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# Sleepiness/Alertness on a Simulated Night Shift Following Sleep at Home with Triazolam

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**Summary:** Physiological sleep tendency during a simulated night shift schedule was examined in 15 middle-aged subjects following daytime sleep after administration of triazolam or placebo. A double-blind, counterbalanced, crossover design involving two tours of five laboratory nights and four daytime home sleep periods was used. Triazolam lengthened daytime sleep as measured by wrist actigraph and improved nighttime alertness as measured by the MSLT. Sleepiness was most profound during the early morning hours (0430 to 0630) but improved significantly across nights for both conditions. Repeated test of sustained wakefulness latencies and simulated assembly line task performance decreased slightly across the night, but there were no significant condition effects. Subjective data tended to support objective measures, although Stanford Sleepiness Scale ratings indicated that subjects did not perceive improved alertness at night after triazolam-aided daytime sleep. **Key Words:** Sleepiness—Shift work—Triazolam.

Recent attention has been given to the use of sedative–hypnotic medication for sleep and alertness disturbances associated with altered sleep/wake schedules. There appears to be little doubt that benzodiazepines reliably lengthen sleep attempted during normal waking hours, as well as reduce stage 1 sleep. This is true for experimental manipulations of the sleep/wake schedule (1–3), as well as for actual shift workers (4). On the other hand, measurement of alertness during waking hours (within an altered sleep/wake schedule), following benzodiazepine-treated sleep, has provided somewhat conflicting results.

Bonnet et al. (3) have demonstrated that increasing daytime sleep with triazolam on the day following a rather typical night of sleep does seem to promote alertness on the subsequent night (2300–0700). This was true for both 0.25- and 0.5-mg doses (but not 0.125 mg). Absolute mean increases on the multiple sleep latency test (MSLT) were quite small (1.6–2.2 min), however, especially when considering that daytime sleep was increased a mean of 73 min (0.25 mg) or 140 min (0.5 mg) compared to placebo. Seidel and colleagues (2) examined the effect of triazolam, flurazepam, and placebo upon daytime sleep and nighttime alertness during three consecutive 24-h periods. Triazolam 0.5 mg and both doses of flurazepam (15 mg and 30 mg) improved daytime sleep relative to placebo, whereas 0.25 mg triazolam did not. However, only 0.5 mg triazolam also significantly improved alertness on the MSLT at night. Once again the absolute mean increase in MSLT latency was rather modest (1.8 min). Triazolam (0.5 mg) increased daytime total sleep time (TST) by 85.9 min versus placebo.

In a prior study in our laboratory (4), triazolam (0.5 mg) significantly increased TST during daytime sleep relative to placebo on four consecutive days. However, the mean nighttime MSLT scores for triazolam and placebo did not differ. The failure to detect differences on the MSLT in this study may be due to the greater number of days of study, the younger age and smaller size of the sample, relatively greater sleep deprivation, or other factors.

In the present study, we have used a similar design with the modification that subjects slept at home to more accurately simulate real world shift worker behavior. Once again, our primary research focus was to determine if increasing sleep during the day, by administration of triazolam, would result in a significant change in alertness during typical night shift hours.

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## METHODS

### Subjects

Fifteen healthy volunteers (4 males, 11 females) with a mean age of 41.1 years (range: 32–53) were recruited through media advertisement. Participants were free of significant psychopathology, medical disorders, and central nervous system active medications as determined by physical examination, medical history, and laboratory tests. Each participant's habitual sleep time occurred during nighttime hours, and none had been on night shift or rotating shift for at least the past year. Subjects were screened for significant sleep disturbance by nocturnal polysomnogram. Subjects provided written informed consent and were paid for participation.

**TABLE 1.** *Laboratory schedule*

| | |
|---|---|
| TP0 | |
| 2100–2150 | SALT practice session |
| TP0–TP4 | |
| 2230 | MSLT |
| 2300 | RTSW |
| 2330 | SALT |
| 0030 | MSLT |
| 0100 | RTSW |
| 0130 | SALT |
| 0230 | MSLT |
| 0300 | RTSW |
| 0330 | SALT |
| 0430 | MSLT |
| 0500 | RTSW |
| 0530 | SALT |
| 0630 | MSLT |
| 0700 | RTSW |

SSS administered 5 min prior to each MSLT subtest.

### Procedures

Subjects practiced a simulated assembly line task (SALT) for about 3 h at the time of the physical. The SALT visual performance measure presents subjects with images of electronic circuit boards that pass across a video monitor as objects might travel on a typical assembly line conveyor belt. The participant uses a "mouse" interface with the computer and is required to perform quality control inspections on each object in order to identify and reject faulty "products" or repair certain types of defective boards. Subjects must also respond to "alarms," which represent assembly line down time. Subjects were told that if they did not maintain an unspecified minimal performance level, they would be terminated from the study. They were also instructed that they could earn extra money with good performance. An 80% correction rate during the final 50 min of training was deemed the minimum criteria for inclusion in the study. At the time of this study, the SALT was under development and later versions are now being used.

A wrist activity monitor (actigraph, Ambulatory Monitoring, Inc.) was attached to the subject's nondominant wrist on the nocturnal polysomnogram screening night to acquaint him/her with the procedure and to provide data for validation of actigraph-scored sleep. A second screening night was performed to acquaint subjects with the SALT performance and the MSLT procedures (described below) and to make sure that all subjects showed the normal circadian trough of alertness in the morning.

A double-blind counterbalanced design was employed. Subjects participated in two tours of five nighttime laboratory test periods (TP0–TP4) and four daytime home sleep periods (SP1–SP4), with tours separated by a minimum of 7 days of normally timed sleep. Subjects were instructed to take a nap at home, between 1700 and 1900, prior to arriving at the laboratory for the initial test period (TP0) of each tour. Subjects arrived at the laboratory at approximately 2030 on the first night of each tour for a 50-min SALT practice session from 2100 to 2150. Subjects were scheduled to arrive at 2130 on all other test periods. Electrodes for polysomnographic recordings were attached to subjects following the practice session on the first night, or upon their arrival to the laboratory on other nights. Transportation to and from the laboratory was provided via taxi.

The laboratory schedule is shown in Table 1. The MSLT and repeated test of sustained wakefulness (RTSW) (5) were administered, each at 2-h intervals, during all test periods beginning at 2230, respectively. Each subtest, for both MSLT and RTSW, was terminated after the appearance of stage 2, rapid eye movement (REM), or three consecutive epochs of stage 1. Sleep latency was scored as the first epoch of any sleep stage. The Stanford Sleepiness Scale (SSS), a subjective sleepiness scale ranging from 1 (wide awake) to 7 (almost in reverie), was administered 5 min prior to each MSLT subtest.

The first of four periods of work on the SALT began at 2330. Work periods were 50 min long and occurred at 2-h intervals. Subjects had a break of a minimum of 10 min duration between any consecutive procedure (SALT work period, MSLT subtest, or RTSW subtest).

An actigraph was attached to the subject's nondominant wrist at approximately 0700, just prior to the subject's departure from the laboratory. The actigraph was to remain attached until the subject returned to the laboratory, except for times of showering. Subjects were instructed to maintain a consistent bedtime for each day sleep period beginning sometime between 0730 and 0900 and to take medication 5 min prior to bedtime. Subjects were instructed to try to obtain all

**TABLE 2.** *Mean (standard deviation) actigraph estimated sleep duration in minutes for four consecutive daytime sleep periods*

| | SP1 | SP2 | SP3 | SP4 | $\bar{x}$ |
|---|---|---|---|---|---|
| Triazolam | 356.9 (63.3) | 371.5 (70.9) | 352.5 (55.1) | 335.5 (69.9) | 354.1 (47.0) |
| Placebo | 316.3 (79.1) | 319.2 (65.6) | 314.9 (82.9) | 328.1 (67.5) | 319.6 (64.3) |

of their sleep during one sleep period (i.e., refrain from napping) prior to test periods 1–4 and avoid caffeine, except between 1600 and 1900 during which time they were allowed one caffeinated beverage. Post-sleep questionnaires were completed upon awakening. Subjects completed daily activity logs documenting bedtime, end of sleep period, time of actigraph removal, unscheduled naps, and times of sedentary activities.

Actigraph measures of wrist movement were taken at 1-min epochs. Actigraph records were scored with sleep defined as the first of three successive epochs with activity level less than or equal to 20 units (accumulated movements). Wake was defined as any epoch with activity level greater than 20 units and those epochs with activity level less than or equal to 20 units not meeting the above criterion for sleep. These criteria were established by comparing actigraph recordings with polysomnographic recordings during the subject's screen night in the laboratory. Mean activity level per epoch for the 15 subjects ranged from a minimum of 0 to a maximum of 406. During sedentary activities (e.g., reading, watching TV), activity level ranged from 0 to 350, but there were rarely more than two consecutive epochs with accumulated movements less than 20 units.

All subjects received one capsule (either 0.25 mg triazolam or placebo) for the first sleep period of both tours. Subjects reporting less than 7 h of sleep on the first sleep period were instructed to take two capsules (i.e., 0.5 mg triazolam or placebo) for the remaining three sleep periods of the tour.

## RESULTS

Data were examined with analyses of variance for repeated measures. Six subjects received 0.5 mg triazolam on sleep periods 2–4 of the triazolam tour, whereas nine received 0.25 mg on these days. Fourteen of the 15 subjects received 2 placebo capsules on sleep periods 2–4 of the placebo tour.

Actigraph recordings were not obtained for three subjects on the screening night because of technical problems. Comparison of polysomnographically determined TST (mean 409.9 min) to the actigraph estimated sleep duration (ESD) (mean 414.4 min) for the remaining 12 subjects showed no reliable difference between the two measures. Further, there was at least 90% agreement between the two measures for 10 of the 12 subjects. This is consistent with others who have reported reliable estimates of TST with wrist activity monitors (6–8).

There was no reliable difference between triazolam and placebo conditions for estimated nap duration prior to arrival for the initial test period. Subjects estimated a mean nap duration of 38.2 min prior to the triazolam condition and 39.7 min for the placebo condition.

Table 2 shows mean actigraph ESD for triazolam and placebo across sleep periods. Actigraph recordings revealed a significant increase in ESD for subjects on triazolam versus placebo ($F = 10.5$; df = 1,14; $p < 0.006$). On the average, ESD was 34.5 min greater for the triazolam condition averaged across the four sleep periods. There was no reliable change across the four sleep periods and no condition by sleep period interaction.

Analysis of ESD by drug dosage revealed that dosage had relatively little effect. Mean ESD across SP2–SP4 for those taking 0.25 mg was 349.1 min as compared to 359.3 min for the 0.5-mg subjects. Mean ESD for SP1 was 385.0 min for those subjects remaining on 0.25 mg and 314.7 min for those whose dose was increased to 0.5 mg for SP2–SP4 ($F = 6.59$; df = 1,13; $p < 0.023$). This indicates that dose was rather successfully titrated in this study.

Data from TP0 were excluded from drug condition analyses because it did not follow a daytime sleep period. Separate analyses of MSLT, RTSW, and SALT data from TP0 showed no significant differences between the two tours. Table 3 contains mean MSLT and RTSW sleep latencies across the night for each test period of both drug conditions. MSLT data revealed a significant drug effect ($F = 9.407$; df = 1,14; $p < 0.008$) for test periods 1–4 with the overall mean latency in the triazolam tour being 10.31 min as compared to 8.28 min on placebo (see Fig. 1). There was also a reliable main effect for test period with mean sleep latency increasing from TP1 to TP4 in both drug conditions ($F = 2.206$; df = 3,12; $p < 0.001$). No drug × test period interaction was found.

Mean MSLT scores also showed a reliable time of night effect in both conditions ($F = 13.43$; df = 4,11; $p < 0.001$) across all test periods (see Fig. 2). Mean latencies typically were between 14 and 17 min at 2230

**TABLE 3.** *Sleep latency means and standard deviations (SD) for MSLT and RTSW subtests*

| Time | 2230 Mean (SD) | 0030 Mean (SD) | 0230 Mean (SD) | 0430 Mean (SD) | 0630 Mean (SD) | x̄ Mean (SD) |
|---|---|---|---|---|---|---|
| MSLT | | | | | | |
| Triazolam | | | | | | |
| TP0 | 15.9 (6.1) | 9.6 (7.7) | 5.3 (4.2) | 2.6 (2.1) | 2.5 (2.0) | 7.18 (3.5) |
| TP1 | 14.0 (7.1) | 10.4 (7.4) | 7.3 (6.2) | 4.5 (5.4) | 4.5 (5.2) | 8.14 (4.5) |
| TP2 | 15.1 (6.8) | 12.7 (6.1) | 10.9 (7.3) | 7.4 (6.4) | 5.6 (5.3) | 10.34 (5.0) |
| TP3 | 13.8 (8.2) | 13.1 (6.8) | 9.6 (7.4) | 6.3 (6.3) | 7.5 (6.2) | 10.06 (5.6) |
| TP4 | 16.3 (6.9) | 12.8 (6.5) | 11.4 (7.2) | 10.4 (6.9) | 12.5 (7.3) | 12.68 (5.5) |
| Placebo | | | | | | |
| TP0 | 13.7 (7.5) | 11.8 (7.6) | 7.5 (5.4) | 4.6 (5.1) | 3.1 (2.6) | 8.14 (4.8) |
| TP1 | 14.4 (7.1) | 9.9 (7.1) | 5.7 (4.4) | 4.4 (4.7) | 2.8 (2.3) | 7.44 (3.6) |
| TP2 | 13.7 (6.7) | 11.6 (6.7) | 8.0 (6.8) | 3.3 (1.7) | 2.5 (1.2) | 7.82 (3.6) |
| TP3 | 14.5 (6.8) | 8.2 (5.8) | 7.3 (6.0) | 5.9 (6.4) | 5.1 (5.2) | 8.20 (4.6) |
| TP4 | 15.7 (6.3) | 13.0 (7.4) | 8.5 (7.4) | 6.6 (6.2) | 4.4 (2.8) | 9.64 (4.8) |
| | 2300 Mean (SD) | 0100 Mean (SD) | 0300 Mean (SD) | 0500 Mean (SD) | 0700 Mean (SD) | x̄ Mean (SD) |
| RTSW | | | | | | |
| Triazolam | | | | | | |
| TP0 | 16.8 (4.8) | 12.6 (7.8) | 9.5 (7.2) | 8.0 (6.5) | 7.7 (6.8) | 10.91 (5.4) |
| TP1 | 17.1 (6.1) | 14.5 (6.3) | 9.9 (6.6) | 7.9 (6.2) | 8.5 (7.6) | 11.58 (5.2) |
| TP2 | 18.1 (4.4) | 15.6 (6.6) | 12.5 (6.3) | 10.6 (6.7) | 12.6 (6.9) | 13.87 (5.2) |
| TP3 | 16.3 (5.8) | 15.7 (5.9) | 13.3 (6.5) | 11.4 (8.0) | 12.6 (7.4) | 13.85 (5.2) |
| TP4 | 18.6 (4.1) | 17.1 (5.9) | 15.7 (6.2) | 13.9 (6.6) | 16.3 (5.9) | 16.31 (4.6) |
| Placebo | | | | | | |
| TP0 | 15.6 (5.4) | 13.7 (7.1) | 11.2 (7.0) | 7.8 (6.7) | 9.6 (7.4) | 11.6 (5.7) |
| TP1 | 16.7 (6.0) | 14.4 (6.6) | 9.6 (6.3) | 7.9 (7.0) | 8.1 (6.9) | 11.36 (5.4) |
| TP2 | 16.4 (5.5) | 13.0 (6.9) | 9.9 (6.8) | 9.0 (6.4) | 9.5 (7.1) | 11.57 (5.2) |
| TP3 | 16.9 (6.0) | 14.4 (7.7) | 12.1 (7.7) | 10.7 (7.7) | 11.8 (7.7) | 13.18 (5.6) |
| TP4 | 18.4 (3.7) | 12.3 (6.7) | 11.8 (8.2) | 10.4 (7.7) | 11.3 (6.7) | 12.84 (5.6) |

and were generally less than 8 min at 0430 and 0630. No significant interaction for condition × time of night was found. Furthermore, there was no condition × time of night × test period interaction.

RTSW data tended to be consistent with MSLT data, although variability was higher for this measure. There was a trend for a condition effect for RTSW sleep latencies in the same direction as the MSLT scores, with a mean sleep latency of 13.91 min for the triazolam tour and 12.24 min for the placebo tour; however, this difference was not significant ($p < 0.053$). RTSW sleep latencies did show a significant time of night effect ($F = 7.36$; $df = 4,11$; $p < 0.004$). No reliable interaction effects were found.

Three performance measures from the SALT were analyzed: 1) SCORE: the percentage of items correctly handled (accepted, repaired or rejected), 2) ATR: average time for repair/rejection, and 3) AAT: average time to respond to alarms. There were no significant condition effects for any of the three SALT measures. SCORE ($F = 3.75$; $df = 3,13$; $p < 0.041$) and ATR ($F = 4.21$; $df = 3,12$; $p < 0.03$) showed small but reliable time of night effects. No other reliable differences were found. Later versions of the SALT task have been shown to be more sensitive to sleepiness/alertness fluctuations and will be published elsewhere.

There was no significant difference between triazolam and placebo on the SSS, nor was there a main effect for test period. SSS scores collapsed across test periods revealed a significant time of night effect ($F = 17.2$; $df = 4,11$; $p < 0.001$). No interactions were observed.

Table 4 shows the means for the subjective reports from the post-sleep questionnaire for sleep periods 1–4. Subjective estimates tended to follow the same trend as objective measures of sleep. Subjects reported a significantly longer TST on triazolam ($x = 382.9$, $SD = 37.7$) than on placebo ($x = 325.0$, $SD = 50.5$) ($F = 27.2$; $df = 1,14$; $p < 0.001$). There was no main effect of sleep period for subjective estimates of TST. There was a significant drug × sleep period interaction effect ($F = 5.58$; $df = 3,12$; $p < 0.012$) for TST. Thus, subjects estimated that TST decreased slightly across sleep periods on triazolam, whereas TST on placebo was judged to increase slightly from SP1 to SP4.

Reported sleep latency did not differ between the two conditions. Subjects reported significantly fewer awakenings ($F = 7.609$; $df = 1,14$; $p < 0.015$) and

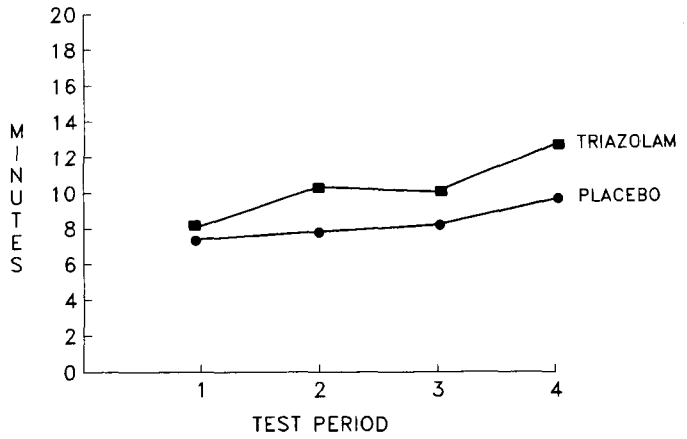


**FIG. 1.** Mean MSLT latencies for each test period (night) for the triazolam and placebo conditions. Each point represents the mean of five MSLT subtests.

higher quality sleep ($F = 12.5$; df = 1,14; $p < 0.003$) during the triazolam condition than during the placebo condition. They also felt that they slept more deeply on triazolam than placebo ($F = 8.294$; df = 1,14; $p < 0.012$) and were equally alert upon awakening in both conditions. A significant interaction effect for drug × sleep period was found for depth of sleep ($F = 5.24$; df = 3,12; $p < 0.015$). Subjective estimates of depth of sleep decreased slightly across days for the triazolam tour but increased across days on placebo.

A comparison of SP1 was made for drug dosage. As expected, there was a significant difference for subjective estimate of TST on SP1 between the 0.25 mg and 0.5 mg groups as the subjective estimates of total sleep time determined the drug dose for sleep periods 2–4. The 0.25-mg group estimated that they slept 437.8 min during the first triazolam sleep period, whereas the 0.5-mg group reported sleeping only 347.5 min ($F = 6.137$; df = 1,13; $p < 0.028$). As presented above, the data from the actigraph were consistent with this finding.

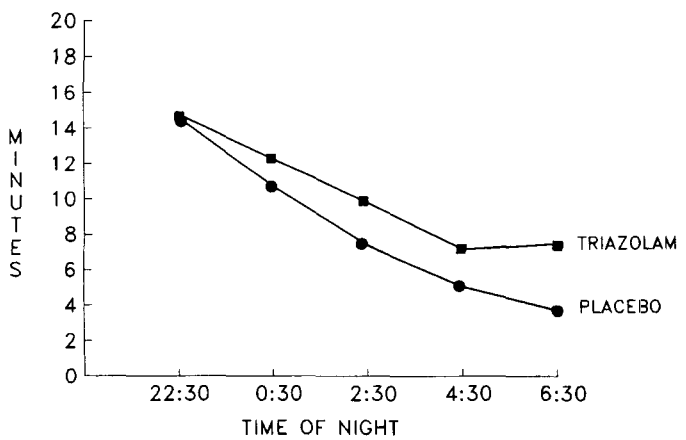


**FIG. 2.** MSLT latencies by time of night for the triazolam and placebo conditions. Each point represents the mean of four test periods.

**TABLE 4.** *Mean (SD) subjective reports of daytime sleep*

| | Triazolam | Placebo |
|---|---|---|
| TST (min) | 382.9 (37.7) | 325.0 (50.5) |
| SL (min) | 16.7 (10.1) | 27.2 (31.3) |
| Number of wakes | 1.4 (0.7) | 2.1 (1.1) |
| Sleep soundness[a] | 5.2 (0.8) | 4.4 (1.1) |
| Overall quality[b] | 5.2 (0.7) | 4.4 (1.1) |
| Feeling refreshed[a] | 3.9 (1.2) | 3.6 (1.1) |
| Sleepy now[a] | 2.4 (0.7) | 2.6 (0.7) |
| Feeling tense[a] | 1.8 (0.8) | 2.3 (1.0) |

[a] 1 = not at all; 7 = extremely.
[b] 2 = extremely bad; 7 = extremely good.

## DISCUSSION

Consistent with past studies, triazolam was shown to reliably increase sleep length (as estimated by wrist actigraph monitoring) during atypical sleeping hours. This was true for both 0.25 mg and 0.5 mg following dose adjustment according to subjective report. As expected, these middle-aged subjects tended to sleep less at home than a similar group in a laboratory study (1). Somewhat unexpected, however, was the finding that the mean increase in ESD with triazolam as compared to placebo in the current study was only 34.5 min. In our previous laboratory study, middle-aged subjects slept an average of 75.9 min more on triazolam compared to placebo during daytime sleep. We had expected a similar, or perhaps an enhanced, triazolam versus placebo difference, because subjects were more restricted in time of arising in the laboratory study. It is possible that, upon awakening at home, our subjects were not able to judge accurately whether or not they had fulfilled their sleep need. Or perhaps they simply chose to terminate the sleep period in order to do other things. Shift workers report sleep durations during daytime sleep that are quite consistent with those observed here; and it appears that although administration of a sedative–hypnotic significantly increases daytime sleep length, shift workers also require firm instruction to allow adequate time to sleep (return to sleep), even if taking sleep-promoting medication. In fact, such instruction may be most important when taking hypnotic medication because of potential carry-over sedation.

In addition to the increased ESD with triazolam, subjects consistently reported an increased depth and quality of their sleep on active medication. Although polysomnography was not performed in this study, prior work from our laboratory, and that of others, shows that triazolam, and benzodiazepines in general, have effects upon daytime sleep that are very similar to those seen for nocturnal sleep. That is, there are

reductions in the amount of stage 1 sleep and number and length of awakenings. It is likely that the subjective reports of higher quality sleep by our subjects with triazolam are at least in part related to these electrophysiological descriptions assumed to indicate deeper, more restorative sleep.

Perhaps the best measure of global sleep quality in healthy individuals, including amount of sleep, is sleepiness/alertness level during desired waking hours. The MSLT is currently the most well-established measure of physiological sleepiness/alertness. In the current study, the MSLT indicates a significant increase in alertness during typical night shift work hours following triazolam-aided daytime sleep. The triazolam-placebo difference in MSLT latencies suggests that triazolam reduces the severity of cumulative sleep loss. However, enhanced alertness at night appears to require more than 1 day of triazolam-aided daytime sleep.

MSLT data from a previous study (1) showed a tendency toward increased alertness at night with triazolam-aided daytime sleep. However, this trend did not reach statistical significance. This may be due to the fact that 9 of 18 subjects in that study were young adults, who, in the placebo condition, were relatively alert at night after only one or two night shifts. Review of MSLT data from only the middle-aged subjects in that study reveals considerable similarity to the current subjects in the profile of mean latencies across test periods. The data from both studies suggest that sedative–hypnotic medications may be more helpful for middle-aged shift workers as compared to young adults. Young adults have consistently reported less severe sleep/wake disturbances associated with shift work (9,10).

The MSLT profiles indicate increasing alertness across the four nighttime test periods for both triazolam and placebo conditions, similar to results of a previous study (1). This increase in alertness occurs despite a relatively constant amount of sleep during the four daytime sleep periods and is most likely an indication of circadian adaptation.

RTSW data were generally consistent with the MSLT findings, although more variable. This variability is that probable reason that statistical significance was not achieved for all comparisons.

The version of the SALT performance measure employed was barely sensitive to the profound dip in alertness across the night shown by the MSLT and RTSW. Therefore, it is not surprising that performance did not change significantly between drug conditions or across test periods. More recent versions of the SALT task currently under development appear substantially more sensitive.

Subjective reports clearly indicated that subjects perceived their sleep to be longer, deeper, and of higher quality on triazolam. We found no evidence of carry-over sedation. Interestingly, SSS ratings in the triazolam condition suggest that subjects did not perceive improved alertness at night, despite MSLT evidence that they were less sleepy. This probably reflects relative insensitivity of the SSS compared to the MSLT; however, subjects volunteered no information to indicate they perceived a difference in alertness. We have observed a similar finding in a study of caffeine prior to a simulated night shift. The MSLT clearly showed that caffeine increased alertness, but subjects' reports showed no awareness of this improvement (11).

The data from this study, and others, suggest that triazolam may be useful in the management of some patients with sleep/wake disturbances secondary to night shift work. It appears that middle-aged individuals are more likely to benefit than younger individuals, both in terms of improved sleep and enhanced alertness. As with any sedative–hypnotic, chronic use of triazolam in this population is not advised because of the potential development of tolerance and/or drug dependency insomnia. A more rational approach would involve use of a sedative–hypnotic only after night shift work with no drug used on days off. Most steady night workers have only 4 days per week on which daytime sleep comprises the main sleep period of the day. In the case of rotating shift workers, no drug should be needed when on the day or afternoon shifts.

Dosage should be carefully titrated so that the lowest therapeutic dose is used. The majority of subjects in this study showed improvement in daytime sleep and nighttime alertness with triazolam 0.25 mg. Although dosage was increased to 0.5 mg following the first daytime sleep period for six of our subjects, we do not know whether these individuals would have shown improved sleep or alertness with the lower dose. The possibility of carry-over and withdrawal effects with the higher dose indicate it should be used with significant caution. Shift workers using triazolam should be properly informed about the possibility of carry-over sedation, particularly at higher doses and in circumstances in which patients choose early termination of their sleep period. Previous studies, however, suggest that carry-over sedation is not a significant problem with triazolam at doses up to 0.5 mg in normals sleeping at night (12,13) or following a 12-h shift in the sleep/wake schedule (2). Insomniacs given 0.5 mg triazolam also showed no decrease in daytime alertness (14,15).

Certainly, there are some night shift situations in which use of triazolam, or any sedative–hypnotic, is contraindicated. Individuals who have unpredictable schedules and who may be required to function during hours planned for sleep should not take sedative–hypnotics. General contraindications for sedative–hyp-

notic therapy, such as a history compatible with sleep apnea or substance abuse, also apply.

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