SLEEP AND PERFORMANCE IN YOUNG ADULTS AND OLDER NORMALS AND INSOMNIACS DURING ACUTE SLEEP LOSS AND RECOVERY *

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Many changes occur in sleep as a function of aging, but it is not known whether these changes result in sleep being less restorative. To examine the sleep restorative process, groups of 12 normal young adults and 12 normal and 12 insomniac male subjects, age 55–71, were totally sleep deprived for 64 hours and then allowed recovery sleep. Response speed, immediate recall, sleepiness, and body temperature were tested at approximately 2300, 0115, 0330, 0530 and 0800 during baseline, sleep loss, and recovery nights. Significant group (age or insomnia) by sleep loss condition interactions were found for reaction time and immediate recall performance measures. Similar significant interactions were found for oral temperature and all EEG sleep variables except total time in bed, percent stage 1, and percent REM. It was concluded that performance recovery following sleep loss was no slower in older subjects than in younger subjects despite very different recovery sleep stage parameters. This implied that aging effects on sleep are developmental rather than degenerative.

1. Introduction

In recent years an increasing number of studies have focused on changes in the sleep process as a function of aging. Many studies have shown a general disintegration of the sleep process including decreases in slow wave sleep (SWS) and increases in nocturnal awakenings and wake time. In males, these changes may begin as early as the fourth decade (Williams, Karacan, & Hursch, 1974) and are clearly evident in the sleep of 55-year-old males. Williams et al. (1974) and others (Webb & Levy, 1982) have also commented

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on increases in variability in total sleep requirement and sleep distribution in older individuals. Other investigators have hypothesized that these changes may be related to tremendous increases in sleep disruptive respiratory and muscular events (Ancoli-Israel, Kripke, Mason, & Messin, 1981; Carskadon, Brown, & Dement, 1982) that occur as a function of aging.

Changes in alertness, memory, and general performance are so common in elderly individuals that they have long been considered "normal". However, these symptoms are also consistent with those seen in young adults after sleep loss (Johnson & Naitoh, 1974) or sleep disruption (Bonnet, 1985b). As a result, it is important to understand the correlation between sleep and performance in aging individuals. The effect of altered patterns of sleep on performance can be studied effectively either by comparing respective groups of older individuals with varying degrees of sleep impairment (Carskadon et al., 1982) or by subjecting a group of older individuals to a sleep stress such as sleep deprivation and comparing the resulting loss of function to that seen in younger individuals in similar conditions. These comparisons are important because the normal sleep changes that occur with age are often defined as insomnia and treated long-term with medications which themselves may have associated risks (Bonnet & Kramer, 1981a; Bonnet & Kramer, 1981b). The use of such medications may be justified if the sleep changes accompanying aging can be associated with functional decrements, such as impaired performance or mood. However, if correlated decrements do not exist, then the use of such medication is questionable.

A number of studies have documented the effects of both chronic (Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973; Webb, 1985) and acute (Rosa, Bonnet, & Warm, 1983) loss of sleep in decreasing performance efficiency and mood and increasing sleepiness in young adult subjects. These effects have generally been replicated in the only two sets of studies (Webb & Agnew, 1974; Webb, 1981; Froberg, Karlsson, Levi, & Lidberg, 1972) which have examined sleep loss in 40–50 year-old participants. However, the studies of sleep loss in middle age males have come to opposite conclusions. Whereas one found decreased performance accuracy on baseline and less sensitivity to sleep loss in the older subjects as compared to younger subjects (Froberg et al., 1972), the other found both higher performance output and greater accuracy in the older subjects on baseline, and increased sensitivity to sleep loss (Webb & Agnew, 1974). These effects may have reflected the use of military personnel in the first study and university faculty (vs. newspaper responders) in the second. The differences may also be explained by the fact that the groups showing highest performance levels on baseline have shown the greatest performance loss during deprivation.

Very recently, three groups have examined the effects of sleep deprivation in groups of normal older individuals age 50–70 years (Bonnet, 1985a; Bonnet, 1986; Carskadon & Dement, 1985; Webb, 1985). The studies all examined
sleep, sleep deprivation (38–64 hours), and recovery. In general, all the studies agreed that older subjects became sleepier and performed more poorly as sleep deprivation continued. Baseline sleep was representative of that normally seen in older individuals. Recovery sleep resulted in increases in total sleep time and slow wave sleep on the first recovery sleep night. Two of the studies (Bonnet, 1985a; Carskadon & Dement, 1985) indicated that the older subjects might actually have been less impacted by sleep loss than young adults, but neither study presented comparable young adult data. The third study (Webb, 1985) reported that older subjects had less subjective decrement associated with sleep loss but had generally increased performance loss. Unfortunately, this study compared daytime baseline performance with nighttime sleep loss performance and, therefore, confounded circadian time and sleep loss.

The present study sought to examine the effect of 64 hours of sleep loss and recovery in carefully selected groups. Groups of normal young adults without sleep disorders were compared to (a) a group of normal older males screened to have normal sleep efficiency and to be free of disruptive sleep pathology, and (b) a group of older males complaining of long-term insomnia not related to periodic leg movements or sleep apnea. Older subjects were specifically chosen in the 55–70 year-old range to assure that age-related decreases in SWS had occurred. Repeated performance, mood, auditory arousal threshold and oral temperature observations were made during baseline sleep conditions, sleep deprivation, and recovery sleep in an attempt to document the magnitude of performance loss and the rate of performance recovery in the three groups. It was hypothesized that if the sleep stage changes which occur with aging are degenerative (as opposed to developmental), then the pattern of performance restoration during sleep should be faster in normal younger than older subjects. It was also hypothesized that if decreases in total sleep attainable per 24 hours are a degenerative disease called insomnia (as opposed to a normal phenomenon), then the pattern of performance restoration during sleep in chronic insomniacs (without sleep pathology or psychopathology) would be different from both young adults and older normal sleepers.

2. Method

2.1. Subjects

Twenty-four males aged 55–71 and twelve males aged 18–28 were chosen based upon their written responses to a Selected Medical History, Sleep Behavior Questionnaire, Brief Michigan Alcohol Screening Test, Brief Drug Abuse Screening Test and depression scale from the MMPI. The Normal Young and Older Normal subjects reported normal sleep and a habitual 6–9 hour sleep routine including usual sleep latencies of less than 30 min and two
or fewer awakenings per night. Subjects in the Older Insomnia group reported having chronic insomnia for two or more years and habitually tried to sleep 6–9 h but reported spending more than 60 min awake each night. For all groups, subjects were required to be infrequent nappers, to have no obvious symptoms of sleep apnea or nocturnal myoclonus, to have no history of alcohol or drug abuse, and to score below the 75th percentile on the depression scale of the MMPI. Subjects were not allowed to use psychotropic medication within two weeks of the experiment, and potential subjects with uncontrolled medical illnesses were excluded from the study. However, many subjects were using medications of long duration and primarily for hypertension or gout. Subjects meeting these criteria and without significant uncorrected vision or hearing loss were invited to the laboratory for an explanation of the study. At this time an informed consent was completed, and subjects practiced all tests and questionnaires used in the study.

Subjects who agreed to participate in the study were scheduled for an adaptation/screening night. In the young group (YN), evidence of any sleep disorder would have excluded the subject (but no disorders were found). Older subjects had airflow, chest movement, EKG, leg EMG, and usually percent SaO2 recorded. Any participant with sleep apnea or nocturnal myoclonus syndrome (ASDC, 1979) was excluded. Seven potential normal subjects were so excluded (two for apnea and five for myoclonus), and five potential insomniac subjects were so excluded (one for apnea and four for myoclonus). Further, older normals (ON) were required to have a sleep efficiency (total time asleep divided by time in bed) of greater than 85% whereas older insomniacs (OI) were required to have sleep efficiency of less than 80%. Subjects were scheduled to sleep during their normal hours in bed as reported on their Sleep Behavior Questionnaire. Subjects who awakened sol before their normal time of arising were asked to remain in bed and try to sleep if possible. The time was used in the calculation of sleep efficiency. Of potential subjects screened, eight potential normal subjects were disqualified for having a sleep efficiency that was too low and seven potential insomniac subjects were disqualified for having a sleep efficiency that was too high.

2.2. Performance and physiological measures

2.2.1. Performance

In all groups, performance was assessed with the Williams Word Memory Test of immediate free recall (Williams, Geiseking, & Lubin, 1966) and with a simple auditory reaction time measure (Lisper & Kjellberg, 1972). For word memory, two lists of 15 one- or two-syllable nouns were presented via tape player at each testing session. The subject was requested to write each word as he heard it and then rewrote (in any order) as many words as he could recall within a 1–2 min time period. The score was the sum of the words recalled
from both lists. For reaction time, stimulus tapes as described by Lisper and Kjellberg (1972) were played for 10 min at each test period. Stimuli were 1000 Hz tones, 300 ms in duration, presented via cassette tape player through headphones at a level of 60 dB (SPL) at both ears. The mean interstimulus interval was 4s with a variance of 2s, and four different stimulus tapes were randomly used. Reaction times were recorded in ms on a digital clock interfaced with a printer. The score for each testing period was the medium reaction time for the entire 10 min. For statistical analysis, these scores were transformed to their reciprocals (Myers, 1979) in order to better approximate a normal distribution. The scores were then transformed back for presentation in tables and figures.

2.2.2. *Sleepiness*

Sleepiness was assessed at each test session with the Stanford Sleepiness Scale (Hoddes et al., 1973).

2.2.3. *Body temperature*

Sublingual temperature was recorded with an electronic thermometer (Ivac Model 811). The temperature probe was allowed to warm for 10 min during the reaction time task to assure a reliable recording.

2.2.4. *Sleep EEG*

Continuous recordings of left and right EOG (referred to A2) and central (C2-A2) and occipital (Oz-Al) EEG were obtained using gold cup electrodes applied to the face and scalp, interfaced with a Grass Model 78 polygraph. Sleep stages were identified according to the EEG criteria of Rechtschaffen and Kales (1968). Traditional SWS amplitude criteria (75µV) were used exclusively. Sleep stage percentages were calculated as minutes of sleep stage divided by the difference between time in bed and latency to the initial occurrence of Stage 1 sleep.

2.3. *Design*

After a screening/adaptation night, each subject had three or four baseline (B) nights of sleep, followed by two nights (64 h) of sleep deprivation (SD1, SD2), followed by two nights of recovery sleep (R1, R2) and a post study baseline (R3). For all subjects, the initial baseline night was discarded and only data from the two baseline nights immediately preceding sleep deprivation were averaged for the baseline (B) condition. For the two performance tasks, data from the post study baseline (R3) were averaged with the initial data to control learning effects before the data were compared to that from the sleep loss condition.
2.4. Procedure

Each subject slept in the same private, sound-attenuated room throughout the study. Sleep was scheduled for each subject based upon his normal sleep times between 2200 and 0900 on both baseline and recovery nights. Subjects reported to the laboratory 1 h before bedtime for electrode application and testing. Three additional 20-min awakenings for performance testing were equally spaced throughout each night at about 0130, 0345, and 0545. Each awakening occurred after at least 5 min of continuous Stage 2 sleep, and was accomplished via audiometer using standard methodology reported elsewhere (Rosa et al., 1983). Other data indicate that forced awakenings occurring at intervals greater than 20 min do not have significant effects on following performance (Bonnet, in preparation). The final morning awakening was also made with the audiometer although the stage of sleep from which it was made was not controlled. The latency to return to sleep after each standard awakening was recorded. To equalize for sleep lost during performance testing, subjects normally went to bed 30 min earlier than normal and arose 30 min later than normal. On baseline and recovery days subjects maintained their usual routine with a reminder to avoid naps and unusual events if possible.

Subjects were typically evaluated in pairs (although they were separated for sleep and each test session). During deprivation periods, subjects remained at the laboratory (under technician watch) and engaged in leisurely activities such as talking, watching television, reading, taking walks, or playing video games or pool with each other or the technician. Subjects were asked to avoid over-exertion and lying down. Non-startling procedures were used by the technicians to awaken faltering subjects. Subjects were provided meals at the hospital cafeteria and had the option of bringing food back to the laboratory to keep in the refrigerator there. Drinking caffeinated beverages was prohibited during the night, but a cup of coffee, when habitual, was allowed with breakfast. After one night awake, the subjects had the option of leaving the lab for a few hours (to go to work, etc.) if they returned before evening. After two nights awake, technicians accompanied subjects during their daytime activities.

2.5. Data analysis

Data were analyzed as follows:
1. A series of $3 \times 5 \times 5$ (Groups $\times$ Condition $\times$ Time of test) analyses of variance with repeated measures on condition and time of test was performed for performance, mood, body temperature, threshold of arousal at each session, and latency to sleep onset after each test session.
2. A $3 \times 5$ (Group $\times$ Condition) analysis of variance with repeated measures on condition was performed for each of the standard sleep variables.
3. Significant \( p < 0.05 \) group and condition effects were determined using the corresponding \( F \)-value and Greenhouse-Geisser calculated degrees of freedom (Winer, 1971). As the current report is concerned with the interactive effects of age and sleep loss, only effects containing a significant \( p < 0.05 \) group effect are reported here. More extensive reports of the sleep and performance data from the older groups are presented elsewhere (Bonnet, 1985a; Bonnet, 1986). In the presence of a significant condition effect, group by condition interactions were considered significant if the \( F \)-ratio using Greenhouse-Geisser-calculated degrees of freedom was significant at the \( p < 0.1 \) level. This more liberal criterion was used because smaller group differences were occasionally lost in the variance from very large differences among conditions. In all analyses, Newman-Keuls pairwise comparisons, based on Greenhouse-Geisser computed degrees of freedom, were performed at the \( p < 0.05 \) level to determine significant pairwise group or condition differences.

3. Results

3.1. Performance, sleepiness and oral temperature

Significant three-way interactions were obtained for reaction time \( [F(15,253) = 1.61, \ p < 0.07] \), word memory \( [F(15,253) = 2.18, \ p < 0.01] \), and body temperature \( [F(15,253) = 2.17, \ p < 0.01] \). There was no significant age effect or interaction for the Stanford Sleepiness Scale \( [F(18,304) < .1] \).

3.2. Reaction time

The data for the three groups for reaction time on each night are plotted in fig. 1. Under all conditions, the groups did not differ significantly from each other at the first evening test session (2300). However, after awakening from sleep, the YN had significantly slower reaction times than the OI at all twelve observation points (four each on B, R1 and R2) and significantly slowed reaction times than the ON at 11 of 12 observation points. Additionally, it was found that the older normals had significantly slower reaction times than the older insomniacs throughout the baseline condition and at the first two awakenings on R1 (0130 and 0345 AM). During SD1, there were no significant differences in reaction time except at the fourth test period, where the YN responded more slowly than the OI. On SD2, the YN responded more slowly than both of the older groups at both the third and fourth test periods.

In terms of recovery from sleep deprivation, the YN responded significantly more slowly than B throughout R1. Both older groups responded significantly more slowly at the first three test periods of R1 than they did at the
corresponding times during B. No differences from B were found during R2 for any group in these analyses.

3.3. Word memory

Word Memory performance in the YN followed a standard circadian curve with the poorest performance at about 0345 AM. A similar curve was obtained in both older groups except that the initial performance level was significantly lower in the older subjects (11 vs. 20 words recalled) and the circadian decline much less (to 10 vs. 14 words recalled) than in YN (see fig. 2). Performance declined significantly in the YN during sleep loss but remained close to baseline in both older groups. On R1, before sleep, YN recalled about 16 words whereas both older groups recalled significantly less (about 11 words). At the first awakening on R1, the young adults remembered about seven words whereas the older groups remembered significantly more (about 9.5
words). By the morning following R1, the young group again recalled significantly more words (17) than both older groups.

3.4. Body temperature

Body temperature for the three groups across conditions may be seen in fig. 3. Under all conditions, oral temperature did not differ in the three groups at the first evening test session (2300 hours). Under all sleep conditions both preceeding and following sleep loss, the YN had significantly lower oral temperature after each awakening from sleep (0130, 0345, 0545, and 0800 h). During sleep loss, the groups did not differ in oral temperature except at two points (the morning after SD1 and 0545 a.m. on SD2). During SD1 and SD2, the OI had lower oral temperature than the ON at the former time point and higher oral temperature than the YN at the latter time.

3.5. Sleep EEG

Standard sleep parameters were scored for all subjects. However, as there were multiple groups, conditions, and interactions, the data are presented as a
Fig. 3. Oral temperature in young normal (YN), older normal (GN), and older insomniac (GI) subjects at 2300, 0115, 0530, and 0800 h. The sections are: Baseline (A); first sleep deprivation night (B); second deprivation night (C); first recovery night (D); and second recovery night (E). Significant group differences ($p < 0.05$, Neuman-Keuls) are marked in each section.

Table 1
Interaction $F$-Values for sleep variables $^a$

<table>
<thead>
<tr>
<th>Sleep parameter</th>
<th>Significant effect</th>
<th>$F$</th>
<th>$df$ $^b$</th>
<th>$p$</th>
<th>Figure</th>
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</thead>
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<tr>
<td>Time in bed</td>
<td>None</td>
<td>0.87</td>
<td>5,82</td>
<td>0.5</td>
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<tr>
<td>Total time asleep</td>
<td>G * C</td>
<td>2.66</td>
<td>4,71</td>
<td>0.03</td>
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<tr>
<td>Awakenings</td>
<td>G * C</td>
<td>2.00</td>
<td>5,85</td>
<td>0.08</td>
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</tr>
<tr>
<td>Wake during sleep</td>
<td>G * C</td>
<td>3.24</td>
<td>3,43</td>
<td>0.03</td>
<td></td>
</tr>
<tr>
<td>%1</td>
<td>G</td>
<td>10.47</td>
<td>2,33</td>
<td>0.0003</td>
<td></td>
</tr>
<tr>
<td>%2</td>
<td>G * C</td>
<td>7.96</td>
<td>4,71</td>
<td>0.0001</td>
<td>4</td>
</tr>
<tr>
<td>%3</td>
<td>G * C</td>
<td>5.41</td>
<td>5,86</td>
<td>0.0002</td>
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</tr>
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<td>%4</td>
<td>G * C</td>
<td>9.46</td>
<td>4,73</td>
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</tr>
<tr>
<td>%R</td>
<td>G</td>
<td>18.83</td>
<td>2,33</td>
<td>&lt; 0.0001</td>
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</tr>
<tr>
<td>LAT1</td>
<td>G * T, C</td>
<td>6.52</td>
<td>4,64</td>
<td>0.0002</td>
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<tr>
<td>LAT2</td>
<td>G * T, C</td>
<td>3.90</td>
<td>4,70</td>
<td>0.005</td>
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</tr>
<tr>
<td>LTR (log transform)</td>
<td>G * C</td>
<td>3.97</td>
<td>5,81</td>
<td>0.003</td>
<td>7</td>
</tr>
<tr>
<td>Arousal threshold</td>
<td>G</td>
<td>3.07</td>
<td>2,33</td>
<td>0.05</td>
<td></td>
</tr>
</tbody>
</table>

$^a$ G = Group (GI, GN, YN); C = Condition (B, R1, R2, R3); T = Time of night (sleep onset 1, 2, 3 or 4); G * C = Interaction.

$^b$ $df$ differ for each analysis because the presented numbers are $df$ permitted by Greenhouse-Geisser criteria.
summary table of $F$-values followed by a series of figures. Table 1 presents sleep stages and significant age interactive effects. The only parameter on which significant age effects were not obtained was total time in bed, which averaged 458 min for the YN and 448 min for both older groups.

There were significant group by condition interactions for all three sleep time variables. Total time asleep is plotted in fig. 4. On baseline, the older insomniacs spent significantly less time asleep than the older normals, who spent less time asleep than the young normals. All groups had increased total sleep time on R1, but the OI improved most dramatically to spend as much time asleep as the ON. By R3, however, the OI were spending significantly less time asleep than the ON (although still spending significantly more time asleep than they were on baseline). The data for number of awakenings ($> 30$ s) during sleep and total time spent awake during sleep were similar to those reported for total time asleep.

For sleep stages, there were significant group effects for Percent Stage 1 and REM, but the groups did not respond differentially to sleep loss or recovery. It was found that young adults had less Stage 1 (6%) than older insomniacs (8%), who had less Stage 1 than older normals (10%). Older insomniacs had less REM (16%) than either other group (24%).

There were significant group $\times$ condition interactions for percent Stage 2 (fig. 5), 3, and 4 (Stage 3 + 4 is presented in fig. 6). As shown in fig. 5, Older
normals had a higher proportion of Stage 2 on baseline than either other group. However, on R1, Stage 2 increased significantly in OI to reach the level of ON while Stage 2 decreased significantly in YN. Stage 2 then remained
Fig. 7. Latency to Stage 1 sleep in young normal (YN), older normal (GN), and older insomniac (GI) subjects at five points across the night. The sections are: Baseline (A) and recovery nights one (B), two (C), and three (D) after 64 h of sleep deprivation. Significant (*p < 0.05, Newman-Keuls) group differences are marked.

Elevated in GI throughout R2 and R3. Young adults had significantly more slow wave sleep (because of more Stage 4) than older subjects throughout all conditions (see fig. 6). Slow wave sleep increased significantly on R1 in all groups. However, that rebound was almost exclusively Stage 4 in YN and Stage 3 in older subjects. In all groups, the slow wave sleep rebound was confined to R1.

For latency variables, there were significant group by time of night interactions for latency to Stage 1 (plotted in fig. 7) and latency to Stage 2. When compared to GI, the YN had significantly longer latencies to sleep onset at the beginning of the night on B and R3. However, after the second awakening (0130 h), it was taking GI significantly longer to fall asleep than the YN, and, after the third awakening (0345 h), it was taking the GI significantly longer than the ON to fall asleep. On R1, latencies to sleep onset in all groups were very short. On R2, the baseline pattern had begun to redevelop.

The latency to REM sleep (i.e. the time from initial sleep onset to first appearance of REM) showed an interesting group by condition interaction (fig. 8). Although REM latencies in the three groups were very comparable on
Fig. 8. Latency to REM sleep in young normal (YN), older normal (GN), and older insomniac (GI) subjects on baseline (BL) and recovery sleep nights one, two, and three (R1, R2, and R3) after 64 h of sleep deprivation. Significant group differences (*) and difference within the young normal group (+) and within the older normal group (**) are marked (*p < 0.05, Newman-Keuls).

Baseline, REM latency decreased significantly on R1 in older normals, stayed the same in older insomniacs, and increased significantly in young normals. The groups differed significantly from each other on R1. On R2, all groups had reduced REM latency (significant for YN but not older groups) as compared to baseline, and there were no group differences.

Analysis of the arousal threshold data revealed only a simple age effect. Young adults had significantly higher arousal thresholds (60 dB) than other older groups (44 dB and 46 dB respectively for older insomniacs and normals).

4. Discussion

4.1. Sleep variables

Total time in bed, which was held constant within subjects but not rigidly controlled between subjects, was very similar in the three groups. As might be expected from previous work (Roffwarg, Muzio, & Dement, 1966), total time spent asleep was less in the older groups. Sleep in the older normals was within one standard deviation of previously published norms (Williams et al., 1974) for all sleep parameters regardless of the three nightly awakenings. As might be expected by selection criteria, total time spent asleep was least in the
older insomniacs. Although all groups improved their sleep efficiency after sleep loss, there was a disproportionate increase in the older insomniacs, who improved to the level of the older normals (fig. 4). Whereas the OI were unable to maintain this level of sleep efficiency on R2 and R3, their total time asleep was still significantly higher than B on R3. Both normal groups had returned to their baseline level by R2. This interactive increase in sleep efficiency came primarily from a 25% increase in Stage 2 (fig. 5) in older insomniacs on R1 versus a 2% increase in Stage 2 in older normals and a 29% decrease in young adults. These changes indicate a differential EEG recovery process in the three groups. Young normals, starting with a high sleep efficiency, had large increases in Stage 4 which were offset by decreases in Stage 2 and REM during recovery. Older normals, with an intermediate baseline sleep efficiency, had increased Stage 3 with no Stage 2 change and a decreased REM latency during recovery. Older insomniacs, with poor sleep efficiency, had large increases in Stage 2 and 3 during recovery to account for their increased sleep time. Slow wave sleep returned to baseline levels in all groups on R2, but Stage 2 remained elevated in the older insomniacs through R3 to account for their increased total sleep time.

In terms of SWS (fig. 6), differential effects were also obtained in the three groups. Young adults had a large rebound in Stage 4 on R1 with no change in Stage 3 sleep. In the older groups, the greatest increase on R1 was in Stage 3 while Stage 4 increases from baseline to R1 were relatively small.

The latency to REM results (fig. 8) are of potential interest because they may have been related to the differential amounts of SWS and SWS rebound in the groups. Young adults, with the most SWS and greatest absolute increase in SWS (from 20 to 35%) on R1, had significantly increased REM latencies on R1. The older normals, with small amounts of SWS and the smallest absolute increase on R1 (from 4 to 12%), had significantly decreased REM latency on R1. These REM latency findings are supported by at least two other sleep deprivation studies (Carskadon & Dement, 1985; Webb, 1981) carried out in older subjects. The implication of this interrelationship is that if there is less pressure for SWS, then increased REM pressure, as indicated by shorter REM latency, can be shown earlier. This interpretation is upheld by the SWS and REM latency results on R2. SWS had returned to baseline in all groups by R2, and REM latency was significantly reduced as compared to baseline only in the young adults, who had increased REM latency on the preceding night. These data imply that the reduced SWS seen in the older individuals is a change in need for this state rather simply a decrease in EEG amplitude (Webb, 1982). If aging changes in SWS were simply amplitude reductions, then no differences in latency to REM should have existed in the study groups during R1. No change in REM latency in OI and the decrease in ON imply a decreased SWS requirement in these groups allowing earlier appearance of REM on R1.
The sleep latency data, with onset systematically recorded at four points during each night (2300, 0130, 0345, and 0545), describe convincingly the nature of insomnia in the elderly. At 2300 on baseline nights, young normals actually had significantly longer latencies to Stage 1 sleep than did older insomniacs (16 vs. 10 min). However, by the first awakening point, the older insomniacs had sleep latencies significantly longer than the YN (16 vs. 6 min). By the second awakening point, the OI had longer sleep latencies than both ON and YN (16 vs. 9 vs. 4 min respectively). On baseline, the YN had a standard circadian latency curve (Bonnet & Webb, 1979; Richardson, Carskadon, Brown, & Dement, 1982) with the shortest latencies to sleep onset around 0345. The ON had 8–10 min latencies throughout the night and the OI had the previously described increase in latency across the night. These data may be explained by the underlying oral temperature curve (fig. 3). Both older groups had elevated temperature during sleep as compared to the young adults, and elevated temperature may have accounted for some of the increased latency during the night. However, this explanation does not account for the reduced sleep latencies on R1 and R2. On these nights, the group latency differences largely disappeared but the oral temperature differences remained.

4.2. Performance

Reaction time at bedtime (2300) on baseline, sleep deprivation, and recovery nights (except R1) was similar in all three groups and varied little from study night to study night. After arousal from sleep under baseline conditions, the OI had significantly faster reaction times than the ON, who had significantly faster reaction times than the YN. Faster reaction time in the older groups during awakenings was marginally related to higher body temperature at awakening during the night (average baseline \( r(34) = 0.29 \) ( \( p < 0.1 \)). Faster reaction times in older subjects may also be related to the fact that their Stage 2 arousal thresholds were lower, so they might have been more ready to respond when awakened (Bonnet, 1983). The average baseline correlation across 36 subjects between threshold and response speed was \( r(34) = 0.33 \) ( \( p < 0.05 \)). However, despite these correlations, the older insomniacs as a group had neither significantly lower arousal threshold nor higher oral temperatures than the older normals on baseline nights. Some data from young adults have shown insomniacs to have faster reaction speed than normals (Bonnet, Rosa, Smitsont, & Kramer, 1980), but this has usually been explained as a higher underlying activation level. The difference in reaction time between OI and ON should also be considered relatively marginal because they were not found in an earlier analysis which included only the older performance data (Bonnet, 1985a). Regardless, both the reaction time differences and the oral temperature differences are sleep specific phenomena because (as discussed
above) few consistent group differences were obtained on either variable at presleep testing or during sleep deprivation.

Of most importance to the specific hypothesis of this study was the time course of return to baseline performance following sleep loss in the three groups. Reaction time in both older groups had returned to baseline levels by 0800 on R1 while reaction time was still significantly slower than baseline in young adults at that time. It can also be seen from fig. 1 that the young adults’ reaction time remained at a lower level than the older groups throughout the second recovery night. In terms of statistical effects, this can be seen at the 0800 testing point, where the young adults differ significantly from both older groups on R2 where they differed only from the O1 on baseline. These data indicate that the young adults not only recovered more slowly from sleep loss on R1 but also that they had some extension of their performance recovery into R2. These data agree with data recently presented by Carskadon and Dement (1985), who found that older subjects had daytime Multiple Sleep Latency Test (MSLT) values at baseline levels following sleep loss and a single night of recovery sleep while shorter than normal latencies continued in young adults.

The group interaction for word memory generally showed that overall memory performance was much worse in the older subjects. Further decrease in memory ability during sleep loss was minimal in the older groups, perhaps because baseline performance was already extremely poor, sleep inertia was less, and underlying oral temperature was higher. Sleep loss and early recovery had a greater impact on the memory abilities of the young adults. Whereas the young adults performed much better than either of the older groups during normal waking hours, their memory performance was significantly worse than the older subjects early on R1. It only improved to its normally superior relationship by the end of R1.

The greater impact of sleep loss on the young group was also seen in their significantly slower reaction time during the middle of the deprivation nights as compared to the older subjects. These data indicate that although the groups did not differ from each other in report of sleepiness throughout the study, there were consistent group differences in oral temperature and performance. The reaction time data are of particular importance because they indicate that, if anything, older subjects, and particularly older insomniacs were (a) less affected by sleep loss, (b) less affected by initial recovery sleep, and (c) actually returned to baseline performance levels more quickly than young adults.

4.3. Age/sleep/performance interactions

Older insomniacs in the current study were chosen to exhibit chronic insomnia for a median of over twenty years. Their subjective report of 5.1 h of
sleep per night (and recorded total of less than 6 h of sleep per night) should have resulted in the accumulation of a considerable sleep debt associated with degraded mood and performance. The data indicate that long-term insomnia, when not associated with significant underlying myoclonus, apnea, or psychopathology, does not result in increased sleepiness or decreased performance. Further, participants with long-term insomnia had faster reaction time during nocturnal testing and appeared to tolerate the sleep deprivation period more easily than did young adults. Despite rapid recovery of performance, total sleep time was still increased in insomniacs on their third recovery night. It must be concluded, at least in terms of memory and reaction time performance, that neither (a) habitual sleep habits, nor (b) the differential sleep stage distribution during recovery sleep seen in elderly subjects made their sleep in any way less restorative. In fact, contrary evidence was presented. Young adults were more impacted both by total sleep loss and by extremely deep recovery sleep during R1. These results are best explained by a combination of factors. First, young adults slept more deeply and were therefore more likely to be affected by sleep inertia during waking testing (Bonnet, 1983). One might expect that effect to be greater following sleep loss. Second, circadian rhythms, here indexed by oral temperature, appeared to be stronger in the young adults. Thus, their nocturnal performance trough was somewhat larger (Reinberg et al., 1980; Weitzman, Moline, Czeisler, & Zimmerman, 1982), and performance somewhat worse even during total sleep loss.

The fact that the elderly groups had shorter latencies to initial sleep onset than young adults does not support a reduced sleep need hypothesis. Such a result is consistent with an alternative explanation of a flattened circadian rhythm with a shorter period in the elderly (Reinberg et al., 1980). According to this hypothesis, the elderly should not reach the low points in alertness in response to sleep that are evident in the young and therefore have improved nocturnal performance with disrupted sleep. Additionally, they should not reach the high points in alertness reached by the young during the daylight hours. Their circadian rhythm has a generally shorter period, they should initially fall asleep more easily than young adults but have trouble maintaining sleep throughout the night. One could also posit that older insomniacs have a shorter circadian period length than older normals to account for their nocturnal sleep latency data. Such an explanation is supported very marginally by the response speed data (see fig. 1), where insomniac values increase a bit faster than normals in the early morning. The oral temperature data, however, do not support this hypothesis.

The current study has demonstrated significant differences in EEG sleep response to total sleep loss in young adults, older normals, and older insomniacs. A differential decrease in REM latency on recovery nights in the older groups was interpreted as evidence of decreased SWS need in older individuals. Differences in sleep stage distribution on recovery nights, how-
ever, were not related to within sleep restoration of performance, which proceeded at a similar rate in the older subjects, irrespective of their chronic sleep pattern, and at a slower pace in young adults. One can interpret these results either as evidence of a reduced sleep need in older individuals or as an effect of simple sleep process changes with aging. There are two potential implications of these data for older individuals, and they carry slightly different implications for treatment. If there is actually a decreased requirement for sleep in the elderly, then the only treatment necessary is re-education or adjustment or an archaic attitude toward sleep need. Alternatively, if the perceived sleep problem is actually an expression of a shortened or flattened circadian rhythm, then a more active form of treatment, such as increased daytime activity to accentuate the body temperature curve, could be indicated. Neither interpretation of the data indicates use of medication in initiating or preserving sleep in elderly patients with insomnia which is not secondary to sleep pathology or psychopathology.

References


