Sleep Loss in Aging

M. H. Bonnet, PhD, ACP,* and D. L. Arand, PhD, ACP†

As described elsewhere in this issue, sleep becomes shortened and less consolidated as a function of normal aging. These changes imply either that there is an increase in insomnia as age increases, that there is an increasing inability to maintain sleep, or that there is a reduction in total sleep need. Because differentiation of these alternatives is important not only for our knowledge of aging but also to understand the nature of sleep and insomnia, several recent studies have examined the effect of experimentally produced sleep deprivation in older individuals. Finally, sleep loss is currently used as a therapeutic intervention in patients with depression. Because of the importance of depression in aging, studies of sleep loss in older depressed patients have also been reported. The article will review these studies and their implications following a brief background of sleep loss effects in general.

Many studies have documented the effects of both chronic and acute loss of sleep on decreasing performance efficiency and mood and increasing sleepiness in young adult subjects. In a typical study, subjects are adapted to the laboratory and then have one or more normal baseline nights of sleep. Various measures of cognitive and motor performance, mood, and sleepiness usually measured by multiple sleep latency tests (MSLTs) are made under normal conditions. A period of time usually ranging from 40 to 64 hours follows during which subjects remain awake under close scrutiny in the laboratory. During the period of sleep loss, the measures of psychomotor performance, mood, and alertness are repeated frequently. Studies of sleep loss in young adults are consistent in finding significant impact on sleepiness and mood after a single night of sleep loss or significant sleep reduction.

Performance of psychomotor tasks may be maintained after one night of sleep loss, depending on the nature of the task. After two nights without sleep, large and consistent decline in performance ability is seen on many tasks. Tasks most sensitive to sleep loss in young adults are typically (1) long lasting; (2) with minimal knowledge of results; (3) with increased difficulty;

*Associate Adjunct Professor of Medicine, University of California, Irvine, Director of Sleep Laboratory, Long Beach Veterans Administration Hospital, Long Beach, California
†Assistant Professor, University of California, Los Angeles, Director of Sleep Laboratory, UCLA Neuropsychiatric Hospital, Los Angeles, California

Supported by the Sleep-Wake Disorders Research Institute

Clinics in Geriatric Medicine—Vol. 5, No. 2, May 1989
Table 1. Core Aging Sleep Loss Studies

<table>
<thead>
<tr>
<th>STUDY</th>
<th>SUBJECT TYPE</th>
<th>AGE</th>
<th>LENGTH (h)</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bonnet et al.</td>
<td>Normals</td>
<td>18-25</td>
<td>64</td>
<td>12 (12 M)</td>
</tr>
<tr>
<td></td>
<td>Normals</td>
<td>55-71</td>
<td>64</td>
<td>12 (12 M)</td>
</tr>
<tr>
<td></td>
<td>Insomniacs</td>
<td>55-71</td>
<td>64</td>
<td>12 (12 M)</td>
</tr>
<tr>
<td>Carlsson and Dement</td>
<td>Normals</td>
<td>61-77</td>
<td>40</td>
<td>10 (2 M)</td>
</tr>
<tr>
<td>Reynolds et al.</td>
<td>Normals</td>
<td>56-79</td>
<td>40</td>
<td>10 (10 M)</td>
</tr>
<tr>
<td></td>
<td>Normals</td>
<td>56-79</td>
<td>40</td>
<td>10 (10 M)</td>
</tr>
<tr>
<td></td>
<td>Depressed patients</td>
<td>56-79</td>
<td>40</td>
<td>10 (10 M)</td>
</tr>
<tr>
<td></td>
<td>Demented patients</td>
<td>56-79</td>
<td>40</td>
<td>10 (10 M)</td>
</tr>
<tr>
<td>Webb et al.</td>
<td>Normals</td>
<td>30-60</td>
<td>40</td>
<td>12 (12 M)</td>
</tr>
<tr>
<td></td>
<td>Normals</td>
<td>20-25</td>
<td>40</td>
<td>12 (12 M)</td>
</tr>
</tbody>
</table>

(4) of newly acquired skills; (5) with increased complexity; (6) involving short-term memory, and (7) relatively lacking in intrinsic interest or motivation. Typical changes in performance include lengthened reaction time and a decrease in speed on problem-related tasks in an attempt to preserve correct responses. Performance changes tend to follow the circadian curve, with relatively normal performance near the time of peak body temperature and increasing compromise as body temperature reaches its nadir in the early morning.

The effects of total sleep loss on sleepiness and recovery sleep have also been well documented in young adults. Sleepiness, as measured by the MSLT, increases rapidly during the first night of sleep loss so that sleep latencies are usually 2 minutes or less on the morning following the first night of total sleep loss. Recovery sleep in young adults is characterized by a short sleep latency, larger increases in stage 4 and stage 3 sleep, and decreased wake time. Increases in REM sleep are frequently found on succeeding recovery nights.

Because a decline in psychomotor performance is normal as a function of aging and because there are common changes in sleep stage distribution during aging, it is easy to hypothesize that the decrease in performance ability is related to increasing sleep fragmentation, decreased slow wave sleep, or decreased ability to maintain sleep. Insomnia is a common complaint in the elderly, but is is not known if the decreased ability to sleep is a normal change with aging or whether it is the variable mediating changes in performance. Because it is known that the unwanted sedating side effects of hypnotics may have negative consequences in older individuals, the treatment of insomnia without significant consequences with hypnotics may not be justified. All of these issues can be explored by use of sleep deprivation to determine whether older individuals are more sensitive to sleep loss (and are thus chronically more deprived of sleep) or whether older individuals are less sensitive to sleep loss than young adults (and thus have a decreased need for sleep).

This review will concentrate on sleep loss studies performed by four groups during the past few years (Table 1). Relevant characteristics of the studies are reported in the table. It can be seen that while the number of subjects in each study was small, independent groups have studied normal older males and females, and insomniacs, depressed and demented patient groups. Both EEG changes and psychomotor performance changes have been described. The sleep changes will be described first.

### Sleep Stages

Baseline and recovery sleep stage values (means across comparable studies) for older subjects after one and two nights of sleep loss can be seen in Tables 2 and 3. One study has specifically examined differences in sleep loss as a function of gender. Statistically significant differences were found between older men and women on the expected sleep dimensions. However, the only age by sleep deprivation interaction was for minutes of stage 4 sleep, which was initially higher in women and therefore increased more during the first night of recovery sleep. The studies of recovery sleep are all consistent in reporting the following changes on recovery nights:

Table 2. Baseline and Recovery Sleep Stages: Males*

<table>
<thead>
<tr>
<th>STAGE</th>
<th>BASELINE</th>
<th>RD1</th>
<th>RD2</th>
<th>RD4</th>
</tr>
</thead>
<tbody>
<tr>
<td>TIB</td>
<td>445</td>
<td>458</td>
<td>431</td>
<td>442</td>
</tr>
<tr>
<td>TST</td>
<td>392</td>
<td>419</td>
<td>395</td>
<td>429</td>
</tr>
<tr>
<td>SL efficiency</td>
<td>86</td>
<td>92</td>
<td>88</td>
<td>97</td>
</tr>
<tr>
<td>Latency</td>
<td>14</td>
<td>5.2</td>
<td>5.6</td>
<td>5.6</td>
</tr>
<tr>
<td>SW</td>
<td>11</td>
<td>5</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>%1</td>
<td>5.4</td>
<td>2.7</td>
<td>4.9</td>
<td>3.4</td>
</tr>
<tr>
<td>%2</td>
<td>56</td>
<td>60</td>
<td>58</td>
<td>52</td>
</tr>
<tr>
<td>%3</td>
<td>5.6</td>
<td>8.1</td>
<td>7.1</td>
<td>10</td>
</tr>
<tr>
<td>%4</td>
<td>10</td>
<td>4.5</td>
<td>3.1</td>
<td>1.4</td>
</tr>
<tr>
<td>REM latency</td>
<td>19</td>
<td>22</td>
<td>21</td>
<td>25</td>
</tr>
<tr>
<td>REM latency</td>
<td>65</td>
<td>32</td>
<td>39</td>
<td>24</td>
</tr>
</tbody>
</table>

*RD1 = Recovery night 1; 40 - 40 hours of sleep loss.

Table 3. Baseline and Recovery Sleep Stages: Females

<table>
<thead>
<tr>
<th>STAGE</th>
<th>BASELINE</th>
<th>RD1</th>
<th>RD2</th>
<th>RD4</th>
</tr>
</thead>
<tbody>
<tr>
<td>TIB</td>
<td>438</td>
<td>456</td>
<td>444</td>
<td></td>
</tr>
<tr>
<td>TST</td>
<td>353</td>
<td>426</td>
<td>392</td>
<td></td>
</tr>
<tr>
<td>SL efficiency</td>
<td>82</td>
<td>84</td>
<td>89</td>
<td></td>
</tr>
<tr>
<td>Latency</td>
<td>20</td>
<td>8.5</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>SW</td>
<td>18</td>
<td>5.5</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>%1</td>
<td>5.4</td>
<td>2.5</td>
<td>4.1</td>
<td></td>
</tr>
<tr>
<td>%2</td>
<td>56</td>
<td>51.5</td>
<td>54</td>
<td></td>
</tr>
<tr>
<td>%3</td>
<td>11</td>
<td>15</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>%4</td>
<td>8.5</td>
<td>15</td>
<td>9.4</td>
<td></td>
</tr>
<tr>
<td>REM latency</td>
<td>18</td>
<td>18</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>REM latency</td>
<td>72</td>
<td>63</td>
<td>39</td>
<td></td>
</tr>
</tbody>
</table>
Total Sleep Time

Total sleep time (Fig. 1) is increased on the first recovery night in all groups. There is one indication that while the increased total sleep in normal elderly males closely parallels the increased total sleep seen in a young adult control group, there is a relatively greater increase in total sleep in a group of geriatric insomniacs such that total sleep time for the insomniacs was equal at that point to the total sleep for the older normals (see Fig. 1). In the study with depressed and demented patients, the increase in total sleep in the patient groups did not bring their total sleep to normal baseline levels although this was only a one-night sleep study. As in young adults, total sleep time decreased during succeeding recovery nights in the older subjects in each study. Total sleep times usually were not increased over baseline by the second recovery night except for the insomniac group, which continued to have increased total sleep time compared with their baseline levels throughout three recovery nights.

Sleep Latency

As expected from studies on young adults, all studies reported reduced sleep latencies when subjects returned to sleep after sleep deprivation.

Significant additional data was presented in the Bonnet studies, where subjects were awakened briefly during the night and return-to-sleep latencies were measured, and in the Carskadon and Dement study, where MSLT recordings were done throughout the study. Sleep latency data from the Bonnet and Rosi study are presented in Figure 2. For latency variables, there were significant "group by time of night" interactions for latency to stage 1 (plotted in Figure 2) and latency to stage 2. When compared with older insomniacs, the young normal subjects had significantly longer latencies to sleep onset at the beginning of the night on baseline nights and on their third recovery night. However, after the second awakening (1300 hours), it was taking older insomniacs significantly longer to fall asleep than the young normal subjects, and, after the third awakening (0345 hours), it was taking the older insomniacs significantly longer than the older normal subjects to fall asleep. On the initial recovery night, latencies to sleep onset in all groups were short. On the second recovery night, the baseline pattern had begun to redevelop. Carskadon and Dement MSLT findings will be described in the performance section.

Transient Arousals

Of the sleep deprivation studies, brief arousals were quantified in only one. It was found that transient arousals decreased on the first recovery night from 10.3 to 5.6 per hour.

Stage 1 Sleep

Stage 1 is normally increased in older as compared with younger subjects and is normally reduced in all subjects on initial recovery nights (Tables 2 to 4). No differential effects were found in any clinical group.

Stage 2 Sleep

Stage 2 sleep may actually be decreased in young adults during their first recovery night from sleep deprivation. In the older subjects, stage 2 sleep was significantly increased in two studies and did not change from baseline in the others. In the Bonnet and Rosi study, stage 2 remained significantly elevated in the older insomniacs throughout three recovery nights. Such continuing effects were not seen in the other patient groups.

Stage 3 Sleep

Stage 3 sleep was significantly increased in all groups on their initial recovery night. The stage 3 effect was limited to the first night in all groups.

Stage 4 Sleep

As with stage 3, all groups reported an increase in stage 4 sleep after sleep loss. The stage 4 increase was also limited to the first recovery night and was relatively small in some studies due to very small baseline amounts of stage 4. Figure 3 summarizes slow wave sleep changes in young adults, older normal subjects, and older insomniacs. It can be seen that while young adults (and to a lesser extent, older women who had more baseline SWS) rebound primarily in stage 4 sleep, older subjects rebound primarily in stage 3 sleep.
REM Sleep

The findings for recovery for REM sleep variables are perhaps among the most complex and interesting. In young adults, it is usual for REM latency to increase and REM sleep to decrease on the initial recovery night. Decreased REM latency and increased REM time are frequently found on subsequent recovery nights. No consistent changes in REM per cent are reported in the studies of older subjects following sleep loss. Two studies report no change, one study reports a significant decrease in REM per cent coming primarily from depressed and demented subjects, and one study reports a significant increase, primarily in older normals. Some evidence for rebounds continuing for as long as three recovery nights exists.

The REM latency findings are more consistent and more surprising. Bonnet first reported the appearance of sleep onset REM periods (SOREMPs) on the initial recovery night in normal older subjects. In that study, 6 of 24 (5 normals and 1 insomnia) had sleep onset REM periods on their initial recovery night. Sleep onset REM periods did not appear on any other night, and the participants had been screened to rule out depression, narcolepsy, or other sleep disorders. Overall a significant interaction was found between age and sleep night (Fig. 4). REM latency increased on the first recovery night in normal young adults, remained constant in older insomniacs, and decreased significantly in older normals (to a mean value of 24 min). On the second recovery night, the group interaction disappeared, and all groups had a significantly decreased REM latency. Reynolds et al. also reported SOREMPs in their carefully selected normal elderly subjects in five of 20 subjects on one of their two recovery nights. The other studies of sleep loss in older individuals have also shown a decrease in REM latency on the initial recovery night, and the Reynolds et al. study actually shows a significant interaction in REM latency with REM latencies decreasing in normal older groups and increasing in depressed and demented subjects on recovery sleep nights. Two studies have attempted to determine the relationship between REM latency and the reduced SWS in the elderly via...
correlational methods. In older normals and insomniacs, Bonnet found a significant correlation between SWS on baseline nights and REM latency on recovery nights. Reynolds et al. found a significant correlation between delta count and REM latency on the recovery night in normal subjects, but neither study found a significant correlation between SWS and REM latency on baseline nights.

Respiratory Disturbance and Leg Movements

Carskadon and Dement reported no change in the number of apneas or leg movements in normal older subjects from baseline to recovery sleep. They reported that existing apneas were significantly longer (by 3.5 sec) and that the leg movements which existed were less frequently associated with arousals on the first recovery night.

Arousal Threshold

Arousal thresholds were lower in older subjects than in younger subjects and increased in a parallel fashion in younger and older subjects during the initial recovery night.

SUMMARY

The sleep studies agree in describing the effects of sleep on recovery sleep in the elderly. SWS is increased, but not to the extent seen in young adults. Sleep time increases and sleep latency decreases similarly in normal younger and older subjects. All studies indicate at some level that there is a decrease in REM latency during recovery sleep in normal older individuals and that the decrease results in SOREMPs in about 20 per cent of subjects. The REM findings do not apply to depressed or demented individuals.
PERFORMANCE IN OLDER SUBJECTS DURING SLEEP LOSS

As described, changes in psychomotor performance during sleep loss have been extensively studied in young adults. A few studies have examined various measures of performance and alertness in older individuals during sleep loss as well. Most of these studies have also had young adult control groups.

The latest and most varied test battery was used by Webb in a study which compared baseline (postrecovery) daytime performance with performance measured between 2200 and 0600 hours on the second night of total sleep loss in normal young and older subjects. Tests ranged from subjective measures (sleepiness and mood) to attention (vigilance, visual search) to cognitive (memory, reasoning, digit symbol substitution, anagrams). Under daytime baseline conditions, the older subjects performed significantly more poorly than the young adults on both attention and cognitive tasks. During sleep loss, performance in both groups declined on most tasks but there were significant (P < .05) age by deprivation interactions on only three — two sleepiness scales and the digit symbol substitution task. In all of these cases, the decline in performance and mood in the young subjects was greater than in the older subjects (it should be noted that Webb concluded that overall performance declines were greater in the older participants based on other tests where performance per cent decline was greater in the older subjects than in the younger, but where the age by deprivation interactions were nonsignificant with P-values between 0.05 and 0.20).

In our studies, simple reaction time and short-term word memory ability were measured at bedtime, during three brief awakenings during the night (on sleep nights), or at 0115, 0330, and 0530 (during two nights of sleep loss) and after morning awakening in young adults, older normals, and older insomniacs. The data for the three groups for reaction time on each night are plotted in Figure 5. Under all conditions, the groups did not differ significantly from each other at the first evening test session (2200). However, after awakening from sleep, the young normals had significantly slower reaction times than the older insomniacs at all 12 observation points (four each on baseline and two recovery nights) and significantly slower reaction times than the older normals 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 the first recovery night (0130 and 0345 AM). During the sleep loss, the young normals generally responded more slowly than both of the older groups. On the second sleep loss night, the young normals responded more slowly than both of the older groups.

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 the first recovery night while reaction time was still significantly slower than baseline in young adults at that time. It can also be seen from Figure 5 that the young adults' reaction time remained at a lower level than the older groups' throughout the second recovery night. These data indicate that the young adults not only recovered more slowly from sleep loss on the
first recovery night but also that they had some extension of their performance recovery into the second recovery night. Similarly, Carskadon and Dement 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 reaction time and MSLT data are of particular importance because they indicate that, if anything, older subjects, and particularly older insomniacs were (1) less affected by sleep loss, (2) less affected by initial recovery sleep, and (3) actually returned to baseline performance and alertness levels more quickly than young adults.

These conclusions are not supported by all studies and measures. For example, in the Bonnet and Rosas study, 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 the first recovery night. It only improved to its normally superior relationship by the end of the first recovery night.

Mertens and Collins studied the performance of 30- to 39-year-old men and 60- to 69-year-old men before and after a night of sleep loss on vigilance and problem-solving tasks during morning and afternoon test sessions. When they found the expected decline in performance with sleep loss and that the older men performed more poorly in general during their daytime test sessions, they found no interaction of sleep loss with age on their tests.

**Summary**

Tests of performance and alertness in normal older subjects undergoing sleep loss reveal loss of performance and alertness similar to that seen in younger individuals. Recovery of performance ability occurs with one night of normal sleep, even following periods of sleep loss up to 64 hours in length. Older individuals, including older individuals with primary insomnia, may tolerate sleep loss with less decrease in ability compared with their baseline than young adults and may recover function more quickly than young adults. While it has been frequently shown that older individuals perform more poorly than young adults on a broad range of tasks, these findings do not hold well for periods of nocturnal performance or performance during sleep loss. It is possible that these findings may be accounted for to some extent by the decrease in amplitude of the circadian body temperature curve in older individuals. It is unfortunate that Reynolds et al. did not attempt to collect performance data in their depressed and demented patients to determine if the differential effects on mood and EEG would also be reflected in psychomotor performance.

**Sleep Disruption as Sleep Deprivation**

Carskadon, Brown, and Dement found that there was a correlation between brief nocturnal arousals and daytime sleepiness in elderly subjects. Several studies have recently shown that there is an empirical relationship between the rate of sleep fragmentation and the ability to sleep to restore function. When periodic sleep fragmentation becomes frequent (once per minute of sleep), sleep becomes nonrestorative and alertness and performance begin to approximate total sleep loss levels. One study has specifically examined periodic sleep fragmentation in young adults and in older normal individuals. As implied by the Carskadon et al. studies, brief arousals were more frequent in the 55- to 70-year-old subjects (about 25 brief arousals per hour versus nine brief arousals per hour in young adults). The experiment added about 15 standard arousals per hour in both young adult and older groups. The additional arousals were sufficient in number to result in significant decline in morning performance on simple reaction time, vigilance, and subjective sleepiness after two nights of disturbance in both young adults and older subjects. However, a significant age by disturbance interaction was found for correctly completed addition problems, with the young subjects completing fewer problems after sleep disturbance and the older subjects actually completing more than on baseline. These data were used to support the contention that older individuals may actually be less sensitive to sleep loss than young adults.

**Depression and Sleep Deprivation**

Sleep deprivation is one therapeutic approach in the treatment of depression. Many reported studies of sleep loss in depression have examined a wide range of patient ages and have not collected EEG data. Because the therapeutic effects of sleep loss which have been reported have not differed as a function of age, the sleep loss and depression studies will not be reviewed in detail here. Two studies have specifically looked at sleep loss in older depressed patients. Both studied sleep-deprived, depressed patients (average ages between 68 and 71 years) for one night. The studies suggest that older endogenous depressives, like younger endogenous depressives, show a relatively short lasting clinical improvement following sleep loss. Patients tend to remit following their first night of recovery sleep. Reynolds et al. reported that patients who responded with most clinical improvement to the sleep loss also fell asleep more quickly following sleep loss, had the greatest increase in sleep efficiency following sleep loss, and had the greatest increase in SWS following sleep loss. The correlations between sleep variables and clinical response led Reynolds et al. to conclude that there is a mutual interaction between the process of sleep regulation and the symptoms of depression.

**Implications and Indications**

Studies of sleep loss in older people have had several goals. I have attempted to determine if normal older individuals with at least a 20-year
history of insomnia (about 5 hours of reported sleep per night) would behave as if they had been chronically partially sleep deprived and would therefore be more sensitive to a sleep loss challenge than their normal-sleeping peers. Evidence was fairly clear that these "insomniacs" were not chronically sleep deprived, because they handled the sleep loss at least as well as or perhaps better than their peers and young adults. Rather, the evidence points more strongly to a reduction in sleep need in this group. It should be stressed, however, that these "insomniacs" were carefully chosen to (1) have objective insomnia; (2) have no sleep pathology such as apnea or periodic leg movements; and (3) be free of depression. Nonetheless, the data support the contention that a simple report of insomnia, even if it is valid and of long duration, may not be sufficient to warrant consideration of hypnotic therapy, because even successful shortening of sleep may not improve function if no deficit exists. Similarly, I have experimentally fragmented sleep in normal older subjects to determine if there is a greater impact of sleep disturbance per se. Such effects were not found, but it should be remembered (1) that sleep fragmentation effects do frequently exist in both young and older individuals; (2) that sleep fragmentation itself is a natural component of aging; and (3) that pathologies which periodically fragment sleep also increase in aging.

At a more theoretic level, the studies of sleep loss in the elderly provide a fascinating empirical test ground to study the interaction of SWS and REM. It is apparent that new rules concerning the interaction of SWS and REM exist. It is well known that SWS, at least as measured by conventional amplitude criteria, is decreased tremendously as one ages. However, several studies suggest that, if one ignores the amplitude scoring criteria, SWS amounts do not change. Clearly the possible decrease in SWS with aging is important for any theories concerning the function of SWS. It is of interest to know at one level if people who produce no conventional SWS are still capable of producing delta waves. The sleep loss studies clearly support this. At another level, one may posit that if it is the delta wave frequency that is important and that remains constant in aging then older people should have longer periods of delta frequency sleep (stage 2 by conventional criteria) following sleep loss, and REM recovery after sleep loss should be the same in older and younger subjects. The studies support the contention that SWS pressure is reduced in older people and that the reduction in SWS pressure is correlated with the amount of conventional SWS produced. Reduction of SWS pressure on the first recovery night allows the early appearance of REM sleep and accounts for the reported reductions in REM latency.

As a result of the consistent findings of reduced REM latency in patients with depression, manipulations which affect REM latency are of importance in understanding the nature of depression. Many studies have also shown that sleep deprivation is effective in reducing depression in depressed patients of all ages. The further findings of increased REM latency following sleep loss in elderly depressed patients implies on the one hand that the sleep deprivation may have been therapeutically useful for them, but on the other hand implies a basic REM mechanism difference between normal and depressed older individuals. Because of "misplaced" REM, depression is sometimes considered a circadian rhythm disorder and sleep deprivation may be considered a means of attempting to reset the biologic clock. It is possible that sleep deprivation could advance or delay the REM/NREM cycle by 40 to 50 minutes because this could account for the REM latency findings in both normal and clinical older groups. However, one would also have to posit that the change, which is not seen in young adults, is masked in young adults by SWS rebound. Regardless, it is clear in all groups that the EEG changes in recovery sleep dissipate rapidly and that baseline values return in all measures within 4 days.

Because the response of older depressed patients seems so opposite to that of normal older patients, it would be of interest to sleep deprive a group of older depressed patients and to follow them with MSLT and performance tests during a period of sleep loss to determine whether they are accumulating fatigue as normal subjects do. Performing tests frequently might help describe circadian rhythm differences in normal and depressed patients. MSLT tests might be able to document REM onsets in either group. Of more importance, it might be possible to correlate or split sleep loss effects (that is, sleepiness) and the antidepressant effects of sleep loss in the depressed group.

REFERENCES

24. Webb WB: A further analysis of age and sleep deprivation effects. Psychophysiology 22:156. 1985

Veterans Administration Hospital (111 P)
5901 East Seventh Street
Long Beach, CA 90822