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Slow-Wave Sleep: A Recovery Period After Exercise

Abstract. *Sleep recordings were carried out on athletes on four successive nights after completing a 92-kilometer road race. Significant increases in total sleep time and slow-wave sleep were found after this metabolic stress. The results show a definite exercise effect on sleep and support sleep-restoration hypotheses.*

Recent research has lent credence to the hypothesis that sleep and in particular slow-wave sleep (SWS) (*1*), is a recovery period for daily metabolism (*2, 3*). Evidence in support of this theory includes the synchrony of growth hormone release with SWS in humans (*4*), the suggestion that optimum conditions for anabolism prevail during sleep (*5*), and studies showing SWS duration to be proportional to preceding wakefulness (*6*). Although many other studies (*7*) have yielded supportive evidence for the theory, the prediction that daytime exercise would increase SWS has produced equivocal results (*8*). Possible reasons for these conflicting findings include the variable fitness of the subjects tested (*9*), the time during the day at which the exercise is performed (*10*), and the absolute amount of exercise (*11*). The absolute amount of exercise is relevant since it is the increase in energy expenditure during exercise over and above basal metabolism that would be expected to influence the amount of SWS. To evaluate the theory that SWS is a recovery process and to resolve the question of the effect of exercise on sleep, an experiment was carried out in which the sleep patterns of six subjects were studied after a 92-km marathon. We thought that this extreme event would highlight the effect of a large increase in energy expenditure on sleep.

All subjects (age, 18 to 26 years; mean age, 21.7 years) slept for two nonconsecutive nights (with two intervening nights) in the sleep laboratory 2 weeks before the marathon. The first of these was to allow for the "first-night effect" (*12*) and was not recorded. The second of these was used as a prerace baseline level. Sleep patterns were recorded on the night of the marathon (night 1) and for the subsequent three nights (nights 2 to 4); they were recorded again 2 weeks after the marathon as a postrace control. Of the six recorded nights, the only day on which any specific exercise had been performed was that of the extended marathon (*13*). None of the subjects were taking medication, and they did not drink alcohol or coffee on the days of the study. Sleep recordings were made in the standard manner and were scored blind by two trained scorers according to standard criteria (*14*). All six subjects had previously completed several standard marathons over the preceding year, and three had in previous years completed this extended marathon. Five of the six subjects were tested for treadmill maximum aerobic power ($\dot{V}_{O_2\ max}$) and degree of fitness 3 weeks before the marathon. Lactic acid turn point (*15*) was over 70 percent of $\dot{V}_{O_2\ max}$ for all but one of these subjects, indicating a high state of fitness. The range of $\dot{V}_{O_2\ max}$ for these five subjects was 3.56 to 4.07 liters (55.8 ± 2.2 ml per kilogram of body weight per minute, mean ± standard error of the mean). The marathon started at 0600, and the subjects required between $8\frac{1}{2}$ and $10\frac{3}{4}$ hours to complete the 92 km (average speed, 10.7 to 8.6 km/hour). Body mass of the runners decreased (despite considerable fluid intake during the marathon) from 70.2 ± 2.6 to 68.1 ± 2.7 kg [intrasubject comparisons, t (5) = 4.20, $P < .01$], and rectal temperature increased from 37.6° ± 0.15° to 39.1° ± 0.16°C [t (5) = 13.33, $P < .001$].

Environmental wet- and dry-bulb temperature ranges during the race were 10.2° to 19.2°C and 10.4° to 26.2°C, respectively, and wind velocity ranged from 0.2 to 3.5 m/sec.

The analysis of the sleep records showed no significant differences between the 2-week pre- and 2-week post-marathon nights; therefore, the mean of these two recordings is used as the baseline sleep values. The baseline values were similar to those obtained in normative studies of males of similar age (*16*).

Total sleep time increased significantly over control times on each of the four nights after the marathon [F(4, 20) = 21.3, $P < .05$] (Fig. 1). Wakefulness was greatest on the night of the marathon, perhaps because of muscle and blister pains; this result could explain why the longest sleep period occurs on the second night after the marathon. Subjective sleep ratings for the seven laboratory nights showed that four of the six subjects reported having slept best on the night 2.

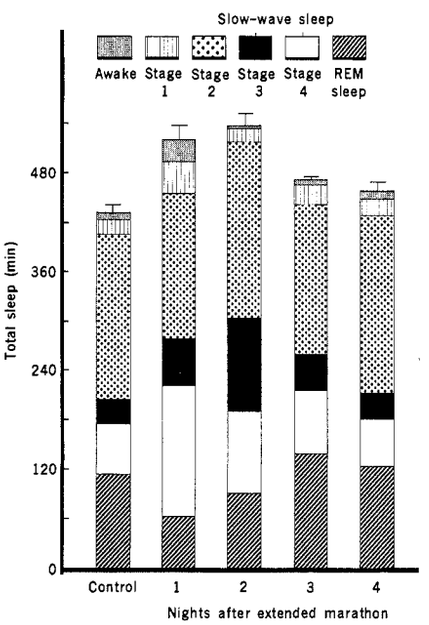

Fig. 1. Total sleep time (mean ± standard error of the mean) and mean number of minutes spent in each sleep stage on control nights, after the 92-km marathon, and on three subsequent nights.

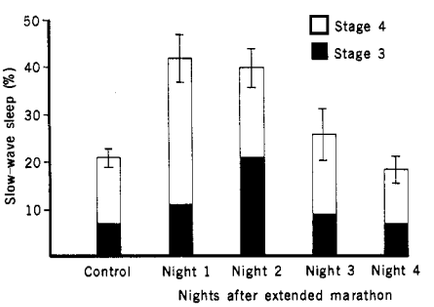

Fig. 2. Slow-wave sleep as a percentage of the total night's sleep on control nights, the night after the 92-km marathon, and on three subsequent nights.

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The percentage of SWS increased on both nights 1 and 2 (Fig. 2) [$F(4, 20) = 44.0, P < .001$]. This is predominately due to a marked increase in stage 4 (the deepest stage) on night 1 but is more equally divided between stages 3 and 4 on night 2 (Fig. 2). Significant increases were shown for both stages 3 and 4 individually on night 2, but only for stage 4 on night 1. When expressed as a percentage of total sleep time, neither SWS nor stages 3 or 4 individually was significantly raised on nights 3 and 4.

The increase in SWS on nights 1 and 2 (Fig. 1) is more remarkable because of the extended sleep period and is accompanied by a decrease in rapid eye movement (REM) sleep particularly. A relative decrease in stage 2 sleep occurred on nights 1 to 3. Sleep onset latency (timed from lights out to first appearance of stage 2) was significantly shorter on night 1 [Friedman two-way analysis of variance, $\chi_r^2(4) = 11.7, P < .05$]; stage 4 (but not stage 3) onset was also significantly shortened on night 1 [$\chi_r^2(4) = 12.1, P < .05$]. There were no changes in sleep onset latency on any of the subsequent nights.

We conclude that in a group of highly fit subjects SWS increases after an extreme metabolic load. This increase appears to have components of both time and intensity. Intensity factors are suggested by the dominance of stage 4 sleep and the decrease of stage 4 onset latency on night 1 but a surge of stage 3 on night 2. Temporal aspects are shown by the significantly raised percentages and absolute amounts of SWS on nights 1 and 2 and the decline over the next 2 days. The possibility that recovery after severe exercise in the form of SWS does not occur completely on the first night was suggested to us by a pilot study on a single subject in whom the typical decline in SWS throughout the night to very low levels toward the end of the sleep period did not occur; that is, the last third of the night after a heavy exercise load still had a high percentage of SWS. We have shown a relationship between a measured amount of exercise and SWS (*11*). Most studies in which fit subjects have been tested show an increase in SWS after exercise (*9*), but this is not the case with unfit subjects (*10*). It may be that an inadequate exercise load was used with unfit subjects, or the strain caused by an adequate load may have disrupted sleep as it did to some extent on postmarathon night 1 of this study. Increased metabolism has been shown to be related to an increase in SWS under a variety of circumstances (*17*).

The quantitative increase in total sleep time and particularly in SWS and the qualitative shift toward more stage 4 sleep immediately after metabolic stress support the theory that sleep (particularly SWS) is a recovery period for daily activity. This conclusion does not imply that restoration is an exclusive function of sleep, and several hypotheses of the function of REM sleep are still tenable (*3, 18*).

COLIN M. SHAPIRO*
RALPH BORTZ
DUNCAN MITCHELL
Department of Physiology, University of Witwatersrand, Witwatersrand, South Africa
PETER BARTEL
National Institute for Personnel Research, Braamfontein, South Africa
PIETER JOOSTE
Industrial Hygiene, Chamber of Mines of South Africa, Johannesburg

References and Notes

1. Slow-wave sleep is defined as stages 3 and 4 of non-REM sleep. It is characterized by low-frequency high-amplitude waves on the electroencephalogram. This pattern occupies 20 to 50 percent of the tracing in stage 3 and more than 50 percent of the tracing in stage 4. Stage 4 may be considered to be a more intense form of the SWS process.
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* Present address: Department of Psychiatry, Royal Edinburgh Hospital, Morningside Park, Edinburgh EH10 5HF, Scotland.

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Retroactive Interference in Discrimination Learning

Abstract. *In stage 1 of this experiment pigeons were trained to discriminate between two levels of noise or two colors by pecking on one of two disks. In stage 2 the discriminative stimuli were not presented, but pecking on the disks was rewarded on a random schedule. The second procedure caused the pigeons to forget the discrimination they had learned.*

Events that affect an organism after it has learned something new may cause it to forget what it learned. This retroactive interference (RI) has been studied in many experiments with human subjects. In most of these, the event considered the possible cause of forgetting is a second learning experience. The usual experimental procedure involves two groups of subjects and three treatment phases. During phase 1 the two groups are trained identically, for example, to recite a list of words or nonsense syllables. During phase 2 the control group rests while the experimental group learns a second task, and during phase 3 both groups are tested for retention of what they learned in phase 1.

Numerous experiments on RI have also been done with animal subjects. Most of the more recent ones have been concerned with the effects of events that intervene between the presentation of a single stimulus and the opportunity to make a learned response to that stimulus (the delayed matching-to-sample paradigm) (*1*). Such experiments differ from the work with humans in two important ways. (i) They deal with short-term rather than long-term memory, and (ii) the postlearning event examined as a possible cause of forgetting is usually a simple

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