of sleep. There are no eye movements and there is further decrease in muscle tone. EEG shows

few (20%) delta and theta waves. This stage lasts for 30 to 60 minutes. Sleep next progresses to the stages of slow wave sleep (Stage III and Stage IV NREM sleep). The delta waves constitute 20% to 50% in stage III and more than 50% in stage IV. These stages are also called deep sleep or delta sleep. This sleep is briefly interrupted by stage II NREM sleep that is followed by first REM sleep of 60-90 minutes after sleep onset. REM sleep is characterized by rapid eye movements, desynchronisation of EEG and marked reduction or absence of muscle tone. The first REM period lasts for only a few minutes and is followed by progression to stage II and then to stage III and IV of NREM sleep before the appearance of the second REM sleep. This constitutes of a full sleep cycle and lasts for 90-110 minutes. Each sleep has about 4 to 6 such cycles with increasing duration of the REM sleep, the last lasting up to 60 minutes.

#### Regulation of sleep

A symphony of carefully orchestrated neurotransmitters in the brain especially the brain stem, the complete understanding of REM regulation neurochemistry is much better understood than NREM. Most of the effector neurons are in the reticular formation, particularly the pontine reticular formation. These neurons are under the influence of excitatory and inhibitory neurotransmitters. Acetylcholine and Histamine are predominantly excitatory and Serotonin, Norepinephrine, Dopamine and Glycine are inhibitory. The excitatory neurotransmitters induce sleep and inhibitory ones cause awakening (37). Extensive studies have been carried out by Mallick et al (1983) and Mohan Kumar et

al (1984) to study the role of preoptic neurons in sleep-wakefulness mechanisms. Their studies have conclusively shown that pre-optic area has a crucial role to play in bringing about changes in states of consciousness on a diurnal basis and otherwise. The reticular activating system exerts an excitatory influence on the pre-optic area and thus affecting the wakefulness of an individual (38, 39).

#### Functions of sleep

One of the most important reasons to go to sleep is to avoid feeling sleepy! It is also suggested that slow wave sleep has more of a role in physical restorative process. Physical exercise causes more sleepiness (40) and this preferentially increases the slow wave sleep. REM sleep is required for maintaining the integrity of neuronal excitability and slow wave sleep has more effect on musculoskeletal system (41). Starving increases the slow wave sleep thereby suggesting that it may be an attempt to preserve and restore proteins (42). Sleep forces a protective inactivity. Sleep prevents the hypothermia, which would have otherwise occurred if the animals were to move around in cold weather. The protective role of sleep is not only with respect to the whole animal but also with respect to the neurons, which are protected by periodic inactivity. It is thought that sleep specially REM component facilitates memory and learning. Sleep-waking-related fluctuation of hormones and neurotransmitters may be modulating memory processes (43). It has also been proposed that REM sleep removes undesirable data from the memory (44). Contrary to all this Horne has suggested

that REM sleep is not necessary for memory consolidation (45). The role of sleep in memory is not so clear.

Moruzzi has suggested a psychophysiological restorative role of sleep (46). The increase in anabolic hormones during sleep and catabolic hormones during awake state supports the restorative theory of sleep. The most frightening revelation about the essentiality of sleep became known in 1983 when Rechtschaffen et al deprived 8 rats of sleep for 5–33 days and none survived to lead a normal life (47). Presently the accepted thinking is that sleep offers rest to the brain, which remains active when awake and is not rested by non-sleep bed rest. Working on this hypothesis, Drummond et al (48) studied the brain activity of sleep-deprived subjects by Blood Oxygen Level Dependent functional Magnetic Resonance Imaging (BOLD fMRI). Interestingly they found paradoxical results. They predicted the prefrontal cortex (PFC) should be less responsive to the cognitive demands of sleep-deprived individuals as it is highly active in awake states. They found that PFC preferentially got activated in response to cognitive demands in sleep-deprived individuals when compared to the temporal activation in non-sleep deprived subjects. This illustrates the plasticity of the brain and its capability to recruit various areas at the time of need even in sleep deprivation. Sleep is also known to have many beneficial effects when exposed to high altitude (HA). A study done by Selvamurthy et al has brought to light that there is a curtailment of slow wave sleep and frequent arousals during sleep at HA. Also there is an augmentation in

sympathetic activity which equilibrates after one week, however the increase in the autonomic arousal continues to be there. It is also seen that those individuals who do not exhibit this response are more prone to HA maladies like Acute Mountain Sickness and High Altitude Pulmonary Edema (49). Sleep deprived individuals do not acclimatize well to high altitude and that recovery sleep following sleep deprivation in high altitude takes longer time to complete (50).

#### Requirement of sleep

Numerous studies have concluded that for maximal performance 4.5 to 5.5 hours of sleep is required everyday and it is better if this quantum of sleep is obtained continuously (7-16, 28, 51, 52). However, various workers have noted that even very short periods of ten minutes each of sleep has recuperative value from fatigue and sleepiness (13, 53-57). It was originally thought that it is not only the time period of sleep but the stages of sleep must also be taken into consideration while looking at the recuperative value of sleep. However now numerous studies have convincing results to confirm that when the discussion is of minimal sleep it is purely temporal with no relation to sleep stages whatsoever (58-60). Horne in 1988 has partitioned sleep into core and optional sleep (45). Core sleep is defined as the first 4-5 hours of sleep i.e. the first three sleep cycles. This sleep is necessary for optimal performance. The remaining sleep is optional and can be easily done away without affecting the performance. To satisfy the core sleep needs of an individual he can adopt any of the following methods: (i) unbroken five hours

of sleep. (ii) Anchor sleep (iii) Prophylactic sleep. (iv) Long naps. (v) Ultra short sleep. Anchor sleep is basically tethering at least 4 hours to a fixed time of the day (61). Dinges et al suggested napping in anticipation of sleep loss (53). Whether sleep can be stored or not is still to be studied in detail. Long naps can be anything from 1 to 4 hours. These only partially satisfy the need of core sleep (6, 13, 62-64).

It is seen that ultra-short naps of 10 minutes also have refreshing and recuperative effects, though may not be as much as that of continuous sleep (12, 55). For recuperative effects, naps must be atleast of 4-10 minutes duration. Ultra short naps are extremely helpful in avoiding the 'behavioral freezing' i.e. a condition seen in sleep deprived individuals wherein they lapse into temporary immobility instead of responding quickly to an emergency (65). The disadvantages of ultra short sleep are the increase in sleep inertia i.e. a feeling of lethargy and lassitude which prevents an individual to immediately perform mentally and or physically to his optimum, and decrease in sleep efficiency i.e. the elimination of feeling of sleepiness following a period of sleep. This reduction in efficiency can be reduced by adequate motivation.

#### Sleepiness

Defined as the transition state between wakefulness and sleep, sleepiness did not receive the attention it deserved in the earlier days. Attention was drawn to it for the first time by Dement in 1989 (66). During sleepiness the whole CNS undergoes adjustments and reorganization in the

neural mechanisms all of which are completely reversible. When does sleepiness end and when does sleep start? Kleitman (1963) suggested that sleep is indexed by the absence of critical reactivity followed by recall (67). Rechtschaffen defined it as stage I sleep (68). Johnson suggested that sleepiness ended with stage II EEG changes of sleep (69). Onset of sleep could be marked by the behavioral response cessation coupled with the sharp increase in EEG synchronization (70, 71). The graded nature of sleepiness may suggest it to be a passive phenomenon—the inevitable result of decrease in the excitatory process of wakefulness. However more recently sleepiness is understood to be an active process (72). Sleepiness is predictably affected by biorhythmic variations in sleep alertness, and physiological measures (73–75).

Sleepiness normally occurs at three times in 24 hours, just after awakening in the morning, in midafternoon and prior to sleep in the evening (73). Interestingly it has been noted that the afternoon sleep propensity is inversely related to the body temperature as compared to the other times of sleepiness. In the morning and evening it has been noted that a fall in temperature is associated with sleepiness. However, in the afternoon, body is sleepy but is at its highest temperature. (76, 77). Sleep-wake cycle (SWC) and endogenous circadian pacemaker (ECP) run at exactly the same period of time and is a group II oscillator, Y oscillator or factor S i.e. this rhythm is more labile and is more like a capacitor than an oscillator (77). It is worthwhile noting that sleepiness is not the same at all times

that it occurs. Qualities of sleepiness vary according to the type of sleep that follows (42).

Broughton has categorized sleepiness as: NREM sleepiness, REM sleepiness and derousal sleepiness, occurring because of pressure for REM, NREM and impaired reticulo-cortical waking processes respectively (78). REM sleepiness is more intense and derousal type may coexist with the other two. The type of sleepiness may reflect the type of sleep, which would ensue. Glovinsky et al (1990) did sleep deprivation studies and reported no stage specific differences in Stanford's Sleepiness Scale (SSS) and Multiple Sleep Latency Tests (MSLT) (79). More studies in this field would be required before any conclusion can be drawn. Another classification of sleepiness has been put forward by Horne in 1988, who describes sleepiness as antecedent to core sleep i.e. the sleep that repairs the effects of wear and tear of wakening and optional sleep that tediously fills the hours of darkness till sunrise when sleep is maintained beyond the core sleep (45). Both can coexist at a given time. The main purpose of sleepiness presently appears to be the smooth transition from wakefulness to sleep and sleep to wakefulness.

Several methods are employed for measuring Sleepiness. Prominent among these are Stanford Sleepiness Scale (SSS), (69, 80), Visual Analogue Scale (VAS) (81), and Thayer Activation Deactivation Adjective Check List (ADACL) (82). A number of performance tests to assess the effects of sleepiness have also been designed like the Simple Reaction Time, Complex

Reaction Time, Vigilance Tests (7, 83, 84). Many workers (7-9, 85-93) have demonstrated the utility of these tests. The drawbacks of these performance tests are large practice effects, intrusive factor of these measurements, and susceptibility to factors like motivation, distraction and comprehension. EEG offers better and more objective possibilities of state discrimination. This has been utilized in many sleep wakefulness pattern studies (68, 94-99). This discriminative variation in EEG pattern has been used as reference for other Sleepiness related tests like Multiple Sleep Latency Tests (MSLT) (100-102). MSLT and its variants have been developed to provide an objective and repeatable quantification of sleepiness (103). However, MSLT is the primary index of sleepiness; the phenomenon of daytime sleepiness has to be studied in conjunction with the study of sleep that follows (104, 105). Polygraphic Index of Sleepiness (PIS) and Polygraphic Score of Sleepiness (PSS) have been developed for the same (104). Cognitive behaviour also undergoes changes during sleepiness (106, 107).

Pivic, Kleitman and Vihvelin in 1948 described stages of sleepiness (75, 108, 109), which were further refined and redefined, by Foulkes and Vogel in 1965 (14). They are (i) Alpha EEG continuous with one or more REMs prior to wakening. (ii) Alpha EEG discontinuous with SEMs at least 20-30 seconds of record prior to wakening call. (iii) Descending stage I EEG. (iv) Descending stage II EEG of 30 seconds to 2.5 minutes. A point to be noted here is the lack of affective intensity in the 'recall' of mental activity in arousal. It was more

dream like. As the sleepiness progresses through the four stages, psychologically subjects progressively relinquished control over the mental activity; then the awareness of the environment was lost and subsequently loss of reality orientation with consequent onset of hallucination (109, 110).

### Sleep and performance

Lapsing, cognitive slowing, memory impairment, vigilance decrease and optimum response shifts are all the recognized adverse effects of sleeplessness. Performance and its change with sleepiness will provide a vital link in understanding performance and improving it in condition that is not conducive to regular sleeping hours. Most of the studies in the past have suggested that the fundamental effect of sleep loss has an adverse effect on performance, especially on all cognitive and sustained attention tasks (3, 7, 9, 45, 60, 75, 83, 84, 111-114). In the first 70 years of this century, only about 36 studies have been done involving sleep deprivation and performance (9). Also there is a lot of variation in results of laboratory and field studies. Performance and actual field performance are not identical and performance serves as an index and not substitutes to field studies.

Earliest sleep deprivation study was by Patrick and Gilbert in 1896, where 3 young adults were studied for 90 hours of sustained wakefulness (115). They observed a general decrease in sensory acuity, quickness of reaction, motor speed and memorizing ability, related to performance following recovery sleep. Five vital

observations are to the credit of this study namely: (i) no matter how much subjects or experiments guarded against them, subjects always experienced brief uncontrollable naps even during certain performance tasks; (ii) these brief sleeps were often accompanied by "semi waking dreams" especially if the subject was giving an oral performance task. (iii) After 72 hours of wakefulness, subjects could not attend to a memory task; (iv) recovery sleep was profoundly deep in terms of arousal threshold to a painful stimulus. Robinson and Hermann (1922) reported their results on three men who underwent 65 hours of wakefulness (116). However this study did not corroborate the results of the previous study. Kleitman (1923) studied 35 healthy males undergoing 60-65 hours of sleep loss (75). He concluded that wakeful bed rest is not a substitute to sleep. In this study issues of motivation and performance change with time on task were highlighted. An interesting development at this time was the concept of 'blocks' put forward by Bills (117-119). Blocks are defined as a pause in response equivalent to two to three times the average response in non-sleep deprived subjects. These blocks served as good and reliable investigative tools in the studies that ensued.

Warren and Clark (1937) analyzed the performance of four adults undergoing 48-65 hours of wakefulness (120). They observed that blocks increased but modal and average performance did not change. Hence it was concluded that evaluation of average performance did not vary but tasks that evaluated performance variability showed more effect of sleep loss. Blocks increasing as a function of sleep loss

suggested an increase in the unevenness of responding. These blocks or pauses are associated with changes in EEG (121). Bills' block is a delay in delivery of stimuli as well as delay in reaction to stimuli. Williams, Lubin and Goodnow (1959) put forward the 'lapse hypothesis' in their study of 40 males undergoing 72-100 hours of sleeplessness (122). They further elaborated on 'paced' and 'unpaced' tasks. In the former the error was lapses while in the latter it was a reduction in speed. The same has been supported by many later studies. (6, 15, 85, 123). The above studies also concluded that there is a definite cognitive slowing due to sleepiness.

There is an interesting association of sleep loss with memory. Studies have shown that there is a reduction in immediate recall and the recall was not impaired if the information was taken prior to the sleep loss. Both delayed and immediate recall was noted to be impaired in information taken after sleep deprivation (115). Patrick and Gilbert (1896), Williams, Lubin and Goodnow (1959) and Polzella (1975) showed that short-term memory impairment was attributed to lapsing during encoding and attention deficit in rehearsal failure (122, 124, 125).

The time on task is also considerably affected in sleepiness. The longer the task duration the more are the chances of impairment. It may also be surmised here that sleeplessness causes a deficit in endurance and not quality. Contradictory conclusions have been drawn in studies in which musculoskeletal performance has been considered (4). Not only in the lapses but also in the 'non-lapsing' period there is



a general increase in the reaction time, suggesting that there is a shift in the optimum response capability of the subjects. There is no role of task complexity sensitivity to sleep loss (126). Effect of sleepiness on performance is not a simple linear function of time awake. There is a circadian pattern of performance in the 24-hour period of wakefulness. Froberg et al (1975) have highlighted the significance of calculating the circadian variation within consecutive days of sleep loss (127). Some parameters of performance are more affected than the others but every parameter shows a circadian pattern, which is affected by sleep loss and vice versa (128). The poorest performance is seen in the circadian nadir and most deterioration is seen by second or third night without sleep. Point to be noted here is that the physiological markers of circadian rhythm like body temperature remains unaltered. Babkoff et al (1989) showed that there is a phase delay in circadian maxima and minima during sleep deprivation (129). The effects of circadian desynchrony and its effects on performance are yet to be studied for definite interpretation. The importance of these results will be in understanding the effects of rapid time zone changes as industrial operations and sustained military operations (64, 130).

The effect of sleepiness on learning as a function is not yet very clear. This is because of the practice curve affecting the learning curves. Efforts are being made to obtund the practice effects on learning (131). Sleep loss does not result in a loss of the ability to comprehend what is being read but it leads to cessation of reading (53). This is

because of difficulty in concentrating and keeping it in memory. The alteration in reasoning, perception and judgment are attributed to change in sustained attention including lapses, cognitive slowing, memory deficits, habituation and optimum response shifts. Another school of thought regarding adverse effects on sleepy performance is primarily reduced or competing motivation (132, 133). In other words the effect of sleep loss are more on the willingness to perform than on the capacity to perform (45). Though motivational factors are salient in demonstrating performance deficits from sleep loss, it does not follow that there is no reduced capacity to perform. Another aspect of sleepiness is the rate of state lability. The individual is oscillating between alertness, lowered vigilance, and microsleeps.

The MSLT in a sleep-deprived individual shows an accelerated stage I sleep during the first night without sleep (103). As the deprivation increases the latency from the initial stages to SWS is reduced considerably (123). Tasks, which are monotonous and require sustained attention potentiate habituation and hence increase the pressure to sleep or micro sleep. In this context, Dinges observed that greater the physiological sleepiness brought about by sleep loss, the more dependent the individual becomes on the environment to maintain wakefulness and it becomes more vulnerable to environmental monotony (114). Loss of ability to sustain attention is the single most adverse effect of sleepiness, which is the corner stone of the deterioration of performance.

### Physical performance and sleep

There are a number of studies in which effects of exercise on sleep deprivation effects (1, 2, 40, 134) have been studied. Also many studies have gone in to the effects of sleep deprivation on physical performance (2, 3, 9, 107, 135-146). Sleep has a tremendous effect on physical activity and vice versa (1, 2, 13, 107, 134-139, 141, 143, 144, 146-151). It has been observed that shift workers report more fatigue than day workers (152, 153). This fatigue is maximum in night shift and minimum in afternoon shift. An important determinant of physical efficiency in shift workers is the number of night shifts in a row. Though normal day work levels are never reached, working seven or more nights continuously have shown good working levels (154). Rated activation, rated alertness and the well being of shift workers is significantly affected in rapidly rotating shifts (155). Permanent night work also does not seem to ameliorate this problem. Shift workers tend to be day oriented even in permanent night shifts (156). 8-hour shifts are less detrimental to performance and well being than the usual 12-hour shifts (157). Four days of recuperation seem to be necessary after a seven-day night shift (158).

Leprault (1997) noticed the effect of exposure of subjects to bright light and or exercise on subjective sleepiness was critically dependant on the time of exposure (1). The most beneficial effect was seen when exercise was done when the subjects felt maximum sleepiness. Lubin et al (1976) concluded that exercise definitely increased performance decrement and sleepiness due to sleep loss (40). Taub et al have concluded

that any alteration in sleep patterns, even an increase in normal amount of sleep is deleterious to performance and mood (145). The comparison of response of males and females has been done in a few studies and the results are not consistent. While Hill et al (1994) found no difference in response (137), Goodman et al observed that sleep disturbances reduces  $VO_2$  max in males but not in females (149).

There is no change in cardio respiratory response to exercise in sleep-deprived individuals (144, 146, 150). There are a number of studies to show that sleep deprivation has deleterious effects on physical performance (2, 4, 60, 113, 134). Martin has observed a reduction in resting heart rate, plasma catecholamine levels, blood pH and an increase in  $CO_2$  production and minute ventilation (139). His study also showed that the ability to perform maximal exercise is reduced but there is no change in exercise endurance. Physical work requiring up to 45% of  $VO_2$  max decreases significantly after 48 hours of wakefulness (143). No change was seen with sleep deprivation in muscle contractile properties and anaerobic power maximum (146, 149). Mougin et al have reported an increase in ventilation and an upward drift in lactate accumulation following partial sleep deprivation (141). There is a reduction in cardiorespiratory function; self selected walking pace and perceived exertion increased (143). There is no change in aerobic or anaerobic energy systems to high intensity exercise even after one day of wakefulness (137). However, some studies show deterioration in performance after sleep deprivation and a beneficial effect is seen after a nap (6, 40, 107, 112). This could

be attributed to the individual variation of delay in sympathetic arousal in early stages of exercises following sleep (107).

After 60 hours of sleep deprivation no change was seen in Wingate aerobic power test, blood lactate response to cycle exercise at 70%  $\text{VO}_2$  max, and hence it was concluded that 60 hours of sleep deprivation did not lead to any impairment in physical work capability (144). Also after 60 hours of wakefulness, no change in endurance time for exercise till exhaustion was seen, however time to exhaustion was reduced (146, 150). Rise in insulin resistance and decrease in glucose tolerance observed in these studies have explained such reduction in exhaustion time. Meney et al (1998) noticed that there was no effect of one night's sleep deprivation on day 1 and by day 2 the muscle strength was reduced in both sleep deprived and control subjects. Partial sleep deprivation causes an alteration in physiological response to exercise (4, 141, 142). Studies have reported a rise in heart rate (141), a decrease in heart rate (4, 142), and no change in heart rate (1, 159). This disparity in various studies should be considered in the light of individual variation of catecholamine response or the varied adrenergic effect of catecholamines (160).

#### Sustained operations in military

Long ago wars were fought from sunrise to sunset. Later with the advent of sophistication in military technology the battle was concentrated in the hours of darkness. Now with the changes in doctrines of war, sustained operations will remain the

key words of war in the years to come. Since long, operational consequences of sleep deprivation have been studied (13, 161, 162). However there are no studies on training the troops for optimal performance in sleepy conditions. For studies on sustained operations, field and lab studies are complementary. Numerous studies have been done on sustained operations in various kinds of weather and terrain (5, 50, 134, 140, 167-171). These studies have not been able to conclusively assess the sustained operation conditions for want of longer duration studies. Most of the research done on sleep deprivation and performance with relevance to military operations is by studying subjects' capabilities on selected tasks at different times of wakefulness (54, 134, 140, 167-171). The major drawback however is that the interaction of environmental-situational and individual stressors cannot be studied or replicated. It is also difficult to get representative tasks of operational tasks. Moreover it is difficult to simulate the stress of war in lab studies and non-operational field studies. It is not possible to study the forces when they are actively engaged in operations. Hence the findings of various studies have to be intelligently interpreted and appropriately extrapolated to the different sustained operations in the military.

One study that has come closest to ideal of continuous performance demands was conducted by Mullaney in 1983 (63). He noted that after 18 hours of testing there was serious deterioration in performance. Johnson and Naitoh had similar results in 1974 but the time after which deterioration was noticed was 40 hours (172). Field

studies have been done in varying conditions from Arctic to desert and in air and ship operations (163–166, 173). The experimental design and experimental tasks for lab studies should be near ideal and should be tailor made to the aim of the study. A general design for the same is a 6-hour block of identical cognitive tasks (62). Examples of typical performance tasks are modified version 4 choice serial reaction time task (174), encode decode task (161, 175), continuous subtraction task (51) and logical reasoning task (176). In addition to these, SAM subjective fatigue checklist (177), and NHRC Mood scale (60) are usually included in performance related sleep deprivation studies (5, 6, 62, 146, 162).

An interesting fact to note is that longer the tasks more sensitive it is to sleep deprivation (3, 6, 84, 93) and longer tasks have effects similar to that of long work sessions. Common observation with all these studies is that sleepiness interferes with the ability of subjects to override the deleterious effects of sleepiness.

Physical conditioning, physical exercise and scheduled physical exercise have not been found to have any significant effect on reduction of sleep loss effects and so also sleep loss does not have any significant effect on physical performance in the initial few days (5, 6). Similarly no beneficial effects of reducing the cognitive load or introducing non-sleep rest periods were observed due to sleep loss. Naps taken at the low point in circadian cycle provide restoration of performance and recuperation of cognitive performance (6, 6).

Performance is known to deteriorate with increasing waking hours (60, 63). Hence for optimal performance, troops should be allowed to sleep for 7–8 hours every day for at least a week before sustained operations (148). Important military tasks should be avoided during sleep inertia time i.e. 5–30 minutes after waking (148). There is no requirement for hour-to-hour restoration of sleep after operations. 10–12 hours of continuous sleep has tremendous restorative and recuperative powers (148).

### Conclusion

Sleep deprivation causes significant deterioration in mood, motivation and cognitive functions including vigilance tests and also has deleterious effects on physical performance but this effect is influenced by many individualistic variables like catecholamine response and adrenergic effect of catecholamines and many others. Few factors to be considered in good sleep management is assessing the time when the individual or the group is required to be at optimum level of performance and tailor his sleep schedule accordingly. Also different sleep schedules will have to be designed depending on the individual's contribution to the assigned task or project. Decision-making staff and executive staff will have different effects of sleep deprivation and hence should be advised accordingly. Motivation can override most of these deleterious effects and hence coupled with good sleep management would be the corner stone for effective shift workers' performance and success in most sustained operations.

## REFERENCES

1. Leproult R, Van Reeth O, Byrne MM, Sturis J, Van Cauter E. Sleepiness, performance, and neuroendocrine function during sleep deprivation: effects of exposure to bright light or exercise. *J Biol Rhythms* 1997; 12: 245-258.
2. Menev I, Waterhouse J, Atkinson G, Reilly T, Davenne D. The effect of one night's sleep deprivation on temperature, mood, and physical performance in subjects with different amounts of habitual physical activity. *Chronobiol Int* 1998; 15: 349-363.
3. Wilkinson RT. Effects of upto 60 hours of sleep deprivation on different types of work. *Ergonomics* 1964; 17: 175-186.
4. Chen HI. Effects of 30-h sleep loss on cardiorespiratory functions at rest and in exercise. *Med Sci Sports Exerc* 1991; 23: 193-198.
5. Angus RG, Heselgrave RJ. The effects of sleep loss and sustained mental work: Implications for command and control performance. (NATO report AGARD-CP-388). In sustained intensive air operations: Physiological and psychological aspects. 1983. Paris: NATO Advisory group for Aerospace research and Development, pp.11.1-11.21.
6. Angus RG, Heselgrave RJ. The effects of sleep loss on sustained cognitive performance. *Behav Res Methods Instrum Comput* 1985; 17: 55-67.
7. Wilkinson RT. Sleep deprivation. The physiology of human survival (Eds. OG Edholm and AL Bacharach), 1968; pp.399-430.
8. Wilkinson RT. Methods for research on sleep deprivation and sleep function. *Int Psychiatr Clin* 1970; 7: 369-819.
9. Wilkinson RT, Edwards RS, Haines E. Performance following a night of reduced sleep. *Psychonomic Science* 1966; 5: 471-472.
10. Webb WB, Agnew HW Jr. The effects of chronic sleep limitation on sleep length. *Psychophysiology* 1974; 11: 265-274.
11. Levine B, Roehrs T, Stepanski E, Zorick F, Roth T. Fragmenting sleep reduces its recuperative value. *Sleep* 1987; 10: 590-599.
12. Husband RW. The comparative value of continuous versus interrupted sleep. *J Exp Psychol* 1935; 18: 792-796.
13. Haslam DR. Sleep loss, recovery sleep, and military performance. *Economics* 1982; 25: 163-178.
14. Foulkes D, Vogel G. Mental activity at sleep onset. *J Abnormal Psychol* 1965; 70: 231-243.
15. Bonnet MH. Performance and sleepiness as a function of frequency and of sleep disruption. *Psychophysiol* 1986; 23: 263-271.
16. Downey R, Bonnet MH. Performance during frequent sleep disruption. *Sleep* 1987; 10: 534-563.
17. Moruzzi G. The historical development of the deafferentation theory of sleep. *Proc Am Philos Soc* 1964; 108: 19-28.
18. Purkinje D. Wachen Schlaf Traum, verwandte zustande. In: H.P. von Wagner, ed. Handwörterbuch der Physiologie. Vol 2 Braunschweig, Vieweg&Sohn, 1846: 412-480.
19. O'Leary J, Goldring S. Science and epilepsy. New York: Raven Press, 1976; 164-165.
20. Greisinger W. Berliner medizinisch-psychologische Gesselschaft. *Arch Psychiatr Nervenkr* 1968; 1: 200-204.
21. Aserinsky E, Kleitman N. Two types of ocular motility during sleep. *J Applied Physiol* 1955; 8: 1-10.
22. Aserinsky E, Kleitman N. Regularly occurring periods of eye motility and concomitant phenomenon during sleep. *Science* 1953; 118: 273-274.
23. McWilliam JA. Some applications of physiology to medicine. III. Blood pressure and heart action in sleep and dreams. *Br Med J* 1920; 11: 1196-1200.
24. Von Economo C. Schlaftheorie. *Ergeb Physiol* 1929; 28: 312-339.
25. Bremer F. Historical development in ideas on sleep. In: Petre-Quadens O, Schlag JD, and Eds. Basic sleep mechanisms. New York: Academic Press, 1974: 3-11.
26. Berger H. Uber das Elektroenkephalogram des Menschen. *Arch Psychiatr Nervenkr* 1929; 87: 527.
27. Moruzzi G, Magoun H. Brainstem reticular formation and activation of the EEG. *Electroenceph Clin Neurophysiol* 1949; 1: 455-473.

28. Richardson GS, Carskadon MA, Orav EJ, Dement WC. Circadian variation of sleep tendency in elderly and young adult subjects. *Sleep* 1982; 5, Suppl. 2, S82–S94.
29. Miles LE, Dement WC. Sleep and aging. *Sleep* 1980; 101: 911–917.
30. Nicholson AN, Stone BM, Jones MMC. Wakefulness and reduced rapid eye movement sleep: Studies with Prolintane and Pemoline. *B J Clin Pharmacol* 1980; 10: 465–472.
31. Adams K, Oswald I. Sleep is for tissue restoration. *J Coll Physicians* 1977; 11: 376–388.
32. Adams K. Sleep as a restorative process and theory to explain why. *Prog Brain Res* 1980; 53: 289–325.
33. Oswald I. Sleep as a restorative process: human clues. *Prog Brain Res* 1980; 53: 279–288.
34. Oswald I. Sleep, the great restorer. *New Sci* 1970; 46: 170–172.
35. Rechtschaffen A, Kales A. A manual of Standardised terminology, techniques and scoring systems for sleep stages in human. Los Angeles: UCLA Brain information service/Brain research institute, 1968.
36. Loomis AL, Harvey E, Hobart GA. Distribution of disturbance patterns in human electroencephalogram with special reference to sleep. *J Neurophysiol* 1938; 1: 413–430.
37. Steriade M, Mc Carley RW. Brain stem regulation of wakefulness and sleep. New York: Plenum, 1990.
38. Mallick BN, Chhina GS, Sundaram KR, Singh B, Mohan Kumar V. Activity of preoptic neurons during synchronization and desynchronization. *Exp Neurol* 1983; 81: 586–597.
39. Mohan Kumar V, Mallick BN, Chhina GS, Singh B. Influence of ascending reticular activating system on preoptic neuronal activity. *Exp Neurol* 1984; 86: 40–52.
40. Lubin A, Hord D, Tracy M, Johnson L. Effects of exercise, bed rest, and napping on performance decrement during 40 hours. *Psychophysiol* 1976; 13: 334–339.
41. Griffin SJ, Trinder J. Physical Fitness, Exercise, and human sleep. *Soc Psychophysiol Res* 1978; 15: 447–450.
42. Karacan I, Rosenbloom I, Londono J, Salis, Thornby J, Williams R. The effect of acute fasting on sleep and the sleep GH response. *Psychosomatics* 1973; 14: 33–37.
43. Mc gaugh JL, Gold PE, Van Buskirk, RB et al. Modulating influences of hormones and catecholamines on memory storage processes. In Gispen WH, van Wimersma-Gridanus TB, Bohus B, et al., eds. *Hormones, Homeostasis and the brain*. Amsterdam: Elsevier, 1975; 151–162.
44. Crick F, Mitchison G. The function of dream sleep. *Nature* 1983; 304: 111–114. Horne J. *Why we sleep*. 1988; Oxford University Press, New York.
46. Moruzzi G. The functional significance of sleep with particular regard to the brain mechanisms underlying consciousness. In: Eccles, J, ed. *Brain and conscious experience*. New York: Springer, 1966.
47. Rechtschaffen A, Gilliland MA, Bergmann BM, Winter JB. Physiological correlates of prolonged sleep deprivation in rats. *Science* 1983; 221: 182–184.
48. Sean PA Drummond, Gregory G Brown, J Christian Gillin, John L Stricker, Eric C Wong, Richard B Buxton. Altered brain response to verbal learning following sleep deprivation. *Nature* 2000; 403: 579–686.
49. Selvamurthy W, Raju VRK, Ranganathan S, Hedge KS, Ray US. Sleep patterns at an altitude of 3500 metres. *Int J Biometeor* 1986; 30: 123–135.
50. Selvamurthy W. Neurophysiological problems in snow bound High Altitude areas. *Def Sci J* 1984; 34: 397–415.
51. Cook MR, Cohen H, Orne MT. Recovery from fatigue. Tech Rep No 55, 1972; Fort Detrick, MD: US Army Medical Research and Development Command.
52. Webb WB. *Sleep: The gentle tyrant*. Prentice Hall, NJ 1975.
53. Dinges DF. Prophylactic napping to sustain performance and alertness in continuous operations (Contract No. N00014-80-C-0380, Progress report 0001AN), 1983, Office of Naval Research, VA.
54. Naitoh P. Circadian cycles and restorative power of naps. In: *Biological Rhythms, sleep and shift work*, 1981. Johnson LC, Tepas WP, Colquhoun WP, Colligan MJ, eds. New York. Spectrum, pp553–580.
55. Stampi C. Polyphasic sleep strategies improve prolonged sustained performance: a field study in 99 sailors. *Work Stress* 1989; 3: 41–55.

56. Stampi C. Ultra short sleep – wake cycles improve performance during one man trans atlantic races. Sleep'84. 1985. Koella WP, Ruthern E, Schulz H, Eds Stuttgart and New York: Gustav Fischer Verlag pp271–272.
57. Stampi C. Why we nap. Evolution, chronobiology and functions of Polyphasic and ultrashort sleep. Ed Claudio Stampi, 1992. Birkhauser, Boston. pp137–175.
58. Lubin A, Moses JM, Johnson LC, Naitoh P. The recuperative effects of REM sleep and stage 4 sleep on human performance after complete sleep loss. *Psychophysiology* 1974; 11: 133–146.
59. Lumley M, Roehrs T, Zorick F, Lamphere J, Roth T. The alerting effects of naps in sleep deprived subjects. *Psychophysiol* 1986; 23: 403–408.
60. Johnson LC, Freeman CR, Spinweber CL, Gomez SA. The relationship between subjective and objective measures of Sleepiness. *Psychophysiol* 1991; 28: 65–71.
61. Minors DS, Waterhouse JM. Anchor sleep as a synchroniser of rhythms on abnormal routines. *Int J Chronobiol* 1981; 7: 165–188.
62. Heselgrave RJ, Angus RG. The effects of task duration and work session location on performance degradation induced by sleep loss and sustained cognitive work. *Behav Res Methods Instrum Comput* 1985; 17: 592–603.
63. Mullaney DJ and Kripke DF, Johnson LC. Sleep loss and nap effects on continuous sustained performance. *Psychophysiology* 1983; 20: 643–651.
64. Webb WB. Sleep in industrialized settings in the Northern Hemisphere. *Psychol Rep* 1985b; 57: 591–598.
65. Folstein MD, Luria R. Reliability, Validity and Clinical Application of the Visual Analogue Mood Scale. *Psychophysiol* 1973; 3: 479–486.
66. Dement WC. Foreword in Sleep and Alertness. 1989. (eds. DF Dinges and RJ Broughton,) Raven Press, New York.
67. Kleitman N. Sleep and wakefulness. 1963; The university of Chicago press, Chicago.
68. Rechtschaffen A and Kales A (Eds) A manual of Standardised terminology, techniques and scoring system for Sleep stages of Human subjects. 1968 (NIH Publ No. 204). US Govt Printing Office, Washington DC, 1968.
69. Johnson LC. Sleep deprivation and performance. Biological rhythms, sleep and performance, (ed WB Webb) 1982; pp111–141. John Wiley, NY.
70. Ogilvie RD, Wilkinson RT. Behavioral versus EEG based monitoring of all-night sleep/wake patterns. *Sleep* 1988; 11: 139–155.
71. Ogilvie RD, Wilkinson RT. The detection of sleep onset: Behavioral and physiological convergence. *Psychophysiol* 1984; 21: 510–520.
72. Serman MB. Brain mechanisms in sleep. 1963; doctoral dissertation. University of California, Los Angeles. pp116.
73. Broughton RJ. Biorhythmic variations in consciousness and psychological functions. *Can J of Psychol* 1975; 16: 217–230.
74. Broughton RJ. Chronobiological aspects and models of sleep and napping in Sleep and alertness. 1989 (Eds. DF Dinges and RJ Broughton,) pp71–98, Raven Press, New York.
75. Kleitman N. The effects of prolonged sleeplessness of man. *Am J Physiol* 1923; 66: 67–92.
76. Campbell SS, Zulley J. Napping in time free environments in Sleep and alertness. 1989 (eds. DF Dinges and RJ Broughton,) pp121–138, Raven Press, New York.
77. Czeisler CA, Wetzman ED, Moore-Ede MC, Zimmerman JC, Kronauer RS. Human sleep: its duration and organisation depend on its circadian phase. *Science* 1980; 210: 1264–1267.
78. Broughton RJ. Sleep attacks naps and sleepiness in medical sleep disorders. In: Sleep and Alertness. 1989 (eds. DF Dinges and RJ Broughton,) pp267–298, Raven Press, New York.
79. Glovinsky PB, Spielman AJ, Carroll P, Weinstein L, Ellman SJ. Sleepiness and REM sleep recurrence: The effects of stage II and REM sleep awakenings. *Psychophysiol* 1990; 27: 552–559.
80. Hoddes E, Zarcone V, Smythe H, Phillips R, Dement WC. Quantification of Sleepiness: A New Approach. *Psychophysiol* 1973; 10: 431–436.
81. Folkard S, Condon R. Night shift paralysis in air traffic controls officers. *Ergonomics* 1987; 30: 1353–1363.
82. Thayer RE. Factor Analytic and Reliability studies on Activation deactivation Adjective Checklist. *Psychol Reports* 1978; 42: 747–756.

83. Horne JA. A review of the biological effects of total sleep deprivation in Man. *Biol Psychol* 1978; 7: 55–102.
84. Wilkinson RT. Sleep Deprivation in Physiology of survival (Eds. OG Edhotm and AL Bacharach), 1965, pp.399–430, Academic Press, London.
85. Glenville PB, Spielman AJ, Wing AM, Wilkinson RJ. Effects of sleep deprivation on short duration performance measures compared to the Wilkinson Auditory Vigilance task. *Sleep* 1978; 1: 169–176.
86. Guilleminault C, Dement WC. Amnesia and Disorders of Excessive sleepiness. Neurobiology of sleep and Memory. 1977. (Eds RR Drucker-Colin and JL Mc Gaugh). pp.441–456, Academic press, New York, 59,63,63 York.
87. Ogilvie RD, Simons IA, Kuderian RH, Mac Donald T, Rustenberg J. Behavioral, Event Related potentials (ERP), and EEG/FFT changes at Sleep Onset. *Psychophysiol* 1991; 28: 54–64.
88. Rosa RR, Bonnet MH, Warm JS. Recovery of performance during sleep following sleep deprivation. *Psychophysiology* 1983; 20: 152–159.
89. Valley V, Broughton R. Day time performance deficits and physiological vigilance in untreated patients with Narcolepsy-Cataplexy compared to controls. *Revue d'Electroencephal et de Neurophysiol Clinique* 1981; 11: 133–139.
90. Valley V, Broughton R. The physiological (EEG) nature of drowsiness and its relation to performance deficits in narcoleptics. *Electroencephal and Clinl Neurophysiol* 1983; 55: 243–251.
91. Wilkinson RT. Interaction of lack of sleep with knowledge of results, repeated testing and individual differences. *J Exp Psychol* 1961; 62: 263–271.
92. Wilkinson RT. Sleep deprivation-eight questions in aspects of human efficiency (Ed. ColquhounWP), 1972 pp.25–30. The English Universities Press, London.
93. Wilkinson RT. Sleep deprivation: Performance tests for partial and selected sleep deprivation. *Prog Clin Psychol* 1969; 8: 28–43.
94. Davis H, Davis PA, Loomis AL, Harvey EN, Hobart G. Changes in human brain potentials during the onset of sleep. *Science* 1937; 86: 448–450.
95. Davis H, Davis PA, Loomis AL, Harvey EN, Hobart G. Human brain potentials during the onset of sleep. *J Neurophysiol* 1938; 1: 24–38.
96. Dement WC, Kleitman N. The relation of Eye Movement during sleep to dream activity. An Objective method for the Study of Dreaming. *J Exp Psychol* 1957; 53: 339–346.
97. Loomis AL, Harvey E, Hobart GA. Cerebral state during sleep as studied by human brain potentials. *J Exp Physiol* 1937; 21: 127–144.
98. Loomis AL, Harvey E, Hobart GA. Further observations on the potential rhythms of cerebral cortex during sleep. *Science* 1935(a); 82: 198–199.
99. Loomis AL, Harvey E, Hobart GA. Potential rhythms of cerebral cortex during sleep. *Science* 1935(b); 81: 597–598.
100. Carskadon MA, Dement WC. Sleep tendency: An objective measure of sleep loss. *Sleep Research* 1977; 6: 200.
101. Carskadon MA. Ontogeny of human sleepiness as measured by sleep latency. (b) in: Sleep and Alertness: Chronobiological, behavioral and medical aspects of napping (eds. DF Dinges and RJ Broughton,) 1989(b) pp.53–69, Raven Press, New York.
102. Richardson GS, Carskadon MA, Flag W, Van Den Hoed J, Dement WC, Mitler MM. Excessive day time sleepiness in man: Multiple sleep latency measurement in narcoleptic and control subjects. *Electroenceph Clin Neurophysiol* 1978; 45: 621–627.
103. Carskadon MA. Determinants of daytime sleepiness: adolescent development extended and restricted nocturnal sleep. Doctoral dissertation, 1979; Stanford University, CA.
104. Roth T, Nevsimalova S, Sonka K, Docekal P. A quantitative polygraphic study of daytime somnolence and sleep in patients with excessive diurnal sleepiness. *Arch Suisse de Neurologie, Neurochirurgie, Psychiatrie* 1984; 135: 265–272.
105. Roth T, Nevsimalova S, Sonka K, Docekal P. An alternative to multiple sleep latency tests for determining sleepiness in narcolepsy and hypersomnia: A polygraphic score of sleepiness. *Sleep* 1986; 9: 243–245.
106. May J, Kline P. Measuring the effects upon cognitive abilities of sleep loss during



- continuous operations. *Br J Psychol* 1987; 78(Pt4): 443-455.
107. Pierce EF, Mc Gowan RW, Barkett E, Fry RW. The effects of an acute bout of sleep on running economy and  $VO_2$  max. *J Sports Sci* 1993; 11: 109-112.
  108. Pivic RT. 'Sleep: Physiology and Psychophysiology' In: *Psychophysiol* (Eds MGH Coles, E Donchin and SW Porges), 1986, pp.378-406. The Guilford Press, New York.
  109. Vihvelin H. On the differentiation of typical forms of some hypnagogic hallucinations. *Acta Psychiatria Neurology* 1948; 23: 359-389.
  110. Tyler DB. Psychological changes during experimental sleep deprivation. *Dis Nerv Syst* 1958; 16: 293-299.
  111. Freidmann J, Globus G, Huntley A, Mullaney D, Naitoh P, Johnson L. Performance and mood during and after gradual sleep reduction. *Psychophysiology* 1977; 14: 245-250.
  112. Lubin A. Performance under sleep loss and fatigue. Sleep and altered states of consciousness (Eds. SS Kety, EV Evarts and HL Williams) 1967; pp.506-513. Williams Wilkins MD.
  113. Johnson LC. The effect of total, partial and stage sleep deprivation on EEG patterns and performance. Behavior and brain electrical activity. 1975. (Eds N Burch and HL Altschuler), pp.1-30, Plenum, New York.
  114. Dinges DF. The nature of sleepiness: Causes, contexts and consequences in eating, sleeping and sex. (Eds. AJ Stunkard and A Baum) 1989b; pp.147-179. Lawrence Erlbaum NJ.
  115. Patrick GT, Gilbert JA. The effects of loss of sleep. *Psychol Rev* 1896; 3: 469-483.
  116. Robinson ES and Herrmann SO. Effects of loss of sleep. *J Exp Psychol* 1922; 5: 93-100.
  117. Bills AG. Blocking a new principle of mental fatigue. *Am J Psychol* 1931; 43: 230-245.
  118. Bills AG. Fatigue in mental work. *Psychology Review* 1937; 17: 436-453.
  119. Bills AG. Some additional principles of mental fatigue. *Psychological Bulletin* 1934; 31: 671.
  120. Warren N, Clark B. Blocking in mental motor tasks during a 65 hour vigil. *J Exp Psychol* 1937; 21: 97-105.
  121. Bjerner B. Alpha depression and lowered pulse rate during delayed actions in a serial reaction test: A study of sleep deprivation. *Acta Physiologica Scandinavia* 1949; 19: (Supl 65), 1-93.
  122. Williams HL, Lubin A, Goodnow JJ. Impaired performance with acute sleep loss. *Psychol Monographs* 1959; 73: part 14, 1-26.
  123. Dinges DF. Differential effects of prior wakefulness and circadian phase on nap sleep. *Electroencephal and Clin Neurophysiol* 1986; 64: 224-227.
  124. Elkin AJ, Murray DJ. The effects of sleep loss on short-term recognition memory. *Can J Psychol* 1974; 28: 192-198.
  125. Polzella DJ. Effects of sleep deprivation on short-term recognition memory. *J Exp Psychol* 1975; 104: 194-200.
  126. Dinges DF, Powell JW. Sleepiness is more than lapsing. *Sleep Res* 1989a; 17: 84.
  127. Froberg JE, Karlsson CG and Levi L. Shift work: A study of catecholamine excretion, self ratings and attitudes. *Studia Laboris et Salutis* 1972; 11: 10-20.
  128. Gillberg M, Akerstedt T. Sleep deprivation in normals: Some psychological and biochemical data from three studies. *Sleep* 1981; 5th European congress on Sleep Research, Karger, Basel. (1980)
  129. Babkoff H, Mikulincer M, Caspy T, Carasso R, Singh H. The implication of sleep loss for circadian performance accuracy. *Work and Stress* 1989; 3: 3-14.
  130. Haslam DR. Sleep deprivation and naps. *Behav Res Methods Intrum Comput* 1985; 17: 46-54.
  131. Webb WB. Experiments on extended performance: repetition, age and limited sleep periods. *Behavior Research Methods, Instruments, and Computers* 1985(a); 17: 27-36.
  132. Kleinhauss G, Schaad G. Sustained military operations with particular reference to prolonged exercise. Sustained Intensive Operation: Physiological and performance aspects 1983; (Ed J Ernsting), Proceedings of NATO advisory group for aerospace R and D (NATO/GARD Report No. CP-338) Specialised Printing Services, Ltd, UK.
  133. Murray EJ. *Sleep, Dreams and Arousal*. 1965; Appleton century crofts, NY.

134. Opstad PK, Ekanger R, Nummestad M, Raabe N. Performance, mood and clinical symptoms in men exposed to prolonged, severe physical work and sleep deprivation. *Aviat Space Environ Med* 1978; 49: 1065–1073.
135. Brodan V, Kuhn E. Physical performance in man during sleep deprivation. *J Sports Med Phys Fitness* 1967; 7: 28–30.
136. Brodan V, Vojtechovsky M, Kuhn E, Cepelak J. Changes in mental and physical performance in sleep deprived healthy volunteers. *Act Nerv Super (Praha)* 1969; 11(3): 175–181.
137. Hill DW, Borden DO, Darnaby KM, Hendricks DN. Aerobic and anaerobic contributions to exhaustive high-intensity exercise after sleep deprivation. *J Sports Sci* 1994; 12: 455–461.
138. Holland J. Effects of limited sleep deprivation on performance of selected motor tasks. *Res Q* 1968; 39: 285–294.
139. Martin BJ. Effects of sleep deprivation on tolerance of prolonged exercise. *Eur J Appl Physiol* 1981; 47(4): 345–354.
140. Morgan BB, Brown BR, Aluissi EA. Effects of sustained performance on a 48 hours of continuous work and sleep loss. *Human Factors* 1974; 16: 406–414.
141. Mougín F, Simon-Rigaud ML, Davenne D, Renaud A, Garnier A, Katelip JP, Magnin P. Effects of sleep disturbances on subsequent physical performance. *Eur J Appl Physiol* 1991; 63: 77–82.
142. Pickett GF, Morris AF. Effects of acute food and sleep deprivation on total body response time and cardiovascular performance. *J Sport Med* 1975; 15: 49–56.
143. Rodgers CD, Paterson DH, Cunningham DA, Nobel EG, Pettigrew FP, Myles WS, Taylor AW. Sleep deprivation: effects on work capacity, self-paced walking, contractile properties and perceived exertion. *Sleep* 1995; 18: 30–38.
144. Symons JD, VanHelder T, Myles WS. Physical performance and physiological responses following 60 hours of sleep deprivation. *Med Sci Sports Exerc* 1988; 20: 374–380.
145. Taub JM, Berger RJ. Acute shifts in sleep wakefulness cycle: Effects on performance and mood. *Psychosom Med* 1974; 36: 164–173.
146. VanHelder T, Radomski MW. Sleep deprivation and the effect on exercise performance. *Sports Med* 1989; 7: 235–247.
147. Angus RG and Heselgrave RJ, Myles WS. Effects of prolonged sleep deprivation, with and without chronic physical exercise, on mood performance. *Psychophysiol* 1985; 22: 276–282.
148. Giam GC. Effects of sleep deprivation with reference to military operations. *Ann Acad Med Singapore* 1997; 26: 88–93.
149. Goodman J, Radomski M, Hart L, Plyley M, Shephard RJ. Maximal aerobic exercise following prolonged sleep deprivation. *Int J Sports Med* 1989; 10: 419–423.
150. Martin BJ, Chen H. Sleep loss and sympatho-adrenal response to exercise. *Med Sci Sport Exerc* 1984; 16: 59–59.
151. Rognum TO, Vartdal F, Rodahl K, Opstad PK, Knudsen-Baas O, Kindt E, Withey WR. Physical and mental performance of soldiers on high- and low-energy diets during prolonged heavy exercise combined with sleep deprivation. *Ergonomics* 1986; 29: 859–867.
152. Anderson FE. Three-shift work. 1970. Socialforskningsinstituttet, Copenhagen.
153. Menzel W. Menschliche Tag-Nacht Rhythmik und Schichtarbeit. 1962. Schwabe, Basel.
154. Froberg JE, Karlson CG, Levi L, Liderg I. Circadian rhythms of catecholamine excretion and shooting range performance and self-ratings of fatigue during sleep. *Bioll Psychol* 1975; 2: 175–188.
155. Williamson AM, Sanderson JW. Changing the speed of shift rotation: A field study. *Ergonomics* 1986; 29: 1085–1096.
156. Folkard S, Monk TH, Lobban MC. Short and long term adjustment of circadian rhythms in 'permanent night nurses'. *Ergonomics* 1978; 21: 785–799.
157. Rosa RR, Colligan MJ. Extended workdays: Effects of 8-Hour and 12-Hour rotating shift schedules on performance, subjective alertness, sleep patterns and psychosocial variables. *Work and Stress* 1989; 3: 21–32.
158. Meijman TF. Analysis subjective de la recuperation après le postes de nuit dans le cas de rotation lente (7 hours). *Travail Humain* 1981; 44: 315–323.

159. Horne JA, Prtitt AN. Sleep deprivation and physiological response of exercise under steady state conditions in untrained subjects. *Sleep* 1984; 7: 168-179.
160. Halberg F, Frank G, Harner R et al. The adrenal cycle in men on different schedules of motor and mental activity. *Experientia* 1961; 17: 282-284.
161. Haslam DR. The military performance of soldiers in sustained operations. *Aviat Space Environ Med* 1984; 55: 216-221.
162. Heselgrave RJ, Angus RG. Sleep loss and performance (NATO DRG Report DS/ADS/(83) 170). 1983; In: The human as limiting element in military systems. *Brussels: NATO Research Group* pp.61-110.
163. Angus RG, Pearce DG, Buguet AGC, Oslen L. Vigilance performance of men sleeping under Arctic conditions. *Aviat Space Environ Med* 1979; 50: 692-696.
164. Buguet A, Roussel B, Angus R, Sabiston B, Radomski M. Human sleep and adrenal individual reactions to exercise. *Electroceph Clin Neurophysiol* 1980; 49: 515-523.
165. Bittel J, Buguet A, Roussel B, Kuehn L, Angus R. Temperatures corporelles et etat d sommeil. In: Physiological, psychological, and biochemical of a daily sustained submaximal exercise (Fast Ball) Radomski: MW, Defayolle MAJ, eds. Toronto: Franco-Canadian Accord, 1981; Theme 8, Vol. II, pp.D1-D14.
166. Brooks CJ, Shergold DJ, Angus RG, Heselgrave RJ, Redmond DP. Actigraphic measurements of work/sleep patterns during a navy operation. *J R Nav Med Serv* 1988; 74.
167. Ainsworth LL, Bishop HP. The effect of 48-hour period of sustained field activity in tank crew performance. Hum RRO Tech Rep 71-16. Alexandria VA: Human resources research office.
168. Banks JH, Sternberg JJ, Farrel JP, Deboe CH, Dalhamer WA. Effects of continuous military operations on selected military tasks. Behavior and systems research laboratory, Office of research and development, Department of army tech. Research report no. 1166, 1970.
169. Drucker EH, Canon LD, Ware JR. The effect of sleep deprivation on performance over a 48 hours period. 1969; Hum RRO Tech Rep No. 69-8. Alexandria VA, Human Resources Research Office.
170. Haggard DF. Hum RRO Studies in continuous operations. 1970; Hum RRO Prof Pap7-70. Alexandria VA, Human Resources Research Office.
171. Naitoh P, Englund CE, Ryman D. Restorative power of naps in designing continuous work schedules. NHRC Tech rep 82-25. 1982; San Diego, Naval Health Research Center.
172. Johnson LC, Naitoh P. The operational consequences of sleep and sleep deficit (AGARD-AG-193) 1974, NATO Advisory group for aerospace research and development, CA.
173. Angus RG, Pearce DG, Oslen L. Performance on a multiple-choice reaction time test during a period of physical exercise. In: Physiological, Psychological and Biochemical aspects of Daily Sustained Submaximal Exercise (Fast Ball), Radomski MW, Defayolle MAJ, eds. 1981. Toronto: Franco Canadian accord, Theme 8, Vol. II, pp.H1-H15.
174. Wilkinson RT, Houghton D. Portable four choice reaction time test with magnetic tape memory. *Behav Res Methods Instrum* 1975; 7: 441-446.
175. Haslam Dr. The military performance of soldiers in continuous operations: Exercise early call II, and I In: Biological Rhythms, sleep and shift work. 1981. Johnson LC, Tepas WP, Colquhoun WP, Colligan MJ, eds. New York Spectrum, pp.435-458.
176. Baddley AW. A 3-minute reasoning test based on grammatical transformation. *Psychon Sci* 1968; 10: 341-342.
177. Harris DA, Pegram GV, Hartman BO. Performance and fatigue in double crew transport missions. *Aviat Space Environ Med* 1971; 24: 980-986.