EFFECTS OF EXERCISE, BEDREST AND NAPPING ON PERFORMANCE DECREMENT DURING 40 HOURS

A. LUBIN
D. HORD
M. L. TRACY
L. C. JOHNSON

REPORT NO. 75-43

NAVAL HEALTH RESEARCH CENTER
SAN DIEGO, CALIFORNIA 92152

NAVAL MEDICAL RESEARCH AND DEVELOPMENT COMMAND
BETHESDA, MARYLAND

PSYCHOPHYSIOLOGY
Vol. 13, No. 4

Printed in U.S.A.

Effects of Exercise, Bedrest and Napping on Performance Decrement During 40 Hours

A. LUBIN, D. J. HORD, M. L. TRACY, AND L. C. JOHNSON
Naval Health Research Center, San Diego

ABSTRACT

Young male Naval volunteers were denied normal nocturnal sleep and maintained on a 60-min treatment–160-min testing schedule during 40 consecutive hrs. Ten subjects bicycled, 20 subjects controlled EEG activity during bedrest, and 10 subjects napped. Eight measures of addition, auditory vigilance, mood, and oral temperature were obtained. The Bedrest group showed significant impairment on all eight measures, and thus, gave no support to the *forced-rest* theory of sleep function. The Exercise group was worse than the Nap and Bedrest groups for all measures. In spite of fragmented, reduced sleep (about 3.7 hrs per 24 hrs), the Nap group had no impairment on six of the measures. The results suggest that exercise increases the impairment due to sleep loss, and naps reduce or remove this impairment. Bedrest is not a substitute for sleep.

DESCRIPTORS: Sleep loss, Performance, Mood, Naps, Exercise.

The purposes of this study were to answer two empirical questions and to test the *forced-rest* theory of sleep function. The empirical questions were: (1) Do naps prevent sleep loss decrement in performance when subjects are deprived of normal nocturnal sleep? (2) Does exercise increase or decrease the impairment of performance and mood during sleep loss?

An early version of the forced-rest theory of sleep function has been traced back to Claparede (1905) by Webb (1974): "It is not because we are intoxicated or exhausted that we sleep: instead we sleep in order to not be that way" (translation by Webb and Lubin). In Webb's version, the primary postulate is that survival requires periods of non-responding. Sleep is an active process which prevents the organism from responding when predator-prey relationships make activity dangerous, or when foraging for food would be inefficient. The existence of both hibernation and estivation among animals lends evidence to the supposition that a decrease in metabolic rate at appropriate times is an important survival mechanism. The forced-rest theory denies that the primary function of sleep is restorative (Webb, 1974).

Zepelin and Rechtschaffen (1974) regard it as a fact that: "sleep in effect sets a ceiling on metabolic expenditures by limiting the amount of time available for activity. The enforcement of rest . . . appears to be the principal contribution that sleep makes in the regulation of energy expenditure [p. 454]." Due to the failure to isolate hypothesized somnotoxins, Berger, Taub, and Walker (1973) also have espoused the forced-rest theory of sleep function. *The simple version of the forced-rest theory makes sleep unnecessary when the subject remains quiet in bed.*

The common-sense view of napping is that it benefits and refreshes any subject who is suffering from sleep loss and/or fatigue. But the substitution of scheduled naps for monophasic nocturnal sleep disrupts the usual sleep/wake pattern. Two studies have concluded that *any* alteration in established sleep patterns, even an increase in the normal amount of sleep, is deleterious to performance and mood (Taub & Berger, 1974a, 1974b).

The common-sense view of exercise is that it provides a short-term arousal from sleep loss but continued exercise combined with sleep loss leads to profound deterioration in mood and performance. Yet the only published study to date concluded that during sleep loss the effect of exercise was no differ-

This research was supported by the Advanced Research Projects Agency of the Department of Defense under Order No. 1596, and by the Department of the Navy, Bureau of Medicine and Surgery, under Work Unit M4305.07-3008DAC5. The views and conclusions contained herein are those of the authors and should not be interpreted as necessarily reflecting the official policies, either expressed or implied, of the Defense Advanced Research Projects Agency, the Department of the Navy, or the U. S. Government.

The authors thank Julie Moses, Bill Jensma, Ray Hilbert, Don Irwin, Marion Austin, Korey Greene, Gary Howell, and Matt Sinclair for assistance in the planning and execution of this study.

Address requests for reprints to: Ardie Lubin, Ph.D., Naval Health Research Center, San Diego, California 92152.

ent from that of bedrest on word list memory, complex additions, and auditory vigilance. For a simple (successive single digit) addition task, exercise had a significantly beneficial effect (Webb & Agnew, 1973). We felt that these unexpected results deserved another look. Our study was designed from an applied point of view in an attempt to answer the following question: given that there is some emergency which imposes at least one night of sleep loss on a subject, what is the best regime to follow before and during sleep loss which minimizes sleep loss impairment?

Method

Subjects

Forty male volunteers (19–28 yrs, mean age = 21) from the Naval Hospital Corps School were chosen on the basis of their willingness to spend Monday through Friday in the lab, and stay awake for 40 successive hrs. Details on their briefing and debriefing have been given in a previous article (Hord, Lubin, Tracy, Jensma, & Johnson, 1976). Informed consent was obtained.

Experimental Design

There were three groups. Ten subjects were allocated to the Nap condition and followed a 220-min cycle of 60-min nap–160-min wake for 10 epochs. The 20 subjects of the Bedrest group followed the same schedule, but tried to regulate their brain waves during their 10 hrs in bed. The 10 subjects in the Exercise group followed the same regimen, but bicycled during the 10 60-min treatment sessions instead of sleeping or resting in bed.

Apparatus

Details of the feedback apparatus used by the Bedrest group are given in another article (Hord et al., 1976).

EEG feedback designed to increase theta activity was given to the Nap subjects to aid sleep onset. The supposed soporific effect of theta enhancement was not tested in this study.

Heart rate was fed back to the Exercise subjects during stationary bicycling. A PDP-12 computer measured each successive R-to-R interval (in msec) and then computed heart rate in bpm. A TV display of the rate associated with each successive heart beat was provided to the exercising subject.

EEG was recorded for Nap subjects as described by Moses, Lubin, Naitoh, and Johnson (1972). Eye movements were recorded by the common mode rejection technique (Hord, 1975). For all 40 subjects, baseline night sleep and recovery night sleep were recorded similarly. Sleep records were scored according to the Rechtschaffen and Kales Manual (1968).

Oral temperature was measured by an electronic thermometer (IVAC, Model 811).

Procedure

The schedule is displayed on the abscissa of Fig. 1. Monday and Tuesday were orientation and practice days. The first experimental session (bedrest, exercise, or nap) started at 0800 on Wednesday morning. The basic unit for treatment and testing was a 220-min epoch. Epoch 1 to epoch 10 occupied 36-hrs and 40-min of the 40-hrs; a total of 3-hrs and 20-min was set aside for breaks and meals. All subjects were awakened at 0600 Wednesday and put to bed at 2200 Thursday. All subjects were awakened between 0600–0630 on Friday. Epochs 11 and 12 (E11 and E12) followed immediately after one full night of recovery sleep.

The 20 subjects of the Bedrest group were given feedback on their EEG activity; 10 were given positive feedback for augmenting alpha activity, and 10 were given positive feedback for suppressing alpha and theta. Subjects were run in pairs so that one subject was receiving feedback for augmenting alpha at the same time that his control was receiving feedback for supressing alpha and theta. Their EEG activity was monitored closely for signs of drowsiness as defined by Rechtschaffen and Kales (1968), and they were not permitted to sleep. Thus, the Bedrest subjects were forced to rest physically but maintained enough mental activity to stay awake. Most Bedrest subjects displayed drowsiness from E7 to E10 and were roused one or more times. Statistical analysis showed no performance or mood differences between the high alpha and low alpha-theta groups (Hord et al., 1976) so both were combined into the Bedrest group. The alpha enhancement group produced significantly greater amounts of alpha activity up through E6.

Exercise and Nap subjects were also run in pairs. The Exercise subject pedaled 1 mile every 12-min for a total of 5 miles while the Nap subject was allowed to sleep. Exercise subjects were instructed to raise their heart rate to 50% above baseline and to hold this level while pedaling. Immediately after lights-on, the Nap subject was roused and pedaled 1 mile in 5-min to eliminate the sleep inertia that usually accompanies waking.

Measures

All 40 subjects were tested in exactly the same way. Following the first hour of nap, exercise, or bedrest, the 40-min Wilkinson Auditory Vigilance test was given and oral temperature was taken. This was followed by 40-min of the Wilkinson Addition test (Wilkinson, 1969). On the even-numbered epochs (E2, E4, etc.) the immediate recall Williams Word Memory Test (Williams, Gieseking, & Lubin, 1966) was given with a digit-span task between the list presentation and the recall (to prevent rehearsal). On odd-numbered epochs, the Profile of Mood States (McNair, Lorr, & Droppleman, 1971) was given. The Stanford Sleepiness Scale (Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973) preceded and followed every 1-hr treatment session. The Nap subjects filled out this scale before exercising.

All of these tasks have been shown in previous studies to be sensitive to one night of sleep loss when 10 or more subjects are used (Lubin, Moses, Johnson, & Naitoh, 1974).

Results

Fig. 1 gives the averages for each epoch for three of the measures. Oral temperature clearly shows the usual circadian cycle under all three treatments. The treatments seem to have an additive effect, i.e., they change the level but not the wave-form of the circadian cycle. Presumably this shows that the treatments have direct physiological effects in addition to any psychological sequela.

For number of correct additions and reported sleepiness, the extreme scores tend to occur at epoch 7, following the lowest oral temperature at about 0500, but there is an apparent non-additive interaction between treatment effects and circadian cycle.

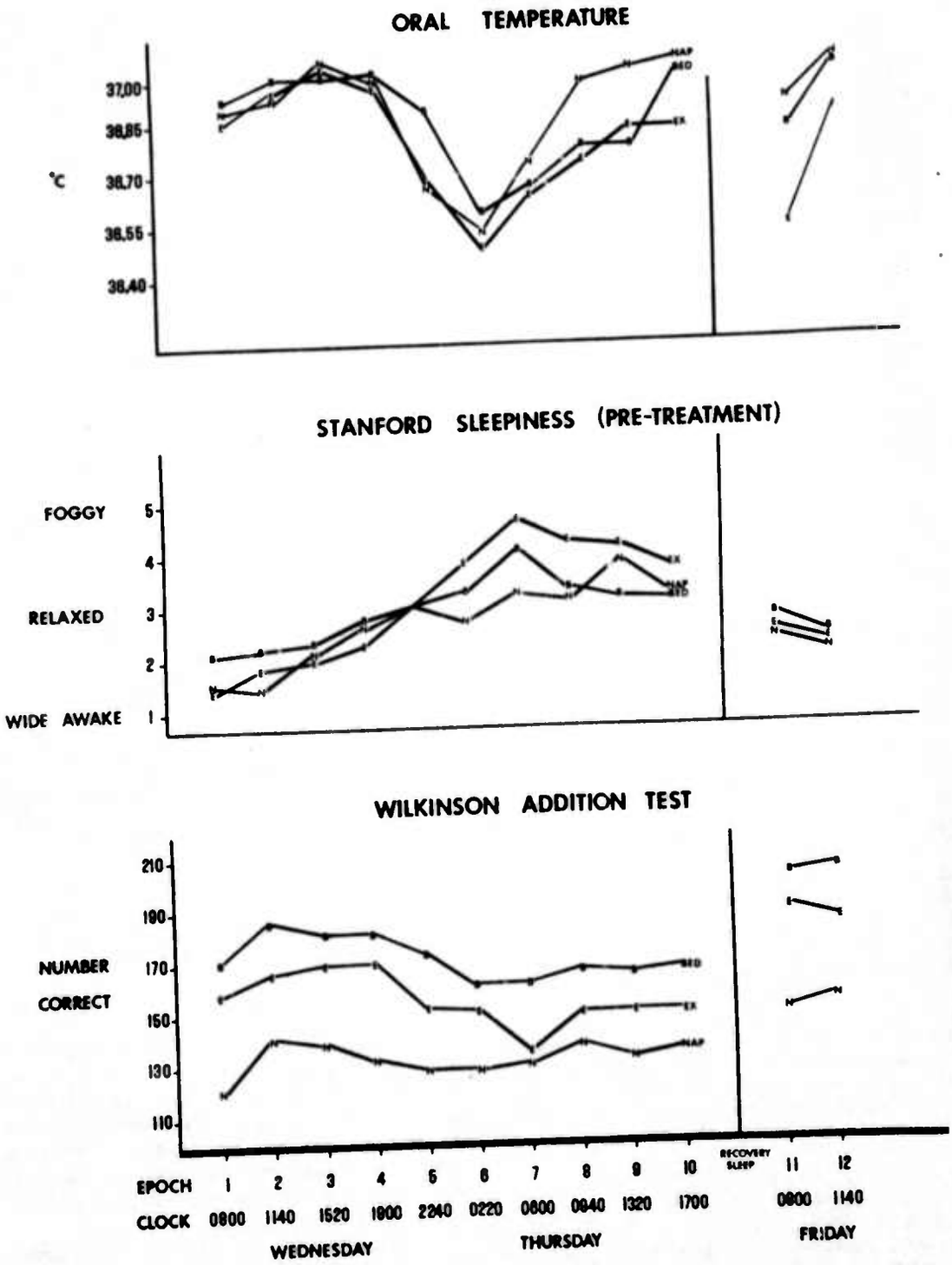

Fig. 1. Averages for oral temperature, reported sleepiness, and number of correct additions over epochs for the three groups.

TABLE 1

Sleep Loss Contrast scores for exercise, bedrest, and nap groups

Measures	Means (Standard Deviations)		
	N=10 Nap	N=20 Bedrest	N=10 Exercise
Wilkinson Auditory Vigilance			
Percent of Correct Hits	51.3 (119)	87.4 (59)*	124.8 (91)*
Errors of Omission	11.6 (35)	22.6 (14)*	30.3 (29)*
Wilkinson Addition			
Percent correct	−5.6 (31)	5.1(10)*	21.5 (22)*
Number correct	−7.4 (121)	83.9 (128)*	107.0 (145)*
Number attempted	1.0 (112)	80.7 (131)*	84.8 (153)
Oral Temperature (°C)	.4 (1.2)	.8 (1.3)*	1.1 (1.0)*
Stanford Sleepiness Scale	16.8 (7)*	14.7 (11)*	26.7 (8)*
Williams Word Memory	8.2 (9)*	3.3 (6)*	11.5 (8)*

Note. The SLC transform multiplies the original score by six, except for Williams Word Memory, which is multiplied by two.

*Significantly greater than zero at .05 level or better, one-tail.

Table 1 gives the numerical summary. For statistical purposes, each set of 10 epoch scores was transformed into a Sleep Loss Contrast (SLC) score, which maximized the effects of sleep loss and fatigue using each subject as his own control. For most of the measures,

$$SLC = 2(E1+E2+E3)-(E7+2E8+2E9+E10).$$

The Williams Word Memory was given only on even-numbered epochs, so an SLC score of (E2+E4)−(E8+E10) was used. For sleepiness ratings and errors of omission on the Wilkinson Auditory Vigilance Test the sign of the SLC was reversed. Therefore a positive SLC score always implies that the subject did worse under sleep loss than during the first 3 baseline epochs. When all 7 (or 4) epoch scores are equal, the expected value of the SLC score is zero. Epochs 4, 5, and 6 could not be matched for time-of-day with the baseline epochs and therefore were not used. For the Stanford Sleepiness Scale, only the scores preceding each treatment session were used, to avoid the nap inertia effect.

The SLC transforms do a fairly good job of minimizing any slow changes due to time-of-day, but learning and practice effects, when they exist, tend to make the SLC score an under-estimate of the true sleep loss effect. Boredom would make the SLC score an over-estimate. However, the differences between the three group means are free of the effects of practice or boredom, as well as any simple interactions of circadian cycle and learning with sleep loss. Higher-order interactions involving the three treatments cannot be distinguished from the direct treatment effect.

Originally we planned to combine E11 and E12 with E1 and E2 to determine treatment-free levels. However, not all measures came back to baseline after recovery sleep. Since the experimental design did not permit exact assessment of possible carry-over effects of treatment, testing, learning, fatigue, etc., only pre-recovery data were used.

The Profile of Mood States was not used since we found that the standard measures (Vigor, Fatigue, etc.) as well as measures specially devised to emphasize the effects of sleep loss, had less validity than the Stanford Sleepiness Scale, and did not increase validity significantly when multivariate weights were used to get an optimal linear composite.

Did the Nap group get much sleep? Over the 40-hr vigil, the maximum sleep time possible was 600-min. The average sleep time was 366-min (±95), or 61% of available sleep time. So, the average subject got 6.1 hrs of sleep. Ordinarily, we would not expect this reduction to damage performance (Wilkinson, 1969). A detailed account of the Nap sleep measures has been presented elsewhere (Moses, Hord, Lubin, Johnson, & Naitoh, 1975).

Table 1 shows little decrement for the Nap group on 6 of the 8 measures. The Exercise group had a significant sleep loss effect on 7 of the 8 measures, and the Bedrest group displayed a significant sleep loss effect on all measures. The Exercise group always shows the most damage, with six of the eight measures displaying the expected rank order: Nap with the least decrement, the Bedrest group with more impairment, and Exercise with the most. The exceptions are the Stanford Sleepiness measure and the Williams immediate recall score, to be discussed later.

Disregarding tests of significance (for the moment) the trend indicates that exercise potentiates the sleep loss effect. However, what do the between-group tests of significance show? When the Student *t*-test and the Wilcoxon rank-sum test were applied, the Exercise group showed *significantly* more impairment than the Bedrest group on only three measures—percent correct additions, Stanford Sleepiness, and Williams Word Memory. The Exercise group showed *significantly* more impairment than the Nap group on percent correct additions, number of correct additions, and Stanford Sleepiness. The Bedrest group showed *significantly* more impairment than the Nap group on only two measures—the number of correct additions and the number of attempted additions.

The Stanford Sleepiness and Williams Word Memory measures are anomalies in several ways. First, the Nap group shows significant impairment on only these two scores. In fact, the increase in reported sleepiness meets the Dunn-Bonferroni criterion (Dunn, 1959), a conservative significance test which takes into account the fact that eight correlated *t*-ratios were computed. Furthermore, the Bedrest group is better than the Nap group on both of these measures, although not significantly so.

Discussion

The Forced-Rest Hypothesis

Webb and Agnew (1973) used a cross-over design to test the forced-rest hypothesis on 8 subjects by comparing the Bedrest condition during 2 days of sleep loss to baseline performance (as well as an exercise condition). Three of their tasks were the same as ours: Williams Word Memory, Wilkinson Addition, and Wilkinson Auditory Vigilance. Their results show impaired performance on the addition and vigilance tasks for the bedrest condition. Essentially, our results confirm those of Webb and Agnew, and we can report, moreover, that Williams Word Memory, Stanford Sleepiness, and oral temperature showed clear effects of sleep loss in the Bedrest group. Therefore, there is no empirical support for the forced-rest theory from these two studies of the bedrest condition. While these experiments demonstrate that *forced-rest is not a short-term substitute for sleep*, they are far from disposing of the theory.

The evolutionary advantage gained by early mammals from regular sleep may have caused genetic control over sleep which is relatively independent of feedback from the activity of the organism. Or sleep could be controlled by aspects of organismic activity quite far removed from measures such as metabolic rate, in the same way that breathing tends to be controlled by CO_2 rather than O_2. Unfortunately, the forced-rest theory is a peculiarly passive postulate, predicting very little from its truth or falsehood, so that crucial tests are hard to devise.

Exercise and Sleep Loss

Webb and Agnew (1973) found no tendency for bedrest-plus-sleep-loss to give better performance than exercise-plus-sleep-loss. In fact, for a simple addition task (the Plus Seven), they report that exercise was compensatory compared to bedrest. Our Exercise group always showed more sleep loss impairment than the other groups, differing significantly from the Bedrest group on percent of correct additions, reported sleepiness, and Williams immediate recall score. The last two measures were significant by the conservative Dunn-Bonferroni test (Dunn, 1959).

So we conclude that exercise definitely increases performance decrement and sleepiness due to sleep loss. The Webb-Agnew study differs from ours in many ways. Their unexpected finding of a neutral or compensatory effect of exercise may be due to an interaction of testing order with treatments—the same 8 subjects were run through all three treatments. This crossover design assumes there is no interaction of carryover effects with treatment. Webb's explanation (personal comments) is quite different. He reports that by the second night of sleep loss his subjects looked forward to the Exercise session as a means of rousing themselves.

Napping during a Vigil

Taub and Berger (1974a, 1974b) have presented convincing evidence that many changes in the usual monophasic sleep pattern are deleterious. Our cycle (60-min sleep–160-min wake) certainly fragments sleep, and, at times, is directly opposed to the circadian temperature cycle. Nevertheless, the 60-min Nap sessions were beneficial, except for immediate recall and reported sleepiness.

A recent study by Carskadon and Dement (1975) used the Stanford Sleepiness Scale. Five subjects were placed on a 30-min sleep–60-min wake regimen for 5 days. There was a significant increase in reported sleepiness on the first nap day, but the sleepiness measure actually decreased almost to baseline over the next 4 days. This confirms our finding that napping during the first day does not alleviate feelings of drowsiness.

The sleep of our Nap subjects on the first recovery night gives no hint of sleep loss. If anything, they took longer to fall asleep (time from lights-out to the first Stage 2) than they did on the baseline night, and there are no significant changes in Stages 3-4, Stage REM, or Stage 2. But the Carskadon and Dement subjects showed the typical signs of sleep loss: large increases in Stages 3-4 with decreases in Stages 1,

2, and REM. Presumably, the shortened sleep of our subjects (about 3.7-hrs per 24-hrs) did not cut significantly into their safety margin, whereas 5 days of napping (with about 5.0-hrs of sleep per 24-hrs) wiped out the sleep reserve for the Carskadon-Dement subjects, even though some adaptation to reduced sleep took place.

Conclusions

Our results do not support the forced-rest theory of sleep function. As in the Webb-Agnew study, bedrest did not take the place of sleep. One-hr naps every 220-min did neutralize the loss of normal nocturnal sleep except for the immediate recall of word lists and complaints of sleepiness. Contrary to the Webb-Agnew reported results, exercise during sleep loss increased impairment on all measures, the hardest hit being accuracy of addition, immediate recall, and sleepiness.

REFERENCES

Berger, R. J., Taub, J. M., & Walker, J. M. Sleep as a biological adaptive process. In U. J. Jovanovic (Ed.), *The nature of sleep*. Stuttgart: Fischer, 1973. Pp. 252–255.

Carskadon, M. A., & Dement, W. C. Sleep studies on a 90-minute day. *Electroencephalography & Clinical Neurophysiology*, 1975, *39*, 145–156.

Claparede, E. Esquisse d'une theorie biologique du sommeil. *Archives de Psychologie*, 1905, *4*, 245–349.

Dunn, O. J. Confidence intervals for the means of dependent normally distributed variables. *Journal of the American Statistical Association*, 1959, *54*, 613–621.

Hoddes, E., Zarcone, V., Smythe, H., Phillips, R., & Dement, W. C. Quantification of sleepiness: A new approach. *Psychophysiology*, 1973, *10*, 431–436.

Hord, D. J. Common mode rejection techniques in conjugate eye movement recording during sleep. *Psychophysiology*, 1975, *12*, 354–355.

Hord, D. J., Lubin, A., Tracy, M. L., Jensma, W., & Johnson, L. C. Feedback for high EEG alpha does not maintain performance during sleep loss. *Psychophysiology*, 1976, *13*, 58–61.

Lubin, A., Moses, J. M., Johnson, L. C., & Naitoh, P. The recuperative effects of REM sleep and Stage 4 sleep on human performance after complete sleep loss: Experiment 1. *Psychophysiology*, 1974, *11*, 133–146.

McNair, D. M., Lorr, M., & Droppleman, L. F. *Manual for the POMS*. San Diego, CA: Educational & Industrial Testing Service, 1971.

Moses, J., Hord, D. J., Luhin, A., Johnson, L. C., & Naitoh, P. Dynamics of nap sleep over 40 hours. *Electroencephalography & Clinical Neurophysiology*, 1975, *39*, 627–633.

Moses, J., Luhin, A., Naitoh, P., & Johnson, L. C. Reliability of sleep measures. *Psychophysiology*, 1972, *9*, 78–82.

Rechtschaffen, A., & Kales, A. (Eds.) *A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects*. Washington, D.C.: Public Health Service, U.S. Government Printing Office, 1968.

Taub, J. M., & Berger, R. J. Acute shifts in the sleep-wakefulness cycle: Effects on performance and mood. *Psychosomatic Medicine*, 1974, *36*, 164–173. (a)

Taub, J. M., & Berger, R. J. Effects of acute shifts in circadian rhythms of sleep and wakefulness on performance and mood. In L. E. Sheving, F. Halberg, & J. E. Pauly (Eds.), *Chronobiology: Proceedings of the International Society for the Study of Biological Rhythms*. Tokyo: Igaku Shoin, Ltd., 1974. Pp. 571–574. (b)

Webb, W. B. Sleep as an adaptive response. *Perceptual & Motor Skills*, 1974, *38*, 1023–1027.

Webb, W. B., & Agnew, H. W., Jr. Effects on performance of high and low energy-expenditure during sleep deprivation. *Perceptual & Motor Skills*, 1973, *37*, 511–514.

Wilkinson, R. T. Sleep deprivation: Performance tests for partial and selective deprivation. In L. E. Abt & B. F. Riess (Eds.), *Progress in clinical psychology*. Vol. 8. New York: Grune & Stratton, 1969. Pp. 28–43.

Williams, H. L., Gieseking, C. F., & Lubin, A. Some effects of sleep loss on memory. *Perceptual & Motor Skills*, 1966, *23*, 1287–1293.

Zepelin, H., & Rechtschaffen, A. Mammalian sleep, longevity and energy metabolism. *Brain, Behavior, & Evolution*, 1974, *10*, 425–470.

(Manuscript received May 28, 1975; revision received January 15, 1976; accepted for publication January 19, 1976)

Unclassified

SECURITY CLASSIFICATION OF THIS PAGE (When Date Entered)

REPORT DOCUMENTATION PAGE

READ INSTRUCTIONS BEFORE COMPLETING FORM

1. REPORT NUMBER: 75-43
2. GOVT ACCESSION NO.:
3. RECIPIENT'S CATALOG NUMBER:

4. TITLE (and Subtitle): Effects of Exercise, Bedrest and Napping on Performance Decrement during 40 hours.

5. TYPE OF REPORT & PERIOD COVERED:

6. PERFORMING ORG. REPORT NUMBER: M4305.5-8077

7. AUTHOR(s): Ardie Lubin, David J. Hord, Mary L. Tracy and Laverne C. Johnson

8. CONTRACT OR GRANT NUMBER(s): M4305.07-3008DAC5, ARPA Order - 1596

9. PERFORMING ORGANIZATION NAME AND ADDRESS: Naval Health Research Center, San Diego, California 92152

10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS:

11. CONTROLLING OFFICE NAME AND ADDRESS: Naval Medical Research and Development Command, Bethesda, Maryland 20014

12. REPORT DATE: 28 May, 1975

13. NUMBER OF PAGES: 7 (13) 90

14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office): Bureau of Medicine and Surgery, Department of the Navy, Washington, D.C. 20372

15. SECURITY CLASS. (of this report): Unclassified

15a. DECLASSIFICATION/DOWNGRADING SCHEDULE:

16. DISTRIBUTION STATEMENT (of this Report): Approved for public release; distribution unlimited

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report):

18. SUPPLEMENTARY NOTES:

19. KEY WORDS (Continue on reverse side if necessary and identify by block number):
Sleep loss
Performance
Mood
Naps
Exercise

20. ABSTRACT (Continue on reverse side if necessary and identify by block number):
Young male Naval volunteers were denied normal nocturnal sleep and maintained on a 60-min treatment—160-min testing schedule for 40 consecutive hours. Ten subjects bicycled, 20 subjects controlled EEG activity during bedrest, and 10 subjects napped. Eight measures of addition, auditory vigilance, mood, and oral temperature were obtained. The Bedrest group showed significant impairment on all eight measures, and thus, gave no support to the forced rest theory of sleep function. The Exercise group was worse than the Nap and →

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE
S/N 0102-014-6601

Unclassified 391 642
SECURITY CLASSIFICATION OF THIS PAGE (When Date Entered)

Bedrest groups for all measures. In spite of fragmented, reduced sleep (about 3.7 hours per 24 hours), the Nap group had no impairment on six of the measures. The results suggest that exercise increases the impairment due to sleep loss, and naps reduce or remove this impairment. Bedrest is not a substitute for sleep.