Homeostatic sleep response to naps is similar in normal elderly and young adults

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Abstract

Delta homeostatic regulation can be challenged by reducing delta need with daytime naps and measuring delta in post-nap sleep. We previously demonstrated that, after a late afternoon nap, young adults reduce the amount of delta in post-nap sleep by the amount in the nap. We compared homeostatic responses of 19 young adults (mean age 22.4 years) and 19 normal elderly subjects (mean age 71.4 years). Each participated in four separate 2-day sessions that consisted of a baseline night, a nap, and post-nap sleep. Nap times were 0900, 1200, 1500 and 1800 h. The 1800 h nap contained the largest amount of delta and produced the largest reduction in post-nap delta. The young and elderly groups respectively produced 28 and 24% of baseline delta in the 1800 h nap. Both groups showed equivalent delta regulation, reducing post-nap delta by 28 and 25%, respectively. In both age groups, the decrease in post-nap delta resulted from a reduced rate of delta production (power/min) and reduced non-rapid eye movement (NREM) sleep duration. Period-amplitude analysis showed that the reduction in power/min resulted from decreases in delta wave amplitude and incidence. None of the responses to nap challenges differed significantly across age groups nor were there gender differences or age by gender interactions. These results show that delta homeostatic responses to naps in the elderly parallel those of young subjects. REM sleep showed no homeostatic reductions following naps in either group. We believe that the striking differences in the delta and REM responses point to different biological roles of the two kinds of sleep.

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Keywords: Aging; Sleep; EEG; Delta; REM; Naps

1. Introduction

Understanding the relationship of sleep to age over the human life span is fundamentally important to gerontology. This relation holds clues to the nature of brain maturation and aging as well as to the function of sleep. The deep sleep EEG shows the most marked age related changes. Deep sleep is characterized by high amplitude slow (delta) waves (visually scored stage 4 or computer-measured 0.3–3 Hz power). The age changes in delta gain interest because this stage of sleep reflects the homeostatic balance of sleep and waking [5,15]. Thus, the intensity (power in 0.3–3 Hz EEG) of NREM delta increases as the duration of prior waking increases. Delta intensity declines systematically across sleep as the homeostatic balance is restored. The age related reduction in deep sleep is most rapid during late childhood and adolescence. However, delta continues to decline slowly during adulthood. As a result, delta EEG power at age 70 years is about 50% of the level at age 20 years [14,17,20,28].

In addition to EEG changes, aging alters the sleep-wake cycle in ways perceptible to the individual. The most prominent change is an increase in the amount of waking across adulthood [2]. The number of awakenings increases linearly across the life span. The duration of awakenings begins to increase in midlife and accelerates in the fifth to seventh decade [18]. The resulting fragmentation of sleep in the elderly often produces complaints of insomnia that are difficult to treat. Current hypnotics act at the GABA–benzodiazepine complex. They provide short-term relief for most patients, but increase the risk of falls and produce tolerance that can progress to dependence. Furthermore, current hypnotics, including the non-benzodiazepine zolpidem, distort the sleep EEG pattern by decreasing the slow wave EEG that is an indicator of homeostasis [22]. The functional significance of these distortions is unknown. One possibility is that they cause less restorative sleep, a frequent complaint of patients taking hypnotics.

The sleep fragmentation in the elderly is likely an aging or degenerative phenomenon. The delta EEG decline across adulthood is also usually interpreted as an aging or degenerative change. However we have recently hypothesized that the same processes that cause the maturational decrease in
delta across childhood-adolescence produce the slower delta decline during adulthood [16,17]. It is generally acknowledged that better treatments for insomnia are needed. A step toward more rational pharmacotherapy would be to establish the physiological causes of insomnia in the elderly. One possibility is that sleep fragmentation in the elderly results from impaired homeostatic sleep regulation. An alternative possibility is that this age change is caused by impaired circadian regulation.

There have been a relatively large number of studies comparing the amplitude and period of circadian rhythms in young and elderly subjects (reviewed in [3]). Fewer studies have examined homeostatic sleep responses in the elderly. The experiments thus far have tested the response to the increased homeostatic need imposed by sleep deprivation. These studies have shown that the elderly can increase visually scored slow wave sleep following prolonged waking [4,8,33,37]. The converse challenge—decreasing homeostatic need by reducing waking duration with daytime naps—has not previously been investigated in the elderly. Nap challenges are also clinically interesting because the elderly often nap and the consequences of such naps for noc- turnal sleep have not been thoroughly tested. Monk et al. [29] have shown that afternoon napping reduces nighttime sleep efficiency in the elderly, but the effects on slow wave EEG were not reported. Compared to sleep deprivation, nap chal- lenges may provide a more quantitative test of homeostatic mechanisms because the average delta response to naps is more precise. In young adults, the amount of delta expressed in naps taken late in the day is subtracted from post-nap sleep. We also examined the homeostatic responses of young and elderly normal subjects to naps taken at four different times of day. Our first report showed that delta increases linearly across daytime naps and declines linearly across non-REM periods (NREMPs) on a baseline night [17]. Because absolute delta is greatly reduced in the elderly, their linear slopes across waking and sleep are significantly flatter. However, when the data are normalized to each subject’s baseline level, the slopes of the two age groups are essentially the same. This means that the normalized homeostatic delta need in young and elderly subjects increases at about the same rate per hour of waking and declines at similar rates across NREM periods.

This article is the second of a series of papers that compare the responses of young and elderly normal subjects to naps taken at different times of day. Our first report showed that delta increases linearly across daytime naps and declines linearly across non-REM periods (NREMPs) on a baseline night [17]. Because absolute delta is greatly reduced in the elderly, their linear slopes across waking and sleep are significantly flatter. However, when the data are normalized to each subject’s baseline level, the slopes of the two age groups are essentially the same. This means that the normalized homeostatic delta need in young and elderly subjects increases at about the same rate per hour of waking and declines at similar rates across NREM periods.

In this report we compared the ability of young and normal elderly subjects to conserve delta across nap and post-nap sleep. We also examined the homeostatic responses of NREM and REM durations in the two groups.

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (years)</th>
<th>Mean</th>
<th>S.D</th>
<th>Median</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young (n = 19)</td>
<td>22.4</td>
<td>1.4</td>
<td>22.0</td>
<td>20.2–25.1</td>
<td></td>
</tr>
<tr>
<td>Female (n = 10)</td>
<td>22.6</td>
<td>1.7</td>
<td>22.2</td>
<td>20.2–25.1</td>
<td></td>
</tr>
<tr>
<td>Male (n = 9)</td>
<td>22.2</td>
<td>1.0</td>
<td>22.0</td>
<td>20.8–24.2</td>
<td></td>
</tr>
<tr>
<td>Elderly (n = 19)</td>
<td>71.4</td>
<td>4.9</td>
<td>70.5</td>
<td>65.3–81.8</td>
<td></td>
</tr>
<tr>
<td>Female (n = 11)</td>
<td>72.3</td>
<td>4.8</td>
<td>72.0</td>
<td>66.4–81.8</td>
<td></td>
</tr>
<tr>
<td>Male (n = 8)</td>
<td>70.2</td>
<td>5.0</td>
<td>69.5</td>
<td>65.3–78.8</td>
<td></td>
</tr>
</tbody>
</table>

2. Methods
2.1. Subjects

Nineteen normal young adult subjects and 19 normal elderly subjects (Table 1) gave informed consent and received payment for participation in the study. Young adults were recruited among UC Davis graduate and undergradu- ate students. Elderly subjects were recruited from the Davis community with newspaper advertisements and presenta- tions at senior centers. Prospective subjects were initially screened via a medical history interview. Those with cur- rent psychiatric illness, or a history of head injury, epilepsy, or stroke were excluded. Subjects using medication with known CNS effects were also rejected. Urinalysis con- firmed that subjects were not using prohibited prescription or non-prescription drugs. Habitual nappers and subjects with known sleep abnormalities were also excluded. The final screening was a two night polygraphic recording in the laboratory. Young subjects who slept less than 5 h or had less than 15% combined stages 3 and 4 were excluded. Elderly subjects who slept less than 5 h were excluded. Sub- jects whose ability to sleep in the laboratory was compro- mised by frequent nighttime awakenings or arousals were excluded.

2.2. Study design

Each subject participated in four 2-day recording ses- sions. Each session consisted of a baseline night, a nap the following day, and a post-nap night. Nap times were 0900, 1200, 1500, and 1800 h. Each subject completed all four nap times with the order varied according to the subjects’ personal schedules. Their busy schedules made systematic or non-random variation of the nap order impractical. Intervals between recording sessions varied from 4 days to several weeks. The median interval between two recording sessions was 11 days for young adults and 9 days for elderly. Subjects repeated the 2-day session if their total sleep in the sched- uled nap was less than the 25 min minimum. Subjects who failed to meet this criterion in three sessions were dropped from the study. One elderly subject and two young adults were eliminated for this reason.

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Nine young adult and 19 elderly subjects passed all the initial screenings and completed all four naps. All elderly subjects and seven of the young subjects were in bed from 23:00 to 07:00 h on baseline and post-nap nights. Twelve young adults had been recorded with a 23:30–07:00 h time-in-bed schedule before any elderly subjects were studied. We had intended to use the 23:30–07:00 h schedule for all subjects, but the elderly proved unwilling to stay up until 23:30 h. Therefore, all elderly, and the remaining seven young subjects were studied with 23:00–07:00 h time-in-bed schedules. These different time-in-bed schedules did not produce significant differences in NREM or REM durations in the two subsets of young adults. The seven young adults who were in bed from 23:00 to 07:00 h had more wake time. All subjects maintained the experimental time-in-bed for the 3 days prior to each study. We explained the importance of maintaining this schedule and of not napping during the day. Each night subjects were questioned about their compliance. Subjects who reported inadvertent daytime napping were rescheduled. To further document compliance, subjects phoned the laboratory upon retiring and upon awakening. These calls were recorded and stamped with the time and phone number of origin.

Sleep EEG was recorded on baseline nights, naps and post-nap nights in the same individual bedroom of a four-bed sleep laboratory. Subjects reported to the laboratory 1–1.5 h prior to bed time for electrode application. Electrode impedance was less than 5 kΩ at the time of application. EEG was recorded from electrodes applied at C3, C4 and O1 referred to the contralateral mastoid. EOG was recorded between the left outer canthus and the mid-forehead. Sigma1 referred to the contralateral mastoid. EOG was recorded at the time of application.

PASS PLUS (Delta Software, St. Louis) software performed period amplitude and power spectral analysis on EEG in all artifact-free 20 s epochs. Power spectral analysis was configured to perform the fast Fourier transform (FFT) on 5.12 s Welch tapered windows with 2.62 s overlap, producing eight windows per epoch. Epochs containing artifact were included in the sum of vigilance states but were excluded from EEG results.

2.4. Scoring

The digitized EEG and eye movement leads were displayed on a computer monitor in 20 s epochs. These epochs were scored visually as NREM, REM and waking according to modified Rechtschaffen and Kales criteria [32]. A second individual checked the scored sleep data, and a third person resolved differences. Epochs with artifact were also marked. Artifacts included low frequency waves caused by sweating or loose electrodes, high frequency EMG artifacts, and 60 Hz activity. Artifact-containing epochs made up about 4% of the total NREM and REM epochs on baseline nights. The visually scored data were linked to the computer EEG analyses for each epoch. Epochs containing artifact were included in the sum of vigilance states but were excluded from EEG results.

2.5. Evaluation of delta EEG in naps and post-nap sleep

We first investigated delta homeostasis by testing whether the naps reduced total 0.3–3 Hz power and integrated amplitude on the post-nap night. To do this we established each subject’s baseline delta production as his/her mean across the four baseline nights of the four experimental sessions. We then compared this mean baseline value with delta power in each of the four post-nap nights using a repeated measures analysis of variance (ANOVA) with age and gender grouping factors. When the ANOVA was significant at α = 0.05 each post-nap night was compared against the baseline mean with post-hoc t-tests using Bonferroni corrections for multiple comparisons. We also examined whether the delta reduction on post-nap nights approximated the amount of delta in the naps. Similar evaluations were made for NREM and REM durations.

We examined the age and gender effects on NREM delta power across the four naps with a repeated measures ANOVA with age and gender as grouping factors. We decided, a priori, to separately evaluate the delta in the young and elderly groups even if the initial ANOVAs revealed no age by nap interaction. Similar ANOVAs evaluated age and gender effects on trends in NREM duration and REM duration across the four naps and the trends in delta power, NREM duration and REM duration across the four post-nap nights. Data were normalized as percents of each subject’s four night baseline mean values for presentation in figures and in tables.
Normalized data were also used in all statistical analyses not involving direct comparisons with baseline values. Because baseline values normalized as a percent of baseline are by definition 100% and therefore have no error, raw data were used for statistical comparisons involving the four night baseline means.

3. Results

3.1. Nap effects on post-nap night NREM and REM durations

Table 2 presents polysomnography data for the mean of the four baseline nights, the 1800 h nap, and the 1800 h post-nap night. The elderly had less total sleep time, less NREM, and less REM on baseline nights, naps, and post-nap nights. On the baseline nights, sleep latency was approximately 17 min in both the young and elderly groups, but the elderly had more wake time. This was due to both decreased total sleep time and the longer time in bed requested by the elderly subjects.

NREM duration in the naps did not increase substantially or significantly \((F_{1,102} = 1.80, \ P = 0.15)\) as naps were taken later in the day (Fig. 1). Nor were there any night NREM duration. In separate analyses of the young and elderly subjects, there were no significant \((F_{1,34} = 0.19, \ P = 0.99)\) or gender effects \((F_{1,34} = 0.65, \ P = 0.43)\) on NREM duration in the naps. Post-nap night NREM sleep decreased following naps, and the decrease was greater following later naps \((F_{1,102} = 5.02, \ P = 0.0027)\). There were no age \((F_{1,34} = 0.03, \ P = 0.85)\) or gender \((F_{1,34} = 0.76, \ P = 0.39)\) effects on the post-nap night NREM duration. In separate analyses of the young and elderly groups, post-hoc multiple comparisons showed that the decrease from baseline in post-nap NREM durations was statistically significant (at \(\alpha = 0.01\)) following the 1500 and 1800 h naps. In addition, post-nap NREM durations were significantly decreased on the post-nap night for the 1200 h nap in the young adults and for the 0900 h nap in the elderly.

The sum of nap plus post-nap NREM durations significantly exceeded baseline levels \((P < 0.0005\) for all nap times in both young and elderly) even when post-nap NREM duration was significantly reduced (Fig. 2). Although, as shown below, a post-nap decrease in NREM duration contributed to delta conservation, NREM duration itself was not conserved.

REM sleep duration in naps decreased \((F_{1,102} = 7.93, \ P = 0.0001)\) as naps were taken later in the day (Fig. 1), and the elderly had significantly \((F_{1,34} = 7.93, \ P = 0.0001)\) less REM in the naps than young adults. Although there was no significant \((F_{1,102} = 0.98, \ P = 0.41)\) interaction between age and nap time effects on REM duration in the naps, separate analyses showed the decreasing trend to be significant only in the young adults. In young adults stage REM duration decreased from a mean of 28 ± 4 min in the 0900 h nap to 13 ± 3 min in the 1800 h nap (significant effect of nap time, \(F_{1,34} = 5.03, \ P = 0.038)\). In the elderly REM duration decreased from a mean 11 ± 3 min in the 0900 h nap to 4 ± 1 min in the 1800 h nap \((F_{1,34} = 2.28, \ P = 0.10)\). REM in naps did not reduce REM duration in post-nap sleep in either group. In young adults post-nap REM duration was slightly but not significantly elevated following the afternoon naps, and in the elderly post-nap REM duration was slightly but not significantly decreased. For young adults the sum of nap and post-nap REM duration significantly exceeded baseline levels \((P < 0.01\) for all nap times) by about 25% (Fig. 3). In the elderly who had small amounts of REM in the naps, nap-post nap REM duration did not differ from baseline.

3.2. Nap effects on post-nap night delta power

3.2.1. Age and gender effects

As previously reported \([17]\), delta power in the naps increased as naps were taken later in the day, i.e. were pre-

### Table 2

<table>
<thead>
<tr>
<th>Vigilance state data for four night baseline mean, 1800 h nap, and 1800 h post-nap night</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time in bed* (min)</td>
</tr>
<tr>
<td>---</td>
</tr>
<tr>
<td><strong>Baseline mean</strong></td>
</tr>
<tr>
<td>Young adult Mean</td>
</tr>
<tr>
<td>S.E.</td>
</tr>
<tr>
<td>Elderly Mean</td>
</tr>
<tr>
<td>S.E.</td>
</tr>
<tr>
<td><strong>1800 h nap</strong></td>
</tr>
<tr>
<td>Young adult Mean</td>
</tr>
<tr>
<td>S.E.</td>
</tr>
<tr>
<td>Elderly Mean</td>
</tr>
<tr>
<td>S.E.</td>
</tr>
<tr>
<td><strong>1800 h post-nap</strong></td>
</tr>
<tr>
<td>Young adult Mean</td>
</tr>
<tr>
<td>S.E.</td>
</tr>
<tr>
<td>Elderly Mean</td>
</tr>
<tr>
<td>S.E.</td>
</tr>
</tbody>
</table>

* Scheduled time in bed. Actual time in bed may differ slightly.
† Does not include movement or stage 1.
Fig. 1. NREM delta power, NREM duration, and REM duration in naps (filled circles) and post-nap nights (open circles). Nap data are means of each subject’s data expressed as a percent of that subject’s four night baseline mean. Post-nap night data are means of each subject’s data expressed as percent change from that subject’s four night baseline mean. In both young and elderly subjects, NREM delta power in naps increased significantly as naps were taken later in the day and NREM delta power on the post-nap night decreased significantly as naps were taken later in the day. Nap NREM duration did not change significantly across the four nap times in either group. However, for the young adults post-nap night NREM duration decreased more following later naps. REM duration decreased significantly across the naps in the young adults, but the decrease was not significant in the elderly. Post-nap REM duration did not change from baseline in the elderly but increased significantly in the young adults following later naps.

3.2. Young adults
Examined separately in the young adult group, delta increased significantly \((F_{3,54} = 14.2, P < 0.0001)\) as naps were taken later in the day. Expressed as a percent of the amount of total delta power on the baseline night, delta power in the naps increased from (mean ± S.E.) 11.4 ± 1.3% in the 0900 h nap to 27.9 ± 2.7% in the 1800 h nap. As the amount of delta in the naps increased, total delta power on the post-nap night decreased significantly \((F_{3,54} = 16.4, P < 0.0001)\) (Fig. 1). However, the decrease from baseline in total delta power on the post-nap night was significant only following the 1500h (paired \(t_{18} = 4.61, P = 0.0001\)) and 1800h (paired \(t_{18} = 4.79, P < 0.0001\)) naps. For the 1800 h nap delta power on the post-nap night was 28.4% below the baseline mean, almost equal to the 27.9% (of baseline) delta expressed in the nap. Therefore, the sum of total NREM delta power in the nap and the total delta power on the post-nap night decreased by longer waking durations. Fig. 1 shows that this increase in delta as naps were taken later in the day was accompanied by decreasing delta in the delta on the post-nap nights. With delta power standardized as the percent of baseline power for each subject, ANOVA revealed no significant effects of age \((F_{1,34} = 1.62, P = 0.21)\), gender \((F_{1,34} = 3.77, P = 0.06)\) or age by gender interaction \((F_{1,34} = 0.02, P = 0.88)\) on delta in the naps. Nor was there any significant interaction between age \((F_{3,102} = 0.40, P = 0.75)\) or gender \((F_{3,102} = 0.67, P = 0.57)\) and the increase in delta as naps were taken later in the day. There were also no effects of age \((F_{1,34} = 0.23, P = 0.64)\), gender \((F_{1,34} = 0.00, P = 0.03)\) or age by gender interaction \((F_{1,34} = 1.24, P = 0.27)\) on the amount of delta on the post-nap nights. There were no significant interactions between age \((F_{3,102} = 0.84, P = 0.48)\) or gender \((F_{3,102} = 0.50, P = 0.68)\) and the amount of the delta decrease across the post-nap nights.
closely approximated total delta power on the baseline night (Fig. 4).

The conservation of delta in the young adult group was an average result that did not hold for every individual. For the 1800 h nap, nap + post-nap night delta sums varied from 74 to 145% of baseline. For the 1800 h nap, nap + post-nap night delta sums ranged from 74 to 145% of baseline. Furthermore, although delta power in the 1800 h nap was significantly correlated ($r = 0.50, P = 0.001$) with the reduction in post-nap night delta, this correlation was absent for the 1500 h nap ($r = 0.01, P = 0.97$). The significant correlation for the 1800 h nap largely resulted from two outliers ($r = 0.26, P = 0.12$ with outliers removed).

3.2.3. Normal elderly

In the elderly, both the pattern of delta accumulation in the naps and the effects of the naps on post-nap delta resembled those in young adults. Again delta power tended to increase as naps were taken later in the day. In the 0900 h nap, total delta power was $9.2 \pm 0.8\%$ of baseline; in the 1800 h nap total delta power was $23.8 \pm 2.6\%$ of baseline. As in the young adults, post-nap delta power was significantly reduced following the 1500 and 1800 h naps. In the elderly but not the young subjects, delta power following the 0900 h nap was significantly below baseline (Fig. 1). As in the young adults, the reduction in delta power following the 1800 h nap (23.3% of baseline) was close to the delta power (23.8%) in that nap. For the elderly, the sum of nap and post-nap delta roughly equaled baseline values.
decreased in both young and elderly subjects and the effects did not significantly differ between the two age groups.

Average sample amplitude $9.6 \pm \ldots$

Time in delta band/min $3.7 \pm \ldots$

Integrated amplitude/min $12.7 \pm \ldots$

Delta power/min $19.3 \pm \ldots$

NREM duration $12.0 \pm \ldots$

Total delta power $28.4 \pm \ldots$

The reduction in wave amplitude (10%) was significantly amplitude/min and the 12% reduction in NREM duration. This change resulted from a 13% reduction in integrated amplitude decreased by 23% in the young adults. In the elderly post-nap night following the 1800 h nap, NREM delta is significantly reduced following the baseline. For the 0900 and 1200 h naps this sum exceeds the baseline night delta. In young adults, NREM total delta power in the post-nap night following the 1800 h nap was 28% below the baseline level. This decrease was produced by a 19% reduction in power/min and a 12% reduction in NREM duration. The same pattern was observed for the 25% reduction in total delta power in the elderly following the 1800 h nap. Delta power/min was decreased by 17% below baseline, and NREM duration by 9%.

A change in delta power/min can be produced by a change in delta wave amplitude and/or a change in delta wave incidence. Period amplitude analysis but not FFT can separate the effects on amplitude and incidence. Integrated amplitude is period amplitude measure homologous to power. On the post-nap night following the 1800 h nap, NREM delta integrated amplitude decreased by 23% in the young adults. This change resulted from a 13% reduction in integrated amplitude/min and the 12% reduction in NREM duration. The reduction in wave amplitude (10%) was significantly greater (paired $t = 3.14, P = 0.006$) than the reduction in incidence (4% reduction in TIB). In the elderly, the same analysis for the delta reduction after the 1800 h nap showed a somewhat different pattern. The 7% reduction in average sample amplitude did not differ significantly from the 5% reduction in TIB (paired $t = 0.80, P = 0.44$). However, across group comparisons of the young and elderly showed no significant difference between the young and elderly for the effect of the 1800 h nap on any delta EEG measure ($t$-test results in Table 3).

3.3. Composition of delta EEG compensation following the 1800h nap

Data are presented here, and summarized in Table 2, only for the 1800 h nap which had the largest effects on post-nap sleep. Total delta power in post-nap sleep could have been reduced by a lower rate of delta production (decreased delta power/min), a decreased NREM duration, or a combination of both. In young adults, NREM total delta power in the post-nap night following the 1800 h nap was 28% below the baseline level. This decrease was produced by a 19% reduction in power/min and a 12% reduction in NREM duration. The same pattern was observed for the 25% reduction in total delta power in the elderly following the 1800 h nap. Delta power/min was decreased by 17% below baseline, and NREM duration by 9%.

A change in delta power/min can be produced by a change in delta wave amplitude and/or a change in delta wave incidence. Period amplitude analysis but not FFT can separate the effects on amplitude and incidence. Integrated amplitude is the period amplitude measure homologous to power. On the post-nap night following the 1800 h nap, NREM delta integrated amplitude decreased by 23% in the young adults. This change resulted from a 13% reduction in integrated amplitude/min and the 12% reduction in NREM duration. The reduction in wave amplitude (10%) was significantly greater (paired $t = 3.14, P = 0.006$) than the reduction in incidence (4% reduction in TIB). In the elderly, the same analysis for the delta reduction after the 1800 h nap showed a somewhat different pattern. The 7% reduction in average sample amplitude did not differ significantly from the 5% reduction in TIB (paired $t = 0.80, P = 0.44$). However, across group comparisons of the young and elderly showed no significant difference between the young and elderly for the effect of the 1800 h nap on any delta EEG measure ($t$-test results in Table 3).

![Image](85x404 to 323x770)

Fig. 4. Comparison of total NREM delta power on the baseline night with the sum of nap and post-nap night total NREM delta power. In young adults, following the 1500 and 1800 h naps, post-nap night delta is reduced (significantly) by the amount of delta expressed in the naps so that the sum of the nap and post-nap delta equals the amount of delta on the baseline. For the 0900 and 1200 h naps this sum exceeds the baseline delta. In the elderly post-nap night delta is significantly reduced following all naps. The sum of nap and post-nap delta for the 1500 and 1800 h naps and also for the 0900 h nap is similar to the baseline night delta. This sum exceeds baseline delta for the 1200 h nap.

![Table 3](85x404 to 323x770)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Young adult</th>
<th>Elderly</th>
<th>Elderly vs. young adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decrease (%)</td>
<td>Paired $t$ vs. baseline</td>
<td>Paired $t$ vs. baseline</td>
<td>$t$-test</td>
</tr>
<tr>
<td>Total delta power</td>
<td>28.4 ± 4.5</td>
<td>$t = 2.8, P &lt; 0.0001$</td>
<td>$t = 1.9, P = 0.05$</td>
</tr>
<tr>
<td>NREM duration</td>
<td>12.0 ± 1.8</td>
<td>$t = 3.91, P = 0.005$</td>
<td>$t = 1.94, P = 0.06$</td>
</tr>
<tr>
<td>Delta power/min</td>
<td>19.3 ± 4.1</td>
<td>$t = 5.13, P = 0.001$</td>
<td>$t = 1.94, P = 0.06$</td>
</tr>
<tr>
<td>Total integrated amplitude</td>
<td>22.7 ± 3.3</td>
<td>$t = 5.40, P = 0.001$</td>
<td>$t = 1.94, P = 0.06$</td>
</tr>
<tr>
<td>Integrated amplitude/min</td>
<td>12.7 ± 2.8</td>
<td>$t = 5.44, P = 0.002$</td>
<td>$t = 1.94, P = 0.06$</td>
</tr>
<tr>
<td>Time in delta band/min</td>
<td>3.7 ± 1.5</td>
<td>$t = 5.11, P = 0.001$</td>
<td>$t = 1.94, P = 0.06$</td>
</tr>
<tr>
<td>Average sample amplitude</td>
<td>9.6 ± 1.9</td>
<td>$t = 4.74, P = 0.001$</td>
<td>$t = 1.94, P = 0.06$</td>
</tr>
</tbody>
</table>

Mean (±S.E.) of data standardized as percent decrease from each subject’s four night baseline mean. Nearly all delta measures were significantly decreased in both young and elderly subjects and the effects did not significantly differ between the two age groups.

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4. Discussion

These data shed light on the homeostatic regulation of sleep in young and elderly normal subjects as evaluated by the ability to conserve delta EEG across a nap and a post-nap night. The data also demonstrate that the mechanism of delta conservation is similar in both groups. Both young and elderly subjects maintain the homeostatic quota by reducing both the rate of delta production and the duration of NREM sleep. In both groups NREM duration was partially conserved, apparently at the service of delta conservation. REM duration was not conserved.

In this study, we tested the homeostatic effects of naps at four different times of day. Trends of effects across the four nap times are discussed briefly, but we focus the discussion on the 1800 h nap, which had the largest amount of delta and the greatest effect on nighttime sleep.

4.1. Regulation of delta EEG homeostasis

These experiments were based on the proposition that sleep homeostasis is demonstrated if the amount of delta EEG expressed in a nap when summed with the amount in the post-nap night, equals the amount of delta EEG in baseline night sleep. Such homeostatic regulation requires that the brain register the amount of delta in the nap, retain that information until night-time, and then reduce night-time delta in post-nap sleep by that amount. It seemed plausible that the elderly brain might lose the ability to accurately recall the amount of delta in the nap. Our data show this is not the case. Delta homeostasis evaluated by this test is remarkably similar in the young and elderly. In young adults NREM delta power in the 1800 h nap was 28% of the baseline night amount, and post-nap delta power was reduced by 28%. In the elderly group the 1800 h nap contained 24% of baseline amount of NREM delta power and post-nap delta power was reduced by 25%. The difference between the young and elderly subjects did not approach significance, suggesting that the elderly conserve delta as well as the young adults. Although we think it unlikely, it is possible that the 3% difference between groups in decreased delta represents a slight impairment in homeostatic regulation in the elderly.

Data from the other naps also point to roughly equal homeostatic regulation in the young and the elderly. Conservation of delta was similar in the two groups following the 1500 h nap and both groups had smaller delta reductions following the morning naps. Delta EEG in sleep is strongly related to the duration of prior waking. If delta regulation is similar in the two groups, the rate of growth of delta as naps are taken later in the day should be similar. With delta power normalized as a percent of baseline night power, both groups showed a significant increase in delta across the naps and ANOVA showed no significant interaction between age and the trend across naps or the trend across post-nap nights. We also found no effect of gender on any of our tests of delta regulation. Women have been reported to have higher levels of delta EEG [7]. However, with delta power normalized, we found no gender difference and no interaction between age and gender.

The delta conservation observed in the two later naps was an average result that was not observed in each individual subject. In fact, correlation coefficients computed between the amount of delta in the nap and the reduction of delta in post-nap sleep were negligible even though the group means showed almost perfect delta conservation. The inability to predict individual behavior from strong and highly robust group averages is typical for sleep EEG data. For example, the decreasing monotonic trend in delta across NREM periods is massively significant with F values often exceeding 100 [24]. Nevertheless, these trends cannot predict the sleep EEG dynamics of individual subjects who can have more delta EEG activity in the second or even third NREM period than in the first (cf. [23]).

The delta conservation found in the young adults confirms two previous investigations of this question with computer measurement [25,38]. The present results are also qualitatively consistent with data of Karacan et al. [27]. They measured delta visually as stage four EEG and found that it was reduced following afternoon naps. They correctly hypothesized that delta represented a “consummatory process”.

The demonstration here that the elderly maintain a homeostatic response to naps is consistent with visually scored data showing a homeostatic response to sleep deprivation. The elderly increase visually scored stages 3–4 following sleep deprivation to a degree similar to young adults [9,33,37]. However, recent studies with computer analysis raise the possibility that young adults have a stronger delta EEG response. Guadreau et al. [26] found that 25 h of sleep deprivation ending at the habitual waking time produced a larger relative (expressed as a percent of baseline) delta EEG response in young adults than in middle aged (40–60 years) adults. The circadian change imposed by this protocol prevents direct interpretation of the sleep homeostatic response. The elderly may have a higher arousal level at the habitual wake time that would diminish their delta response to sleep loss. Murck et al. [31] performed a 40 h sleep deprivation experiment with onset of recovery sleep at the normal bedtime. They found a smaller delta response in the elderly. This was primarily an endocrine study and subjects slept with an indwelling catheter. The catheter may have differentially reduced sleep depth in the elderly group. A sleep deprivation experiment using computer analysis of the EEG without circadian or other confounds is sorely needed.

4.2. Mechanisms of delta conservation in post-nap sleep

In both age groups, a combination of reduced delta EEG intensity and reduced NREM duration acted to maintain delta conservation. Thus, delta power/min and integrated amplitude/min, were significantly reduced on the post-nap nights. This reduction was caused by decreases in both...
the incidence and amplitude of delta waves. It is thought that the amplitude of delta waves recorded from the scalp is produced by synchronous membrane changes in large populations of cortical neurons. Reduced amplitude following the late naps could be produced by either a reduction in the size of the neuronal pool oscillating in synchrony and/or by smaller membrane changes per neuron. Comparison of the age groups found no significant difference between the young and elderly for any delta measure. However, the significantly greater effect on amplitude than on incidence in the young but not elderly suggests that there may be some difference in the mechanism that conserves delta. Moreover, we have not yet completed the analysis of nap effects on delta in each NREM period of the post-nap night. These will be presented in a future publication. Such analyses may reveal differences between how the young and elderly achieve all night delta conservation.

4.3. Homeostatic regulation, homeostatic “drive”, circadian regulation, and sleep disturbances

If homeostatic regulation is essentially intact in the elderly, impaired circadian regulation may cause their impaired sleep. Circadian regulation in the elderly has been extensively investigated, but the results are inconsistent (reviewed in [3]). Some studies [11,34,36] but not others [30] report that the amplitude of circadian rhythms is decreased in the elderly. Studies on the length of the endogenous circadian period and the presence of phase shifts in the elderly have also yielded inconsistent results (cf. [10,11,30]). In a clinically relevant study, Campbell and Murphy found that the body temperature minimum occurred earlier in normal elderly than in young adults. However, there were no differences in temperature rhythm between elderly subjects with insomnia and normal elderly [6]. Campbell and Murphy’s data argue against the possibility that changes in circadian rhythm alone explain sleep disturbances in the elderly. Dijk et al. [13] have proposed that an interaction between weakened homeostatic drive and weakened circadian regulation produces sleep disturbances in the elderly.

4.4. Partial NREM homeostasis across naps and post-nap sleep

NREM duration showed some signs of homeostatic regulation but did not meet all our criteria. In both the young adults and elderly, NREM duration was significantly reduced following the afternoon naps. However, in contrast to delta EEG, the sum of NREM duration in the naps and post-nap sleep exceeded rather than equaled baseline levels. An interesting and unexpected observation was that in both groups NREM duration did not increase significantly across naps, i.e. with increasing prior wake duration. This differs strikingly from the robust increase in delta. Thus, although its reduction on the post-nap nights served as part of the mechanism by which delta EEG is conserved, NREM duration itself is not homeostatically regulated. The elderly response did not differ from that of young adults in any tests of NREM homeostasis.

4.5. Absence of REM homeostasis across naps and post-nap sleep

The total absence of stage REM homeostasis across naps and post-nap sleep found here contrasts strongly with the behavior of delta EEG. This result is consistent with findings of previous nap studies [25,38] and also with investigations of extended sleep. When sleep is extended in young adults by increasing time in bed, REM duration increases by 50–100% [19,35]. This increase does not reduce the amount of REM on the following night or delay REM onset [19,35].

An important clue to the function of REM sleep is that its rate of occurrence increases with prior sleep but is inversely proportional to prior wake duration; in an important early study, Aserinsky showed that the same pattern holds for eye movement density [1]. In the present study REM duration decreased as naps were taken later in the day in both age groups. This decrease may be related to the circadian modulation of REM sleep that causes REM to be greatest at the end of the sleep period and to decrease across the day [12]. However, it is opposite to the trend one would expect if REM were homeostatically related to prior waking.

Although the quantity of REM sleep is not conserved its periodic occurrence in mammalian sleep indicates that it must serve some essential biological function. The data of the present experiment are consistent with our hypothesis that this function is intrinsic to sleep processes and does not directly involve homeostatic recovery from waking brain activity [16,17,23].

5. Conclusion

Nap studies can be a powerful probe for investigating the effects of aging on sleep-wake relations and offer an important complement to sleep deprivation experiments. Together, nap and deprivation studies could lead to new mod-
els of sleep and aging that provide clues to their biological mechanisms. We found that normal elderly subjects showed homeostatic responses to naps similar to those of normal young adults. It would be interesting to extend this nap protocol to young and elderly subjects with insomnia. Such studies could shed light on the possibility that disruption of homeostatic regulation causes age-related insomnia.

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