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Effect of Sleep Deprivation on Tolerance of Prolonged Exercise*

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Summary. Acute loss of sleep produces few apparent physiological effects at rest. Nevertheless, many anecdotes suggest that adequate sleep is essential for optimum endurance athletic performance. To investigate this question, heavy exercise performance after 36 h without sleep was compared with that after normal sleep in eight subjects. During prolonged treadmill walking at about 80% of the $\dot{V}_{O_2}$ max, sleep loss reduced work time to exhaustion by an average of 11% ($p = 0.05$). This decrease occurred despite doubling monetary incentives for subjects during work after sleeplessness. Subjects appeared to fall into "resistant" and "susceptible" categories: four showed less than a 5% change in performance after sleep loss, while four others showed decrements in exercise tolerance ranging from 15 to 40%. During the walk, sleep loss resulted in significantly greater perceived exertion ($p < 0.05$), even though exercise heart rate and metabolic rate ($\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$) were unchanged. Minute ventilation was significantly elevated during exercise after sleep loss ($p < 0.05$). Sleep loss failed to alter the continuous slow rises in $\dot{V}_E$ and heart rate that occurred as work was prolonged. These findings suggest that the psychological effects of acute sleep loss may contribute to decreased tolerance of prolonged heavy exercise.

Key words: Exercise – Sleep deprivation – Perceived exertion – Oxygen consumption – Ventilation

Introduction

Sleep and exercise appear to interact in complex fashion. Most studies of the relationship between exercise and sleep have focused upon the effects of exercise upon the quality and quantity of subsequent sleep (Walker et al. 1978). In

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contrast, relatively scant attention has been paid to the effects of sleep upon subsequent exercise. In particular, it is unknown if acute sleep loss alters the ability to tolerate prolonged submaximal exercise. A variety of studies have obliquely approached this problem, suggesting that sleep loss may (Copes and Rosentweig 1972) or may not (Brodan et al. 1969; Harris and O'Hanlon 1972) cause various indices of exercise capacity to decline. The question is of considerable practical importance, since exercise after sleep loss in encountered in wide-ranging circumstances. For example, military servicemen on maneuvers, traveling athletes with disrupted sleep schedules, laborers working unusual hours, and climbers acutely exposed to high altitude may all face the combined stresses of physical work and sleep loss.

While there is substantial evidence that sleep loss may induce profound psychological effects (Kleitman 1963), the effect that these psychological changes may have upon exercise performance remains unknown. In addition, little is known of the physiological effects of sleep loss, as measured either at rest (Fiorica et al. 1968) or in exercise (Pickett and Morris 1975; Brodan et al 1969). The influence of any possible physiological changes induced by sleep loss on exercise performance also remains ill-defined.

It was thus our purpose in this study to assess the effects of acute sleep loss upon performance of heavy submaximal exercise, and upon the psychological and physiological responses to that exercise.

Methods

Eight subjects in excellent health gave their informed consent to participate in the study. The experimental protocol had been previously reviewed and approved by an institutional committee for the protection of human subjects. Though all of the subjects were physically active, none was an outstanding endurance athlete. Characteristics of the subjects are given in Table 1.

Exercise Protocol

Each subject performed two treatmill walks to exhaustion at an exercise intensity requiring about 80% of the maximal aerobic power ($\dot{V}_{O_2}$ max). One of these walks followed normal sleep, and one followed a 36-h sleepless period. Treadmill grades sufficient to require about 80% of the $\dot{V}_{O_2}$ max were selected for each subject on the basis of heart rates measured during familiarization experiments. In these experiments, heart rates were measured at a variety of treadmill grades, and the work load finally selected was one that provoked a heart rate of 160–170 beat $\cdot$ min^{-1} (Åstrand and Rhyming 1954). It is recognized that this method of selection of the 80% $\dot{V}_{O_2}$ max work load is only an estimate. However, since each subject served as his or her own control, error induced by this estimate was unimportant. In both experiments on a given subject, treadmill speed and grade were identical. Because of individual differences in fitness, the treadmill grade selected varied among subjects from 10 to 21%. Treadmill speed was 5.6 km $\cdot$ h^{-1} for all subjects.

Physiological and Psychological Measurements During Exercise

After 10 min of exercise, and each 7 min thereafter, the subject breathed for 2 min via a Daniels' valve (R-Pel Inc.) into a 5-l mixing chamber, from which mixed expired gas was continuously

analyzed for CO_2 (Beckman LB-2) and O_2 (Applied Technical Products fuel cell) fractions. Studies with and without water absorbant in the sampling lines showed that both analyzers received gas 100% saturated with water vapour; corrections to the dry gas fractions were thus made on that basis. Both gas analyzers were regularly calibrated with gas mixtures established as standard by micro-Scholander analysis. A 1-min sample followed 1 min of system rinsing with expired gas. Gas leaving the mixing chamber was channeled into a gas meter (Parkinson-Cowan CD4) for ventilation ($\dot{V}_E$) measurement. The gas meter was regularly calibrated with a Tissot spirometer. The subject's inspired gas was passed across a 900 cm^2 warm water surface that was shown in prior experiments to provide 90% saturation with water at a flow rate of 100 $l \cdot min^{-1}$. Heart rate was monitored continuously from the ECG (B-D Electrodyne). Ambient temperature was maintained at $20 \pm 1°$ C.

After each measurement of $\dot{V}_E$ and $\dot{V}_{CO_2}$, subjects were asked to rate their overall perceived exertion, leg fatigue, and breathing effort on a 6–20 scale (Borg 1974).

Sleep Deprivation Protocol

Responses to exercise repeated on several days may be altered by training, learning, or habituation (Shephard 1969). Two measures designed to minimize the influence of these effects on the present results were implemented. First, subjects performed a practice experiment that familiarized them with experimental techniques before entering the study. Second, four of the subjects were studied first after normal sleep, with the other four studied first after sleep loss. The two experiments on a given subject were separated by at least 1 week. All experiments were performed at 19.00 h, after a light dinner at least 2 h earlier. The 36-h sleepless period thus included one night without sleep, and two daytime periods in which normal activities were maintained. Subjects remained awake at night in pairs, both to facilitate wakefulness, and to document that a continuously sleepless state had been achieved. As much as possible during this night, subjects assumed a recumbent position, and used passive means such as conversation and television watching to remain awake. They were asked to follow a normal meal schedule, and to maintain normal caffeine intake, during the sleep deprivation period. All subjects refrained from exercise on the day of study in both the normal and sleep deprived conditions.

Experimentors were not "blind" to the sleep status of the subjects. Subjects of course could not be "blind" to their own sleep status. It was thus a particular concern that subject expectations of poorer performance after sleep loss might influence that performance. To mitigate this potential effect, and to encourage maximal effort, subjects were paid for each minute of exercise that they completed. This payment rate was doubled during exercise after sleep loss. To prevent competition with a previous performance, subjects were not allowed to watch a clock during exercise, and they were not told their work times or accumulated wages until all experiments had been completed.

Statistical Analysis

Data obtained from the control and sleep deprived conditions were compared by use of a Student *t*-test. Because exercise tolerance was not expected to be increased by sleep loss, this comparison was made using a 1-tailed test; all other statistical comparisons were made with a 2-tailed test.

Results

Work time to exhaustion at 80% of the $\dot{V}_{O_2}$ max for each of the eight subjects is shown in Table 1. Mean exercise duration after sleep deprivation was decreased by an average of 11% compared with control, a result that was marginally significant ($p = 0.05$). The difference was more significant when expressed as an

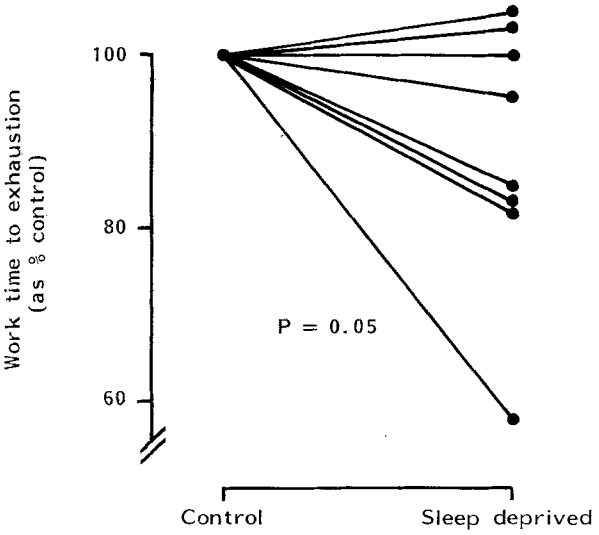

Fig. 1. Individual changes in tolerance of treadmill walking at 80% of the $\dot{V}_{O_2}$ max as induced by 36 h of sleep deprivation. The mean change in work performance (−11%) was marginally significant ($p = 0.05$)

Table 1. Effect of acute sleep loss on tolerance time of exercise at 80% of the $\dot{V}O_2$ max

Subject no.	Sex	Age	Normal duration of sleep (h · d^{-1})	Work time exhaustion (min)	
				Control	Sleep deprived
1	M	25	8.0	79	82
2	M	19	9.0	40	33
3	M	24	8.0	53	31
4	M	23	8.0	156	149
5	F	23	8.5	82	69
6	F	27	6.5	46	46
7	M	25	7.0	57	60
8	F	23	7.5	82	68

absolute change ($p < 0.05$). There was wide individual variation in the effects of sleep loss on exercise tolerance (Table 1 and Fig. 1). Two subjects showed increased duration. Four of the subjects were relatively resistant to the effects of sleep loss, showing endurance changes of less than 5%, while four others were more susceptible to the effects of sleep deprivation, showing endurance decrements of at least 15% (Fig. 1). The distinction between "resistant" and "susceptible" individuals could not clearly be ascribed to differences in sex: both sexes included responders of both types (Table 1). Also, it was unrelated to the amount of sleep normally taken by the subjects each night (Table 1). In addition, the resistant-susceptible categorization did not appear to stem spuriously from effects arising from the order in which control and sleep loss experiments were

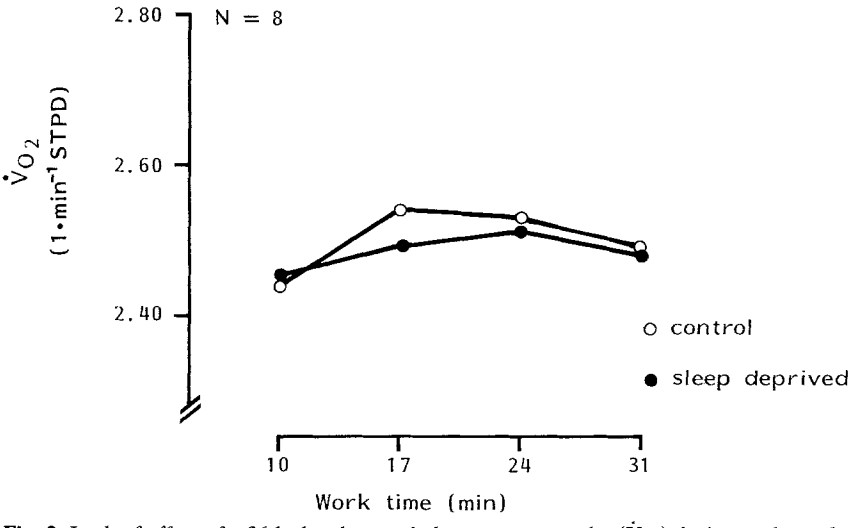

Fig. 2. Lack of effect of a 36-h sleepless period on oxygen uptake ($\dot{V}_{O_2}$) during prolonged treadmill walking at estimated 80% of the $\dot{V}_{O_2}$ max. Each point represents the mean of data obtained from eight subjects

performed. Such effects would be expected to produce improved performance in the second experiment, through learning, habituation, or training (Shephard 1969). As such, these influences could potentially produce artificially "susceptible" individuals in the group studied first after sleep loss, and artificially "resistant" persons in the group studied first after normal sleep. The impact of this factor on the present results was probably minor, since resistant and susceptible subjects were found in equal distribution in both groups.

Changes in exercise performance were associated with few measured changes in the ventilatory, metabolic, and cardiovascular responses to exercise. Because of differences in work time to exhaustion, data from all eight subjects are available only for the first 30 min of exercise (Figs. 2–5). Oxygen uptake during exercise was unchanged by sleep deprivation (Fig. 2). Similarly, $\dot{V}_{CO_2}$ and the respiratory exchange ratio were unaltered by sleep loss. Exercise heart rate was also unaltered by sleep loss (Fig. 3). Minute ventilation was elevated (above control) at 24 and 31 min of exercise after sleep loss (Fig. 4; $p < 0.05$).

Both heart rate and $\dot{V}_E$ undergo upward drift as heavy exercise is prolonged (Dempsey et al. 1977). These drifts were reproduced in this study: in the control walks, heart rate rose 6% ($p < 0.05$) from 10 min of exercise to its conclusion, while $\dot{V}_E$ rose 12% ($p < 0.05$). Sleep loss failed to alter these slow rises: over a comparable time course, heart rate rose 5% ($p < 0.05$) and $\dot{V}_E$ rose 10% ($p < 0.05$) during exercise after sleep deprivation.

In contrast to its relatively minor influence on the physiological responses to exercise measured in this study, sleep loss induced major changes in the perceptual response to exercise. Ratings of perceived exertion were significantly elevated during exercise after sleep loss (Fig. 5; $p < 0.05$). This increase appeared to be greatest in the early stages of exercise, and it remained significant

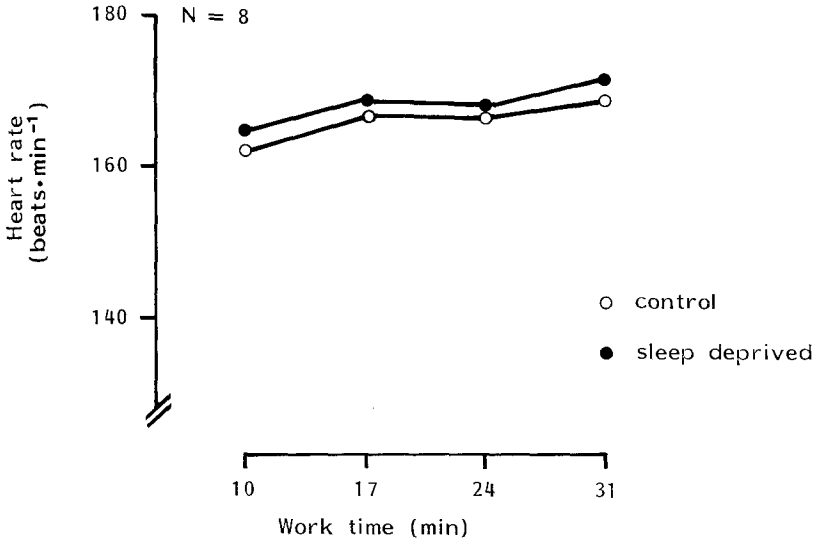

Fig. 3. Lack of effect of a 36-h sleepless period on heart rate during prolonged treadmill walking at 80% of the $\dot{V}_{O_2}$ max. Sleep loss also failed to change the slow rise in heart rate that occured as work continued. Each point represents the mean of data obtained from eight subjects

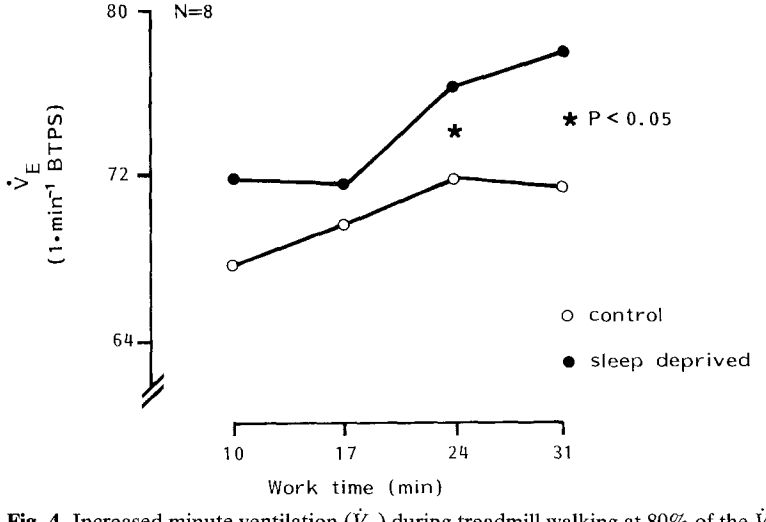

Fig. 4. Increased minute ventilation ($\dot{V}_E$) during treadmill walking at 80% of the $\dot{V}_{O_2}$ max after 36 h of sleep deprivation. Sleep loss failed to change the slow rate of rise of $\dot{V}_E$ as work continued. Each point represents the mean of data obtained from eight subjects

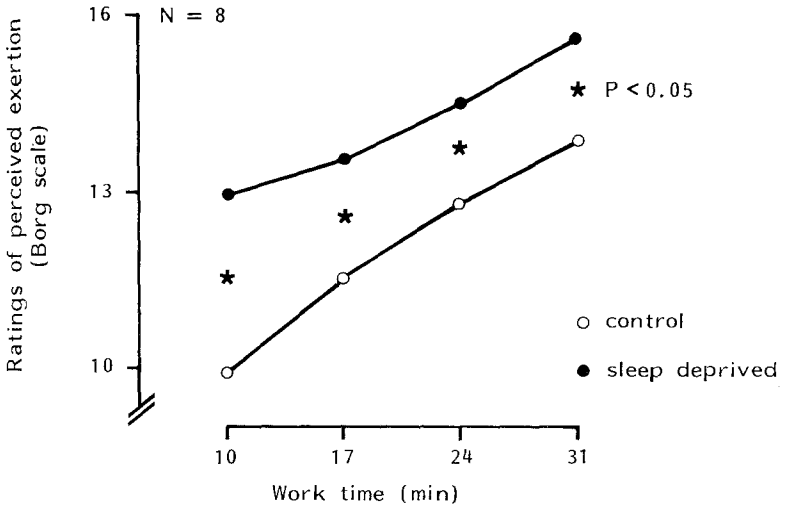

Fig. 5. Increased ratings of perceived exertion [as measured on a 6–20 scale (Borg 1974)] measured during prolonged treadmill walking at 80% of the $\dot{V}_{O_2}$ max after 36 h of sleeplessness. Each point represents the mean of data obtained from eight subjects

throughout the first 30 min of work. There was wide individual variation in the influence of sleep loss upon perceived exertion: two of the subjects reported unchanged perceptual responses, while the other six reported at least 25% greater effort during exercise after sleep loss. The two subjects who reported no perceptual effects of sleep loss during exercise were among the four subjects who were relatively resistant to the effects of sleep loss on exercise tolerance.

Discussion

In this study we found that 36 h of acute sleep deprivation reduced the ability to maintain heavy submaximal exercise. This decrease occurred in the face of unchanged exercise heart rate and metabolic rate. Sleep loss significantly elevated the exertion perceived during exercise.

The mechanism by which sleep loss decreases exercise tolerance in normal persons remains incompletely defined. One obvious possibility is that sleep loss might alter $\dot{V}_{O_2}$ max: reduced $\dot{V}_{O_2}$ max radically reduces the ability to perform prolonged heavy exercise (Ekblom et al. 1972). However, previous work indicates that $\dot{V}_{O_2}$ max is unchanged by sleep deprivation (Martin and Gaddis, in press). This, in combination with unchanged $\dot{V}_{O_2}$ during exercise in this study, indicates that subjects were working at identical percentages of the $\dot{V}_{O_2}$ max during exercise with and without sleep deprivation. Thus, changes in relative usage of a constant $\dot{V}_{O_2}$ max, also powerfully linked to tolerance of prolonged exercise (Rowell 1974) cannot explain the influence of sleep loss on exercise tolerance in this study.

Most evidence points to depletion of glycogen from working muscle (Hermansen et al. 1967), or, more specifically, from slow-twitch fibers of working muscle (Costill et al. 1973), as the factor limiting prolonged heavy exercise. It is unknown if the effect of sleep loss on exercise is exerted through alterations in the rate of glycogen depletion. Even at constant $\dot{V}_{O_2}$ and $\dot{V}_{O_2}$ max, these depletion rates may be changed by a variety of factors, including pre-work activity and alterations in diet (Bergstrom et al. 1967), and a changed hormonal milieu during exercise (Costill et al. 1978). The first of these possibilities remains unlikely as an explanation of the effects of sleep loss, since subjects followed identical dietary and activity regimens before experiments after both normal sleep and sleep deprivation. Changes in the hormonal response to exercise have been found after sleeplessness (Opstad et al. 1980), but in these studies intermittent exercise and caloric deficit were superimposed upon sleep deprivation, rendering identification of the specific effects of sleep loss difficult. Others have failed to find changes in plasma levels or excretion rates of hormones such as catecholamines after sleep loss at rest (Fiorica et al. 1968). In addition, manipulation of exercise tolerance, through manipulation of the carbohydrate fraction of muscle metabolism, results in measurable changes in respiratory quotient (Costill et al. 1978). Such changes were not found in this study, suggesting that major shifts in the relative usage of the various metabolic fuels during exercise was not a result of sleep deprivation.

The absence of obvious changes in either the relative use of carbohydrate and non-carbohydrate fuels, or in the total energy cost of exercise, suggests that other factors may be responsible for reduced exercise tolerance after sleep loss. Most prominent among these is the increased perceived exertion found during exercise after sleep loss. The available evidence suggests that such perceptual changes could lead directly to decreased endurance exercise tolerance. Previous studies indicate that altered psychological states, such as neurosis, anxiety, and depression, tend to be associated with inaccurate assessment of the severity of exercise, and with reduced exercise tolerance (Morgan 1973a).

These considerations do not rule out the possibility that changes in other physiological variables, unmeasured in this study, produced both increased perceived exertion in, and reduced tolerance of, exercise after sleep loss. However, heart rate and metabolic rate, two major physiological correlates of perceived exertion during exercise of this type (Noble et al. 1973), were unchanged by sleep loss. While a third important correlate, $\dot{V}_E$, was elevated by sleep deprivation, subjects reported elevated leg fatigue as well as increased breathing effort, during exercise after sleep loss. This result suggests that perceptual changes induced by sleep deprivation may be largely independent of the usual physiological cues.

The mechanism responsible for increased $\dot{V}_E$ during exercise after sleep loss is also unknown. $\dot{V}_{CO_2}$ was not increased in parallel with the $\dot{V}_E$ rise, so that increased metabolic demands, generally tightly correlated with $\dot{V}_E$ (Wasserman 1978), could not explain the rise. Increases in either blood lactate (Sutton and Jones 1979) or catecholamines (Heistad et al. 1972), both unmeasured in this study, could have produced the $\dot{V}_E$ increase. However, lactate would be expected to be unchanged, since exercise $\dot{V}_{O_2}$, $\dot{V}_{O_2}$ max, and respiratory

exchange ratio were unchanged (Hermansen et al. 1967). In addition, previous work shows that resting plasma catecholamines are unchanged by sleep deprivation (Fiorica et al. 1968). Another possible mechanism for increased $\dot{V}_E$ during exercise after sleep loss stems from studies showing that hypnotic suggestion of increased work load increases perceived exertion and $\dot{V}_E$ without changing metabolic rate (Morgan et al. 1973b). Thus, increased exercise $\dot{V}_E$ after sleep loss in this study may be a hyperventilatory response to increased perceived effort. A final, speculative possibility is that prolonged sleep loss may act as a direct ventilatory stimulant, in a fashion opposite from, but analogous to, the ventilatory depressant effects of sleep itself (Phillipson 1978).

In conclusion, the finding that acute sleep loss reduces exercise tolerance has clear application to a variety of practical situations. In these situations, the wide individual variation in response to sleep loss becomes important. A useful further study would investigate those presently ill-defined factors that distinguish the "resistant" from the relatively more "susceptible" individual.

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