Sleep, 18(7):581–588

24-Hour Metabolic Rate in Insomniacs and Matched Normal Sleepers

M. H. Bonnet and D. L. Arand

Dayton VA Hospital, Wright State University, and Kettering Medical Center, Dayton, Ohio, U.S.A.

Summary: Groups of 10 objectively defined insomniacs and age-, sex- and weight-matched normal sleepers were evaluated on sleep, performance, mood, personality and metabolic measures over a 36-hour sleep laboratory stay. Insomniacs were defined to have increased wake time during the night but also had decreased stage 2 and rapid eye movement sleep. As expected insomniacs reported increased confusion, tension and depression and decreased vigor on the profile of mood states mood scale throughout the evaluation period as compared to the normals. Insomniacs also had decreased memory ability on the short-term memory test and the MAST. These performance and mood differences were not secondary to sleepiness because the insomniacs also had significantly increased multiple sleep latency test (MSLT) values throughout the evaluation period. In conjunction with the consistent mood, performance and MSLT differences during the day and the sleep differences at night, whole body $\dot{V}O_2$, measured at intervals across the day and throughout one night of sleep, was consistently elevated at all measurement points in the insomniacs as compared to the normals. The nocturnal increase in metabolic rate remained even after metabolic values from periods during the night containing wake time or arousals were eliminated from the data set. It was concluded that patients who report chronic insomnia may suffer from a more general disorder of hyperarousal (as measured here by a 24-hour increase in metabolic rate) that may be responsible for both the daytime symptoms and the nocturnal poor sleep. Future studies need to explore 24-hour insomnia treatment strategies that decrease hyperarousal. **Key Words:** Insomnia—Metabolic rate—Psychomotor performance—MSLT—Mood—Personality—Sleep.

Because patients with insomnia frequently have a symptom complex that includes tension, anxiety, fatigue and irritability (1,2), as well as characteristic Minnesota multiphasic personality inventory (MMPI) elevations (3,4) and an insomnia onset following a significant life-stress event (5), many investigators hypothesize that insomnia is the result of internalization of emotions producing emotional arousal. As such, some treatment strategies try to reduce cognitive hyperactivity (6). More traditional thought concerning the development of insomnia has held that the emotional arousal hypothesized above results in physiological activation, which leads to insomnia (7) or even that insomnia develops entirely from physiological activation, as in phase-shift insomnia. Several studies have found significantly increased physiological activation in patients with insomnia. For example, Monroe (8) reported increased rectal temperature, heart rate, basal skin resistance and phasic vasoconstrictions 30 minutes prior to and during sleep in insomniacs as compared to normal sleepers. Careful studies of sleep-onset insomniacs have shown that prior to sleep onset, patients had increased frontalis (9) and mentalis electromyogram (EMG), increased heart rate (10), increased finger temperature, and more β and less α frequencies in their electroencephalogram (EEG) (11,12). In a study that included sleep maintenance insomniacs, the all night heart rate elevation but not the increased vasoconstrictions reported by Monroe (8) were replicated (13). However, significantly elevated body temperature has not been reported in all studies of poor sleepers (14,15). Poor sleepers have increased secretion of corticosteroids and adrenaline (14,16) compared with good sleepers in most but not all studies (17). The inconsistent results in some of these physiological activation studies may indicate that physiological activation is not a major factor in at least some insomniacs

Accepted for publication April 1995.
Address correspondence and reprint requests to Michael H. Bonnet, Ph.D. (151N), VA Hospital, 4100 W. Third Street, Dayton, Ohio 45428, U.S.A.

(6) or that wide variability and small sample sizes may make it difficult to show clear physiological differences. It may be the case that lack of control of daytime activity in the studies might have obscured differences. It is also possible that the involved physiological system(s) differ from patient to patient and that a global measure, such as whole body oxygen use, would more consistently show differences.

Another line of research has examined the daytime functioning of insomniacs to determine the existence of subjectively reported deficits in performance, mood, and alertness. While the cumulative partial sleep deprivation that should arise from chronic insomnia would be expected to produce daytime sleepiness or increased susceptibility to acute sleep loss in insomniacs, studies have consistently found that insomniacs are not sleepier than normal controls on multiple sleep latency tests (MSLTs) (15,18,19) or after sleep loss (20) and may actually have longer MSLT latencies (21,22). Studies have found that insomniacs made more errors on a line tracing task (23), produced fewer responses in a word category test (15), and performed worse on the Romberg (balance) test (24). These results may be interpreted as insomniacs producing worse performance on tests where too much arousal reduces steadiness or blocks higher order associates. However, studies comparing daytime performance in insomniacs to normal controls have generally not found differences on tests that are sensitive to sleep loss (19,24). Based upon the results of these studies and patient reports that they are fatigued or "washed out" during the day, investigators have hypothesized that standard sleep and sleep loss tests are confounded in that they "simultaneously measure sleep need and hyperarousal, which is interfering with sleep onset" (21). This concept is supported by studies (19,21,23) reporting significant negative correlations between total sleep at night and MSLT values on the next day.

In one study, the hyperarousal hypothesis was directly examined by administering 400 mg of caffeine three times a day (TID) to normal young adult sleepers for a week to produce pure physiological arousal independent of psychological factors (25). Level of arousal was documented by metabolic measures, and the normal young adults began to report many symptoms common to insomniacs including poor nocturnal sleep, increasing daytime fatigue even while their MSLT values were significantly longer than on baseline and increasing anger and tension as well as changes in the MMPI PT (anxiety) scale in the direction of psychopathology. If these symptoms and similar symptoms seen in insomnia patients are a direct result of the increased physiological arousal, it follows that metabolic rate should also be increased in insomniacs.

The presence of both poor sleep and daytime dysphoria and fatigue in insomniacs implies the presence of a 24-hour dysfunction. In the present study it was hypothesized that insomniacs have increased metabolic rates secondary to either direct physiological arousal or emotionally produced physiological arousal. To test this hypothesis, metabolic data were collected across the night and day in groups of insomniacs and carefully matched control subjects for a 36-hour period in the laboratory under standard conditions.

METHODS

Subjects

Subjects (Ss) were required to be healthy, 18–50-year-old males and females. Potential Ss were solicited from sleep center referrals and from ads in the local papers for participants in sleep research.

Insomniacs

Individuals completing a screening questionnaire indicating that they had a sleep problem and that it took them 45 minutes or more to fall asleep at least four nights each week or that they were awake for 60 minutes or more each night after falling asleep for at least four nights each week and that this condition had existed for at least 1 year were considered further (see common exclusions below).

Normals

Normals were required to indicate normal sleep on their screening questionnaire. They were also required to report a sleep latency of less than 30 minutes and less than 30 minutes of wake time during the night. Normal subjects were additionally required to match a qualified insomniac by sex, age (within 5 years), weight (within 25 pounds) and general time in bed characteristics.

Exclusions

Potential normal or insomniac subjects who indicated excessive caffeine consumption (more than 250 mg of caffeine per day), who were using psychoactive medication or drugs, or who had completed a drug or alcohol abuse program within the previous year were excluded. Ss with a history of depression or psychiatric hospitalization were excluded. Potential Ss who had histories strongly suggestive of circadian desynchrony (e.g. shift workers), sleep apnea, or periodic leg movements were excluded.

Subjects meeting the above criteria were invited to participate in the study after completing an informed

consent and 2 hours of acclimatization to the laboratory with practice on computer tests and questionnaires to be used in the study.

Design

After practice, subjects were scheduled to spend two nights and the intervening day in the laboratory. On both nights, a standard clinical polysomnogram, including two eye channels, central and occipital EEG channels, chin and leg EMG channels, electrocardiogram (EKG), airflow and chest movements, was performed. On the first night, SaO_2 was also recorded. On the second night, metabolic measures and a time code were recorded instead of SaO_2.

A schematic of the tests and times is presented in Table 1. On the morning of the day spent at the laboratory, an initial 20-minute metabolic measurement was performed immediately after awakening. Ss performed computer tests, completed an MMPI and a sleep history, and were fed the same daily menu of food prepared at the laboratory during the day. Caffeinated beverages were not available. Subjects usually did not leave the laboratory during this day and did not engage in any activity more vigorous than walking to the bathroom. Following the second night in the laboratory, subjects completed some brief computer tests and were allowed to leave.

All subjects were assigned their own room for the course of the study. Each room contained a standard hospital bed and furniture including a desk with an Apple IIGS computer. Subjects participated in the study in groups of one or two individuals. Subjects completed all tests and questionnaires at their individual computer workstation in their room under technician observation via video monitors. Ss were not allowed to sleep while performing computer tests (video monitoring) or during metabolic observations (EEG monitoring). Meals and breaks were scheduled in another area of the laboratory, which was also within technician observation.

TABLE 1. *Study design*

2100–2400	Practice tests
2400–0800	Polysomnogram
0800	Metabolic observation
	Awakening tests
	Breakfast
	Test battery
1000	MSLT
	Metabolic observation
	Test battery
1200	MSLT
	Metabolic observation
	Lunch
1400	MSLT
	Metabolic observation
	Test battery
1600	MSLT
	Metabolic observation
	Dinner
1800	MSLT
	Metabolic observation
2000	MSLT
	Metabolic observation
	Test battery
2400–0800	Polysomnogram with metabolic rate
0800	Postsleep tests

Test battery contents: Visual vigilance, tremor, MAST, word productivity, proofread, visual activation scale, POMS, oral temperature, short-term memory.

Tests

Performance and mood were assessed with a battery of measures including the MAST [1, 3 and 5 letters (26)], proofreading (10 minutes), hand tremor (2-minute insertion of a stylus into a 4-mm opening with percentage of side touching time measured), the digit symbol substitution task from the Wechsler adult intelligence scale (WAIS: 5 minutes) (27), computer-modified Williams word memory test of immediate free recall (28), visual vigilance (30 minutes) (29), subjective sleepiness (10-point analog scale), profile of mood states (POMS) and oral temperature. The tests were administered in repeated batteries, and the scheduling and contents of batteries are summarized in Table 1.

For all subjects on all measures except MSLT, performance during continuous operations was automatically scored by the computer and output in a format suitable for statistical analysis.

MSLT recordings

Four-channel sleep recordings (LE-A2, RE-A2, C3-A2, OZ-A1) were made during MSLT evaluations. Six MSLT evaluations were made during the study proper (at 1000, 1200, 1400, 1600, 1800 and 2000). For all MSLT evaluations, Ss were in bed for 20 minutes or until the first scorable epoch of non-stage 1 sleep. The latencies reported in this paper are all latencies to non-stage 1 sleep.

Metabolic measurements

All metabolic measurements were performed with a SensorMedics Deltatrac Metabolic Monitor. The Deltatrac generates a constant flow of 40 l/min through a canopy or mask and into the metabolic cart. The high flow pulls all expired air and a significant amount of room air from an external inlet into the machine. The

metabolic monitor then calculates the difference between the flow of air from the subject and a separate measure of pure room air to determine oxygen use and carbon dioxide production by the subject. In the current study, Ss were outfitted with a Puritan Bennett full-face resuscitation mask held in place with a continuous positive airway pressure (CPAP) head strap and connected to the input of the metabolic cart, which was located in the adjacent room next to the polygraph. Holes were drilled into the upper section of the Puritan Bennett mask to allow the required air inlet for the high flow system. Occasional leaks from the junction of the mask to the face would serve as an additional air inlet for this system and would not compromise data reliability. When the mask is removed, the Deltatrac provides an auditory alarm of "no breathing" in 10 seconds and notes these data on outputs.

In the current study, waking metabolic data were recorded for 20 minutes immediately after awakening following the first night in the laboratory. Metabolic data were also collected for 20 minutes immediately following each of the six MSLT evaluations. This placement gave a standard rest period prior to the initiation of all of these metabolic measurements. Additionally, 20 minutes of waking metabolic data were collected prior to lights out on the second night and throughout the entire second night of sleep. For all metabolic observations, $\dot{V}CO_2$, $\dot{V}O_2$, and respiratory quotient (RQ), among other variables, were automatically averaged and output by the metabolic cart at the end of each minute during the metabolic observation periods. In addition, the metabolic cart generated a time code that was used to stamp each metabolic observation and was also automatically written out onto the polygraph paper so that metabolic data could be associated with ongoing EEG on a minute by minute basis. For this report, only $\dot{V}O_2$ data are summarized. $\dot{V}CO_2$ data were very highly correlated with the $\dot{V}O_2$ data and are therefore not reported (30). The metabolic data from each pair of subjects were matched minute by minute throughout daytime and nighttime metabolic observation periods starting at the first morning metabolic observation.

For waking metabolic observations, Ss remained in bed and were instructed to move as little as possible. The lights in the room were turned on. However, reading or other activities involving movement or body posture were not allowed. During all metabolic recordings, EEG was recorded to insure wakefulness during waking observations. During all metabolic observations, Ss were also monitored by video camera to assure compliance with the protocol (i.e. no change in posture or removal of mask). Sections of recording scored as "no breathing" were removed prior to data analysis. As such, all remaining observations during the study should have fallen under the generally required assumption of steady-state conditions for the meaningful collection of metabolic data.

To qualify for the study as an insomniac, individuals were required to report insomnia as described earlier and to have EEG sleep latencies greater than 30 minutes on both laboratory nights *or* to have a sleep efficiency of less than 85% on both nights. Subjects with an apnea/hypopnea index greater than 10 or a periodic leg movement arousal index greater than 10 were disqualified. Of 60 insomniac subjects who completed the two-night protocol, 20 met all of the criteria.

To qualify for the study as a normal, individuals were required to report normal sleep as described earlier and to have EEG sleep latencies of less than 30 minutes on both laboratory nights and to have a sleep efficiency greater than 90% on both nights (and not to have sleep apnea or leg movements as defined above). Of 20 normal subjects who completed the two-night protocol, 10 met the above requirements and could be matched with one of the insomniacs on the basis of age, weight and sex. Therefore, the results reported in this paper reflect the data obtained from two groups of 10 age-, sex-, and weight-matched individuals (normals and insomniacs).

Analyses

Subjects were matched by age, weight, and sex specifically to allow within subject-pair comparisons. As such, paired *t* tests or repeated measures analyses of variance were performed on the paired data. Two analyses were planned for the metabolic data. First, for each subject pair, bedtimes were matched to begin each series as a control for circadian time, and all $\dot{V}O_2$ values were compared with a paired *t* test. It is possible that the insomniacs had higher $\dot{V}O_2$ during the night simply because they were awake more and had more body movements and arousals (all of these events would increase $\dot{V}O_2$). To examine this issue, the metabolic data were matched with the sleep data so that each metabolic observation was assigned the lightest stage of sleep for the two 30-second sleep epochs, which made up the 60-second metabolic observation for each metabolic observation. Then, all metabolic observations containing EEG-scored wake, movement or arousals greater than or equal to 3 seconds were eliminated from the data sets. Because arousal or awakening can increase metabolic rate for more than 1 minute, the two following metabolic observations were also eliminated from the data sets. The remaining metabolic data were matched again within subject pairs and the *t* tests were repeated. As a second test, metabolic data from slow-wave sleep (SWS: stages 3 and 4) were also compared within subject pairs.

TABLE 2. *Demographic data: mean and (standard deviation)*

	Insomniacs	Normals	*t* value	p
Number	10	10		
Age	38.3 (7.1)	38.6 (6.8)	0.260	NS
Weight (lbs.)	176 (27)	174 (24)	0.542	NS
Subject latency (minutes)	106 (93)	19 (6.6)	−3.010	0.015
Subjective total sleep (hours)	4.75 (1.7)	7.40 (0.8)	−4.976	0.001
Subjective time in bed (hours)	7.56 (1.9)	7.85 (0.6)	0.444	NS
Length of insomnia (years)	8	—	—	—

TABLE 3. *Sleep parameters: mean values with (average standard deviation)*

	Insomniacs	Normals	*F* value	p value
Total sleep (minutes)	342 (75)	442 (23)	22.19	0.001
% Stage 1	14.6 (9.8)	15.3 (7.5)	0.08	NS
% Stage 2	32.8 (14)	45.1 (11)	6.70	0.03
% Stage 3	7.8 (6.4)	7.2 (2.7)	0.16	NS
% Stage 4	5.2 (5.8)	5.8 (5.7)	0.17	NS
% REM	14.4 (5.4)	20.5 (4.6)	7.91	0.02
% Wake	25 (14)	5.9 (4.5)	21.23	0.001
Sleep latency (minutes)	20.5 (13)	5.8 (0.3)	12.46	0.01
Sleep efficiency	75 (14)	94 (3.4)	21.24	0.001
REM latency (minutes)	132 (69)	87 (44)	3.54	0.1
Arousal index	16.7 (16)	12.9 (7.5)	2.27	NS
Bed time	23:42 (1.6)	23:48 (1.2)	0.04	NS
Wake time	8:00 (1.0)	8:00 (1.1)	0.03	NS
Subjective latency (minutes)	56 (45)	18 (14)	8.48	0.02
Subjective time awake (minutes)	90 (68)	21 (34)	11.98	0.01
Subjective depth[a]	2.2 (0.7)	1.6 (0.6)	6.00	0.05
Subjective quality[a]	3.4 (1.1)	2.0 (1.0)	9.75	0.01

[a] Lower numbers refer to better or deeper.

RESULTS

Demographic data from the 10 pairs of normals and insomniacs can be found in Table 2. The groups did not differ in age, weight or time usually spent in bed per night. As expected by selection criteria, insomniacs reported significantly longer sleep latencies and shorter total sleep per night.

Sleep data from the two groups are presented in Table 3. The objective and subjective sleep data were analyzed by a two-way analysis of variance with terms for night (first and second), group and interaction. Significant interaction effects were not found for any variable, and significant night effects were not found except for the EEG sleep latency variable (latencies were shorter on the second night in both groups). This means that the metabolic apparatus used on the second night did not change sleep parameters in either group with respect to the first night. Because night one to night two differences were not found, only group mean data are presented in the table. It can be seen from the table that sleep was substantially different in the two groups based upon the inclusion criteria. Decreased total sleep time in the insomniac group came primarily from decreased stage 2 and rapid eye movement (REM). The insomniacs also reported significantly longer latencies, more wake time and decreased depth (3-point scale with 3 as light) and quality (5-point scale with 5 as poor) of sleep compared to the normals.

Daytime performance, mood and MSLT data are presented in Table 4. Because the daytime tests were presented in repeated batteries, the ANOVA for each measure included terms for time of day, group and interaction. Significant group by time interactions were not found for any variable. Therefore, the error variance was pooled to test for main effects for group. The resulting group means and *F* values are presented in Table 4. Insomniacs reported significantly more confusion, tension and depression as well as decreased vigor compared to the normals. Insomniacs also had decreased short-term memory and increased MSLT values as compared to normals. There was a group by difficulty (number of search letters) interaction on the MAST test. The interaction indicated that the normals completed more correct searches than the insomniacs in the one-letter search but that there were no group differences in the three- and five-letter searches.

The MMPI values are presented in Table 5. In these matched subjects, there were no significant differences on the MMPI although the insomniacs did score somewhat higher on depression and hysteria (both $p < 0.1$).

TABLE 4. *Mood and performance: mean and (average standard deviation)*

	Insomniacs	Normals	*F* value	p value
Short-term memory (words)	5.7 (3.7)	7.2 (2.7)	6.90	0.02
Proofread (lines)	246 (71)	258 (75)	0.69	NS
Vigilance P(A)	0.875 (0.15)	0.871 (0.16)	0.04	NS
MAST (1 target, correct)	53.2 (24)	60.5 (19)	11.0	0.001
MSLT (minutes)	13.3 (3.1)	9.5 (5.3)	9.05	0.01
POMS				
Confusion	5.4 (4.2)	3.1 (3.2)	9.18	0.01
Tension	6.0 (5.8)	4.0 (3.8)	4.28	0.05
Fatigue	5.7 (5.7)	3.6 (4.3)	3.25	NS
Anger	5.6 (9.0)	3.8 (4.7)	1.09	NS
Depression	9.0 (11.8)	4.1 (5.3)	5.22	0.05
Vigor	16.4 (6.7)	21.5 (7.5)	18.01	0.001

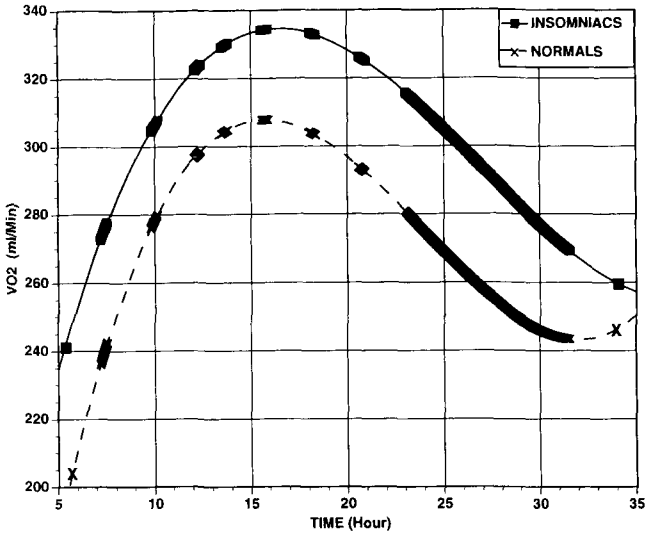

FIG. 1. Average best fit $\dot{V}O_2$ in groups of insomniac and normal patients as a function of time of day (see text). The data represent averaged best fit third degree polynomials. A marker has been added at the right and left margin of both lines to indicate the group.

TABLE 5. *MMPI* t *scores with (standard deviation)*

	Insomniacs	Normals	*t*	p
HS	57 (13)	51 (6.6)	1.215	ns
D	62 (13)	51 (11)	1.951	0.08
HY	62 (10)	54 (5.4)	2.033	0.07
PD	72 (12)	62 (14)	1.328	ns
MF	50 (7.7)	56 (12)	−1.155	ns
PA	60 (6.5)	56 (7.4)	1.04	ns
PT	58 (10)	54 (6.4)	0.973	ns
SC	59 (12)	57 (7.1)	0.476	ns
MA	60 (16)	64 (12)	−0.685	ns
SI	52 (12)	48 (9.3)	0.926	ns

Scales are hypochondriasis (HS), depression (D), hysteria (HY), psychopathic deviate (PD), masculinity/femininity (MF), paranoia (PA), psychasthenia (PT), schizophrenia (SC), hypomania (MA), and social introversion (SI).

Metabolic data

In the analysis containing all of the data, all 10 of the *t* values were in the same direction and 9 of 10 were statistically significant ($p < 0.01$). The average *t* value was 13.10 with 475 degrees of freedom ($p < 0.0001$). The respective means for $\dot{V}O_2$ for insomniacs and normals were: 296 and 266 ml/minute.

In the set of 10 *t* tests with awakenings, movements and arousals eliminated, 9 *t* values were in the predicted direction and 8 of 10 were statistically significant ($p < 0.02$). The average *t* value was 13.38 with an average of 140 degrees of freedom ($p < 0.0001$). The overall mean sleep metabolic rates for the insomniac and normal groups respectively were: 280 and 256 ml/minute.

When SWS was examined, it was found that two subjects (one insomniac and one normal) had no stage 3 or stage 4 sleep. Because the two subjects were in different pairs, those pairs were eliminated, and the SWS analysis proceeded on the eight pairs of subjects who all had SWS. In the set of eight *t* tests comparing metabolic values from SWS observations between matched insomniacs and normals, seven *t* values were in the predicted direction (binomial probability = 0.035), and five of eight were statistically significant. The average *t* value was 5.57 with an average of 51 degrees of freedom ($p < 0.0001$). The overall mean SWS metabolic rates for the insomniac and normal groups, respectively, were 266 and 250 ml/minute.

Finally, the equation of the best-fit third degree polynomial was determined for the complete $\dot{V}O_2$ data set for each subject. The equations were averaged for the normal subjects and for the insomniacs. The average equation for each group has been plotted in Fig. 1. It can be seen from the figure that both groups had a characteristic circadian metabolic curve. The primary difference between the groups appears to be that the entire curve for the insomniacs is elevated about 30 ml/minute.

DISCUSSION

The data presented indicate that the group of insomniacs participating in this study was similar to groups of insomniacs reported in other studies. These individuals reported difficulty sleeping and objectively did sleep poorly. Despite their poor sleep, they still had significantly longer MSLT values. The insomniacs in the current study reported degraded mood compared to the normals and had slightly more abnormal MMPI values. This is also consistent with the literature. None of these differences between the insomniacs or normals could be based on age, sex or weight because subjects were carefully matched on those variables. In addition, the differences could not be based upon immediate activities or diet because subjects remained in the laboratory over the 36-hour data collection period and were fed the same meals. Significant differences in psychomotor performance have only been rarely reported in insomniacs as compared to normals. In this study, no differences were seen in vigilance or proofreading performance, but the insomniacs did have a slightly smaller short-term recall on the word memory test and performed more poorly on the one-letter search of the memory-based MAST test. Decreased word memory, however, could be related to either increased or decreased level of arousal.

The use of the Deltatrac metabolic monitor has greatly simplified the collection of metabolic data during sleep. In addition to ease of use features, the re-

quirement to use a nonsealed mask allowed the normal sleep process to be measured in normal and insomniac sleepers as evidenced by the comparison of the baseline night and the metabolic night in the two groups. The only significant difference in these comparisons was that sleep latency was actually shorter in both groups on the metabolic night. Overall, sleep efficiency did not differ from baseline to the metabolic night (decreasing from 76 to 74% in insomniacs and 95 to 93% in normals). This can be compared to a sleep efficiency from 88% in an earlier study using a sealed-mask system in normal sleepers (30).

The major purpose of the current study was to examine 24-hour metabolic rate in insomniacs. The $\dot{V}O_2$ data were very consistent in showing that these carefully matched insomniacs had significantly increased oxygen use throughout the day and night as compared to their matched normal. The nocturnal increase in $\dot{V}O_2$ remained even when awakenings and arousals were eliminated from the metabolic data (30) and when only data from SWS were examined. These facts, that insomniacs have a 24-hour increase in metabolic rate and that the sleep-related increase in metabolic rate is not related to arousals and awakenings during sleep, are consistent in implying that poor sleep is a result of increased arousal and not the converse.

In a previous study, we have shown that an increase in metabolic rate was associated with many of the symptoms reported by the insomniacs in this study: increased fatigue, increased MSLT values, subjectively and objectively poor sleep, movement of MMPI values toward pathology, etc. (25). In that study, it was concluded that insomnia was perhaps more a disorder of hyperarousal than a disorder of sleep. The current data further support that unusual conclusion. Hyperarousal, as operationally defined in this study as the 24-hour increase in metabolic rate, was not dependent upon sleep disturbance and probably limited ability to fall asleep throughout the 24-hour observation period.

If insomnia is a disorder of inappropriate arousal, it implies that treatment strategies should be directed toward normalizing the level of arousal. If arousal level is decreased, then sleep and MSLT values should also move toward normal. Certainly, many traditional insomnia therapies have reduced arousal level. Benzodiazepines have a direct effect in decreasing muscle tension, body temperature and metabolic rate (31). A goal of progressive muscle relaxation is a decrease in muscle tension, and this is directly tied to metabolic rate. Sleep restriction therapy decreases arousal level by causing sleep deprivation.

Of more importance, does recognition of physiologic hyperarousal suggest new or different treatment strategies? Would 24-hour treatment with low doses of a relatively nonsedating benzodiazepine improve mood and perceived daytime function in insomniacs? Would physical conditioning, which has recently been shown to be beneficial in the elderly (32), decrease nonexercise metabolic rate and improve sleep and daytime function?

Hyperarousal, as indexed here by $\dot{V}O_2$, could be magnified by stress but may be viewed more simply as a higher set point that could be genetic or induced. Persons with an elevated arousal set point would always be prone to insomnia and, based upon our results with caffeine (25), might also be predisposed to degraded mood or psychopathology as a function of simple level of arousal. This line of reasoning suggests that certain patients might have a life-long increased level of arousal and might require life-long treatment to normalize arousal level. As idiopathic hypersomnia may require life-long treatment with stimulants, this idiopathic "hyposomnia" might benefit from life-long treatment with slightly sedating medication or other intervention as well.

Acknowledgement: This work was supported by a Merit Review Grant from the Department of Veterans Affairs and the Sleep–Wake Disorders Research Institute.

REFERENCES

1. Kales JD, Kales A, Bixler EO, et al. Biopsychobehavioral correlates of insomnia. V: Clinical characteristics and behavioral correlates. *Am J Psychiatry* 1984;141:1371–6.
2. Tan TL, Kales JD, Kales A, Soldatos CR, Bixler EO. Biopsychobehavioral correlates of insomnia. IV: Diagnosis based on DSM-III. *Am J Psychiatry* 1984;141:357–62.
3. Kales A, Caldwell AB, Soldatos CR, Bixler EO, Kales JD. Biopsychobehavioral correlates of insomnia. II. Pattern specificity and consistency with the Minnesota multiphasic personality inventory. *Psychosom Med* 1983;45:341-55.
4. Hauri PJ. A cluster analysis of insomnia. *Sleep* 1983;6:326–38.
5. Healey ES, Kales A, Monroe LJ, Bixler EO, Chamberlin K, Soldatos CR. Onset of insomnia: role of life-stress events. *Psychosom Med* 1981;43:439–51.
6. Borkovec TD. Insomnia. *J Consult Clin Psychol* 1982;50:880–95.
7. Soldatos CR, Kales A. Sleep disorders: research in psychopathology and its practical implications. *Acta Psychiatr Scand* 1982; 65:381–7.
8. Monroe LJ. Psychological and physiological differences between good and poor sleepers. *J Abnorm Psychol* 1967;72:255–64.
9. Haynes SN, Follingstad DR, McGowen WT. Insomnia: sleep patterns and anxiety level. *J Psychosom Res* 1974;18:69–74.
10. Haynes SN, Adams A, Franzen M. The effects of presleep stress on sleep-onset insomnia. *J Abnorm Psychol* 1981;90:601–6.
11. Freedman RR, Sattler HL. Physiological and psychological factors in sleep-onset insomnia. *J Abnorm Psychol* 1982;91:380–9.
12. Freedman RR. EEG power spectra in sleep-onset insomnia. *Electroencephalogr Clin Neurophysiol* 1986;63:408–13.
13. Stepanski EJ, Glinn M, Fortier J, Sicklesteel J, Zorick FJ, Roth T. Physiological reactivity in chronic insomnia. *Sleep Res* 1989; 18:306.
14. Johns MW, Gay TJA, Masterton JP, Bruce DW. Relationship between sleep habits, adrenocortical activity and personality. *Psychosom Med* 1971;33:499–508.
15. Mendelson WB, Garnett D, Gillin CG, Weingartner H. The

experience of insomnia and daytime and nighttime functioning. *Psychiatr Res* 1984;12:235–50.
16. Adam K, Tomeny M, Oswald I. Physiological and psychological differences between good and poor sleepers. *J Psychiatr Res* 1986;20:301–16.
17. Frankel B, Buchbinder R, Coursey R, Snyder F. Sleep patterns and psychological test characteristics of chronic primary insomniacs. *Sleep Res* 1973;2:149.
18. Mendelson WB, James SP, Garnett D, Sack DA, Rosenthal NE. A psychophysiological study of insomnia. *Psychiatry Res* 1986; 19:267–84.
19. Seidel WF, Ball S, Cohen S, Patterson N, Yost D, Dement WC. Daytime alertness in relation to mood, performance, and nocturnal sleep in chronic insomniacs and noncomplaining sleepers. *Sleep* 1984;7:230–8.
20. Bonnet MH. Effect of 64 hours of sleep deprivation upon sleep in geriatric normals and insomniacs. *Neurobiol Aging* 1986;7: 89–96.
21. Stepanski E, Zorick F, Roehrs T, Young D, Roth T. Daytime alertness in patients with chronic insomnia compared with asymptomatic control subjects. *Sleep* 1988;11:54–60.
22. Haynes SN, Fitzgerald SG, Shute GE, Hall M. The utility and validity of daytime naps in the assessment of sleep-onset insomnia. *J Behav Med* 1985;8:237–47.
23. Schneider-Helmert D. Twenty-four-hour sleep–wake function and personality patterns in chronic insomniacs and healthy controls. *Sleep* 1987;10:452–62.
24. Mendelson WB, Garnett D, Linnoila M. Do insomniacs have impaired daytime functioning? *Biol Psychiatry* 1984;19:1261–4.
25. Bonnet MH, Arand DL. Caffeine use as a model of acute and chronic insomnia. *Sleep* 1992;15:526–36.
26. Folkard S, Knauth P, Monk TM. The effect of memory load on the circadian variation in performance efficiency under a rapidly rotating shift system. *Ergonomics* 1976;19:479–88.
27. Wechsler D. *Manual for the Wechsler adult intelligence scale—revised.* New York: The Psychological Corporation, 1981.
28. Williams HL, Gieseking CF, Lubin A. Some effects of sleep loss on memory. *Percept Mot Skill* 1966;23:1287–93.
29. Scerbo MW, Warm JS, Fisk AD. Event asynchrony and signal regularity in sustained attention. *Curr Psychol Res Rev* 1987;5: 335–43.
30. Bonnet MH, Berry RB, Arand DL. Metabolism during normal sleep, fragmented sleep, and recovery sleep. *J Appl Physiol* 1991; 71:1112–8.
31. Roald OK, Steen PA, Stangland K, Michenfelder JD. The effects of triazolam on cerebral blood flow and metabolism in the dog. *Acta Anaesthesiol Scand* 1986;30:223–6.
32. Vitiello MV, Prinz PN, Schwartz RS. Slow wave sleep but not overall sleep quality of healthy older men and women is improved by increased aerobic fitness. *Sleep Res* 1994;23:149.