Reprinted from Psychosomatic Medicine Vol. XXXIII, No. 6, November-December 1971 Medical Department, Harper & Row, Publishers, Inc. © 1971 by American Psychosomatic Society, Inc. Printed in U.S.A.

# Relationship Between Sleep Habits, Adrenocortical Activity and Personality

M. W. Johns, MB, BS, BSc, T. J. A. Gay, MB, BS, BMedSc, J. P. Masterton, MB, ChB, DipObst, FRCS, FRACS and D. W. Bruce, MA, MAPsS

Psychologic stress is known to be associated with both sleep disturbances and increased adrenocortical activity. In this experiment, 2 groups of male medical students were selected as poor or good sleepers on the basis of their responses to a sleep questionnaire. The poor sleepers had significantly greater levels of adrenocortical activity than did the good sleepers throughout the day and night. This difference was related to psychologic characteristics. The general level of activation of the central nervous system, largely reflecting one's personality and life situation, may determine one's usual sleep habits as well as the level of adrenocortical activity.

Psychologic stress is known to be a potent cause of adrenocortical activation. Increased adrenocortical activity under these circumstances is related not simply to a particular type of emotional affect, but rather to the overall level of distress and the effectiveness of psychologic defenses invoked to deal with stressful situations (1,2). In a study involving chronic psychologic stress in the parents of children with leukemia, urinary 17-hydroxycorticosteroid (17-OHCS) excretion was closely related to the

effectiveness of their defenses from day to day. This was true even in those subjects who used increased overt emotionality as a relatively efficient, if maladaptive, means of denying awareness of the seriousness of the problem (3, 4). Individual subjects tend to maintain their daily corticosteroid excretion rate within fairly narrow limits. These can be predicted by evaluating their personality characteristics which relate to mechanisms used in tension-relieving activities (5–8).

In a study of women with breast lumps, who were awaiting excisional biopsy in hospital, Katz et al (9) used three criteria derived from interviews to predict the patients' urinary excretion of cortisol metabolites with statistically significant results. The degree of affective distress, the first of these criteria, was inferred from the presence of unpleasant affects such as anxiety or despair in the patients. The second criterion was disruption of function, derived from descriptions of anorexia, insomnia, change of bowel habits, poor frustra-

From the Sleep Laboratory, Department of Surgery, Monash University, Melbourne, Australia.

Supported by NH and MRC Medical Postgraduate Research Scholarship and NH and MRC Medical Research Scholarship.

The authors wish to thank Professor B. Hudson, Dr. J. Owen, Dr. P. M. Dennis, Dr. B. A. Scoggins and Dr. G. Sarfaty for advice in carrying out this experiment.

Received for publication March 4, 1970; final revision received March 8, 1971.

Address for reprint requests: M. W. Johns, MB, Sleep Laboratory, Department of Surgery, Monash University, Alfred Hospital, Prahran, Victoria, Australia 3181.

tion tolerance or lack of concentration. The third criterion was *impairment of defensive reserve*, indicating a tendency to be unduly sensitive to additional stresses occurring spontaneously or introduced by the interviewer.

The major difficulties in predicting the level of adrenocortical activity from such criteria were failure to evaluate adequately a subjects' response to a stressful situation in the context of his basic personality style, and describing the type of defense mechanisms invoked rather than their effectiveness in maintaining psychologic homeostasis. Therefore, it seems important to find a more objective measure of the adequacy of defense mechanisms than that derived from subjective impressions formed during an interview.

Katz et al (9) reported that the functions of sleep, appetite and the ability to concentrate were almost always found to be conspicuously disrupted when levels of adrenocortical activity were elevated in women with carcinoma of the breast. It is difficult to compare such functions as appetite, sexual drive or power of concentration in different people. However, sleep habits and degrees of sleep disturbance can be compared by means of detailed subjective reports or electronic monitoring methods in a sleep laboratory.

Psychologic stress is commonly known to be associated with sleep disturbance in animals and in man (10–14). Different psychiatric diagnoses such as anxiety neurosis, psychotic depression or acute schizophrenia have been thought to be associated with specific aspects of sleep disorder, such as difficulty in getting to sleep initially, frequent night awakenings or early-morning awakening. But there is little objective evidence to substantiate this view; rather, it seems that each of these aspects of sleep disturbance can be found in many psychi-

atric diagnostic groups, with different degrees of severity in individual subjects (10–13, 15). In acute schizophrenia and manic-depressive psychosis, the degree of sleep disturbance, as measured by the total duration of dreaming (REM) and non-dreaming (NREM) sleep each night, is highly correlated with clinical rating of psychologic turmoil, regardless of the type (16, 17). In chronic alcoholics, the degree of sleep disturbance, as measured by electronic methods in the sleep laboratory, is closely related to clinical and psychologic rating of the patient's agitation (18).

Because of the widespread association between sleep disturbance and psychologic distress, it is possible that an appropriate measure of sleep disturbance might reflect accurately the overall efficiency of psychologic defenses in coping with conflicts and life stresses, and hence the general level of adrenocortical activity from day to day. Low efficiency in these psychologic coping mechanisms, relative to the subject's ongoing requirements, may lead to continuously elevated levels of central nervous system activity, especially in the hypothalamus. This in turn would be reflected both in increased rates of adrenocortical hormone secretion and in a tendency to experience more disturbed sleep.

The present experiment was designed as an initial test of this hypothesis. It attempted to show that even mild long-term sleep disturbances, reported subjectively by medical students, do reflect psychologic differences and are also related to the general levels of adrenocortical activity in different subjects.

#### **Assessment of Sleep Disturbance**

As yet, there is no general agreement on what constitutes abnormally disturbed sleep in a given subject. However, most would agree that prolonged delay in falling

asleep, frequent night awakenings and early-morning waking after a short total duration of sleep are important factors. Johns et al (13) have used such parameters, subjectively reported by medical and surgical patients, to describe the usual sleep habits at different ages and with different physical symptoms. Increasing age, neurotic illness and ischemic heart disease were the most important factors associated with disturbed sleep. Monroe (19) showed that among healthy adults, groups of good or poor sleepers selected on the basis of their responses to a sleep questionnaire had significant psychologic differences. The latter had higher scores than the former on most scales of the Minnesota Multiphasic Personality Inventory (MMPI) and the Cornell Medical Index Health Questionnaire (CMI). The 2 groups also had significantly different sleep patterns when measured by EEG methods.

The accuracy of subjective reports of such parameters as the delay-in-sleeping onset, the number and duration of night awakenings, and the time of morning awakening has been studied in the laboratory, using EEG methods (20). Normal subjects tend to overestimate slightly the degree of sleep disturbance and to underestimate the duration of sleep. This is especially true of patients with psychologic symptoms of functional or organic origin. Thus, subjective reports of sleep disturbance may be even more sensitive indicators of psychologic function than are objectively demonstrable aspects of sleep in the laboratory. In this investigation, subjectively reported sleep habits were used as a measure of psychologic function, not as estimates of objective sleep parameters.

Two groups of male medical students were selected to represent good and poor sleepers on the basis of their responses to a sleep questionnaire. Their levels of adreno-

cortical activity were assessed by means of complete urine collections for 72 hours and by assay of urinary free 11-hydroxycorticosteroids. Personality differences were described by means of the Minnesota Multiphasic Personality Inventory (MMPI), the Cornell Medical Index Health Questionnaire (CMI), and the Eysenck Personality Inventory (EPI). Significant differences were found between the groups of good and poor sleepers, both from the point of view of personality characteristics and the levels of adrenocortical activity during the day and night.

#### **METHODS**

#### **Selection of Subjects**

A total of 122 fourth-year medical students at Monash University completed a sleep questionnaire giving information about the usual time that they went to bed and fell asleep on weekdays and weekends, the number and duration of night awakenings, the usual time of morning awakenings, and other related data. From the range of sleep habits reported by this large group of students, 2 groups of 7 male students each were selected to represent good and poor sleepers on the basis of duration of sleep, delay-in-sleeping onset and duration of night awakenings. Their usual sleep habits are summarized in Table 1, with sleep parameters measured over a whole week rather than per 24 hours, thereby partially overcoming differences in sleep habits on weeknights and weekends (13). The poor sleepers obtained less sleep at night and overall than did the good sleepers. The poor sleepers reported taking almost three times longer to fall asleep at night; they woke up more often during the night and tended to lie in bed slightly longer (not statistically significant) after waking up in the mornings. All the latter differences are reflected in the overall difference in time spent lying in bed awake at night. The poor sleepers spent less time overall in bed at night, but slept more during the day than did the good sleepers. All of the good sleepers described their usual sleep as very good, whereas the poor sleepers complained of mild to moderate sleep disturbance. However, none of these students could be considered to have a serious sleep disturbance.

Table 1. Differences in Sleep Habits Between Good and Poor Sleepers

Parameter	Good sleepers		Poor sleepers		Group differences	
	Mean	SD	Mean	SD	Mean differ <b>e</b> nce	t
Night sleep (hr/wk)	58.25	1.85	51.70	3.97	-3.55	3.96*
Day sleep (hr/wk)	0.06	0.00	0.83	0.65	+0.77	3.13†
Total sleep (hr/wk)	58.30	2.00	52.53	4.30	-5.77	3.22†
Delay-in-sleeping onset (hr/wk) Frequency of night awakenings (times/wk)	1.17	0.00	2.50	1.19	+1.33	2.96‡
Total duration of night awakenings (hr/wk)	0.00	0.00	7.90	6.10	+7.90	2.98‡
Lying in bed after morning awakening (hr/wk)	0.00	0.00	1.43	0.93	+1.43	4.07*
Total time spent awake in bed at	1.88	0.53	2.57	1.07	+0.69	1.53
night (hr/wk)	3.00	0.60	6.50	2.30	+3.50	3.90*
Total time in bed at night (hr/wk)	61.30	1.63	58.20	2.93	-3.10	2.45§

Statistical significance (2-tailed t test).

The physical characteristics of the experimental subjects are summarized in Table 2. Their ages ranged from 21 to 24 years and were similar in each group, as were their body surface areas calculated by the Dubois method. None of the subjects was obese. These students had no imminent examinations at the time. They all had a similar daily routine and none reported recent illness. They were asked to participate in the experiment and, with the offer of a small financial reward, all willingly complied.

# **Urine Collection**

Four weeks after completing the sleep questionnaire, the subjects collected all their urine for a period of 72 hours, involving a total of 42 subjectdays. The collection period started at 1.30 PM on Tuesday and ended at 1.30 PM on Friday. Each day was divided into three collection periods, during which urine was pooled for each subject. These periods represented morning (AM), afternoon and evening (PM), and overnight (ON) excretion periods. The ON pooled specimen included any urine voided during the night after initially going to sleep and the urine voided on finally awakening. The latter time varied from 6.30 to 8.30 AM in different subjects. The AM specimen included all urine voided after the first morning specimen up till 1.30 PM. The PM specimen comprised all urine voided between 1.30 PM and the time the subjects went to bed at night. Each collection period therefore was not of the same length but served the purpose, when corrected for these variations, of providing an index of the mean rate of urinary excretion of free 11-OHCS per hour during morning, afternoon and night in each subject for 3 days. Samples, deepfrozen within 4 hours of collection, were assayed within 1 month.

Records of sleep and daily activities were kept by each student during the urine collection period.

Table 2. Physical Characteristics of Experimental Groups (Mean  $\pm$  SD)

Good sleepers	Poor sleepers			
7	7			
$21.9 \pm 1.2$	$22.4 \pm 1.3$			
$\textbf{1.85} \pm \textbf{0.10}$	$1.90\pm0.11$			
	7 21.9 ± 1.2			

<sup>\*</sup> P < 0.005.

<sup>†</sup> P < 0.01.

 $<sup>\</sup>ddagger P < 0.02.$ 

<sup>§</sup> P < 0.05.

The pattern of differences between the good and poor sleepers during this time was similar to that reported in the sleep questionnaire I month earlier. The subjects recorded their own rectal temperatures just before falling asleep at night and again after morning waking. They were permitted to engage in their usual activities, but no alcohol or drugs were allowed. The three personality and health inventories (MMPI, CMI and EPI) were completed during the week when urine was collected.

# Assay for Urinary Free 11-Hydroxycorticosteroids

The fluorometric method of Mattingly and Dennis (21) was used to measure free 11-OHCS (free cortisol, corticosterone and 20 hydroxycortisol) in each of the three urine samples for 42 subject-days. The excretion of urinary free cortisol has been thought to provide a better index of plasma cortisol concentrations than the chemical determination of cortisol metabolites included in 17-OHCS and 17-ketogenic steroids (22). Despite the presence of small amounts of nonspecific fluorescence, the fluorometric assay of free 11-OHCS (of which 40-60% is free cortisol) has been shown to give results which are highly correlated with cortisol production measured by isotopic methods (21). The normal value for daily excretion of urinary free 11-OHCS in male subjects is said to be  $230 \pm 150$  $\mu g/24 \text{ hr } (23)$ .

#### **RESULTS**

### 11-OHCS Assay

Results of the urine assays are shown in Fig 1, with mean excretion rates ( $\mu$ g 11-OHCS/hr) for each period during the 3-day collection in good and poor sleepers. In all subjects, a diurnal variation in excretion rates was observed, but poor sleepers excreted more free 11-OHCS than good sleepers throughout each day and night. The mean total 24-hour excretion of free 11-OHCS for good sleepers was 328.3  $\pm$  57.1  $\mu$ g, whereas that for poor sleepers was 527.9  $\pm$  118.6  $\mu$ g (P<0.01 in a 2-tailed t-test). Analysis of variance of the values for 24-hour excretion of free 11-OHCS during all 42 subject-days indicated that there was

significantly greater variance between the groups of sleepers than within these groups  $(F=12.2;\ P<0.01,\ \text{with 1}\ \text{and 15}\ \text{degrees}\ \text{of}$  freedom). Am excretion rates for poor sleepers were very high and remained significantly higher than on levels during the PM period (P=0.01). By contrast, PM excretion rates in good sleepers were almost as low as on levels, and were significantly lower than AM levels (P<0.01).

There was a significant correlation between body surface area and mean 24-hour steroid excretion in good sleepers (R=0.79; P=0.05, Spearman's rank correlation coefficient), but this was not so for poor sleepers. Mean values for 24-hour excretion of free 11-OHCS per square meter of body surface area were 177.1  $\pm$  26.7 and 280.7  $\pm$  73.0  $\mu$ g/24 hr/sqm for good and poor sleepers, respectively (P<0.02). There were no significant differences between rectal temperatures of the 2 groups, and no subject had a fever during the experiment.

## **Personality Inventories**

Mean scores for the 2 groups on each scale of the personality and health inventories (MMPI, CMI and EPI) are shown in Table 3. On each of the 10 basic clinical scales of the MMPI (Hs to Si), the poor sleepers had higher mean scores than the good sleepers (P<0.01, Sign Test). Significant differences in 2-tailed t tests were found on individual scales relating to masculinity-femininity (Mf), hypochondriasis (Hs) and conversion reaction (Cr) of the MMPI, as well as A-L and total scores of the CMI. Two additional scales, manifest anxiety (Mas) of the MMPI and M-R of the CMI showed significant differences between the groups in 1-tailed t tests. It is noteworthy that extraversion-introversion (E) and neuroticism (N) scales of the EPI were among the least efficient in distinguishing good from poor sleepers.

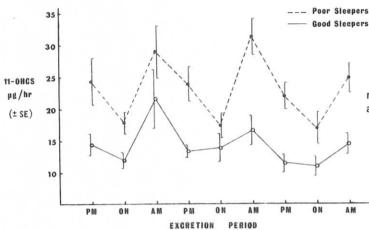


Fig 1. Mean urinary excretion rates for free 11-OHCS in good and poor sleepers.

To obtain such differences between relatively small groups, subjective reports of sleep disturbance must have provided a highly discriminating test of psychologic characteristics. The greater anxiety, concern with body symptoms and sexual identity in poor sleepers represent a neurotic disturbance with more affective distress than that reported by good sleepers. The latter had low scores on all clinical scales except conversion reaction (Cr) which indicates the success of their defense mechanisms in preventing affective distress.

#### DISCUSSION

The results have shown that in subjects selected as good or poor sleepers from the extremes of a population of healthy medical students, significantly different levels of adrenocortical activity were observed throughout the day and night. All subjects had a diurnal variation in free 11-OHCS excretion, but poor sleepers had a different pattern of excretion, high levels being maintained during the afternoon and evening relative to their overnight levels. Differences between the 2 groups could not be attributed to identifiable stresses during the experiment or to variation in body size.

There was a significant correlation between body surface area and steroid excretion among good sleepers. Rose (8) described a similar relationship between body surface area and 17-OHCS excretion in army personnel.

Plasma and urinary corticosteroid concentrations are known to decrease during NREM sleep compared with during the waking state, but they increase again in relation to REM (dreaming) periods which tend to predominate during the latter part of a night's sleep (24-26). However, poor sleepers, in contrast to good sleepers, spend less time absolutely, and a smaller percentage of sleep time dreaming (19). This is not likely to explain the differences in corticosteroid output. Although plasma concentrations of cortisol usually increase to maxima during REM periods in the latter part of a night's sleep and other peaks during the early waking hours (26), there is probably a time delay in the peak rates of urinary excretion of corticosteroids. This may explain why on excretion rates measured here were lower than AM rates in both groups of subjects. Variations in the amount of physical activity during the day, which may cause changes

Table 3. Mean Raw Scores and Differences Between Good and Poor Sleepers on Each Scale of the MMPI, CMI, and EPI

	Good sleepers		Poor slo	eepers	Group differences	
Scale	Mean	SD	Mean	SD	Mean difference	t
MMPI						
L	4.86	1.35	4.42	1.90	-0.44	0.49
F	3.86	2.91	5.00	1.63	+1.14	0.91
K	17.86	3.85	16.43	3.69	-1.43	0.71
Hs	2.29	1.38	5.86	2.41	+3.57	3.40*
D	18.57	5.19	21.29	3.99	+2.72	1.10
Ну	20.57	2.94	21.86	4.34	+0.59	0.65
Pd	13.43	3.99	15.71	4.39	+2.28	1.02
Mf	24.86	3.80	31.71	2.87	+6.85	3.81†
Pa	8.71	2.56	10.29	2.21	+1.58	1.23
Pt	7.00	4.12	10.43	5.80	+3.43	1.27
Sc	7.57	3.87	9.29	4.75	+1.72	0.74
Ma	15.71	4.92	17.71	3.30	+2.00	0.89
Si	21.14	9.69	22.29	6.26	+1.15	0.26
Mas	7.43	4.54	14.00	7.92	+6.57	1.91‡
Cr	59.29	3.30	53.14	6.20	-6.15	2.31§
Dy	13.86	5.34	17.71	6.40	+3.85	1.23
CMI						
A-L	3.86	1.57	11.43	6.02	+7.57	3.22*
M-R	1.14	1.21	4.43	4.43	+3.29	1.89‡
Total	5.00	2.45	16.86	9.94	+10.86	2.819
EPI						
E	15.86	4.52	16.43	2.57	+0.57	0.29
N	6.00	2.77	7.57	5.32	+1.57	0.69
L	1.29	1.38	0.29	0.76	-1.00	1.68

Statistical significance (2-tailed t test).

in 11-OHCS excretion (27) could not explain the differences between good and poor sleepers during sleep. Furthermore, lying in bed does not affect corticosteroid excretion (28).

The rates of excretion of free 11-OHCS for both good and poor sleepers were higher than reported elsewhere for male patients (23). This difference may have been due to the age or body size of the subjects.

Also, it may be that medical students going about their daily activities have higher levels of adrenocortical activity than do patients in hospital. The levels of adrenocortical activity found in poor sleepers were as high as some reported in patients with Cushing's disease as well as in major affective disturbances such as acute psychotic depression. None of these are relevant to the student subjects, nor indeed are

<sup>\*</sup> *P* < 0.01.

 $<sup>\</sup>dagger P < 0.005.$ 

 $<sup>\</sup>ddagger P < 0.10.$ 

<sup>§</sup> P < 0.05.

<sup>¶</sup> P < 0.02.

diagnosed on the basis of increased adrenocortical activity alone. Perhaps poor sleepers were more distressed than good sleepers at being asked to take part in an experiment involving personality assessment. All students were given a similar explanation for participating in the experiment, but poor sleepers, who had other evidence of greater psychologic vulnerability in the personality inventories, may have reacted differently from good sleepers over the 3-day period of urine collection.

There is evidence that it is not the duration of sleep on any particular night which determines the daily excretion of corticosteroids. During the present experiment, the 24-hour excretion of 11-OHCS was not significantly related to the actual duration of sleep reported during that 24-hour period, which fluctuated between 6 and 9 hours for good sleepers and 5 and 9 hours for poor sleepers. Young adults can cope with quite marked voluntary short-term fluctuations in the amount of sleep which they get as a result of nonstressful activities without increased adrenocortical activation (29).

Differences in steroid excretion between good and poor sleepers seem to relate more to differences in psychologic characteristics. Monroe (19) found evidence of less psychologic adaptiveness in poor sleepers who were selected on the basis of sleep questionnaire responses, and who were later found to have objective evidence of sleep disturbance in the laboratory. On the basis of pulse rate, body temperature and number of peripheral vasoconstrictions, he suggested that in poor sleepers, greater activation of the central nervous system was observed during both wakefulness and sleep, than in good sleepers. However, the higher rectal temperature in poor sleepers reported by Monroe was not confirmed in the present experiment.

We have found that subjective descriptions of minor difficulty in falling asleep, night awakening and an overall feeling of disturbed sleep can delineate groups of otherwise healthy students who have significantly different psychologic characteristics. Even in groups comprising as few as 7 subjects, there were highly significant differences on several personality scales. Poor sleepers were more anxious, had more physical symptoms of various kinds, and were less well controlled than good sleepers. Good sleepers had relatively low scores on all personality scales apart from CR, the conversion reaction scale of the MMPI. This suggests that good sleepers had defense mechanisms which dealt successfully with chronic stresses to which they were subjected in their day-to-day lives, so that they were protected against much affective distress. Differences between the groups may not simply reflect greater or lesser degrees of psychopathology in good and poor sleepers. In an extreme case, for example, a person whose conversion reaction mechanisms produced paralysis of both legs as a result of major psychopathology may, nevertheless, show belle indifference. His subconscious conflicts may have been handled in a maladaptive way and thus produced functional paralysis, but in a way which would protect him against affective distress and presumably against sleep disturbance.

The fact that subjective descriptions of sleep habits have also been able to distinguish groups with different levels of adrenocortical activity suggests that the two parameters—degree of sleep disturbance and general level of adrenocortical activity—are closely related. They are probably not causally related, but both may be related to a third parameter—the degree of central nervous system activation which is part of our basic lifestyle reflecting our personality

and life situation. There was an interval of 4 weeks between the time when the sleep questionnaire was completed and when urine was collected, indicating that the relationship between sleep habits and the level of adrenocortical activity is long-term, although it could be expected to hold on a short-term basis too.

Differences between good and poor sleepers in the pattern of adrenocortical activity during the day and night are in keeping with the idea of chronically elevated hypothalamic activity in poor sleepers superimposed upon a pattern of diurnal variation. Sleep disturbance involves the tendency to obtain less sleep than average for one's age, difficulty in falling asleep, and inability to sleep soundly when given the opportunity. It appears then that sleep disturbances, at least in young adults, are usually associated with increased activation of the central nervous system, particularly the hypothalamus, as a result of psychologic conflict and tension. Increased hypothalamic activity is reflected in more secretion of ACTH from the anterior pituitary, and hence of corticosteroids from the adrenal cortex (30). By asking a few simple questions about a person's sleep habits, it may be possible to know as much about how his psychologic defenses are withstanding the stresses to which he is subject as would be possible from extensive psychologic data. Similarly, knowledge of sleep habits may explain marked differences within the normal range of adrenocortical activity in different people, as well as variations due to other factors such as body size and sex.

#### SUMMARY

Psychologic distress is known to be associated with both increased secretion of adrenocortical hormones and sleep disturbances. It is suggested that the degree of sleep disturbance in a given subject may be

directly proportional to the degree of psychologic distress from day to day. This in turn may be proportional to the level of adrenocortical activity. To test this hypothesis, a sleep questionnaire was given to all fourth-year medical students at Monash University, Melbourne. Two groups of male students were selected as good and poor sleepers on the basis of reports regarding their usual duration of sleep, delay-in-sleeping onset and frequency of night awakenings. Urine was collected from each student for 72 hours. Assays for free 11-hydroxycorticosteroids (11-OHCS)representing morning, afternoon and evening, and overnight excretion periodswere made on nine pooled samples from each subject. Poor sleepers excreted more 11-OHCS than did good sleepers during each excretion period. In poor sleepers, evidence of greater psychologic distress was seen in their higher scores on most scales of the Minnesota Multiple Personality Inventory, Cornell Medical Index and Eysenck Personality Inventory. Knowledge of sleep habits may be very useful in determining the efficiency of a person's psychologic defenses in dealing with the stresses to which he is subject, as well as in explaining variations in the normal level of adrenocortical activity in different subjects.

#### REFERENCES

- Rubin RT, Mandell AJ: Adrenal cortical activity in pathological emotional states: a review. Amer J Psychiat 123:387-400, 1966
- Oken D: The psychophysiology and psychoendocrinology of stress and emotion, Psychological Stress. Edited by MH Appley, R Trumbull. New York, Appleton, 1967, pp 43-76
- 3. Wolff CT, Friedman SB, Hofer MA, et al: Relationship between psychological defenses and mean urinary 17-hydroxycorticosteroid excretion rates. 1. A predictive study

- of parents of fatally ill children. Psychosom Med 26:576–591, 1964
- 4. Wolff CT, Hofer MA, Mason JW: Relationship between psychological defenses and mean urinary 17-hydroxycorticosteroid excretion rates. 11. Methodologic and theoretical considerations. Psychosom Med 26:592–609, 1964
- 5. Fox HM, Murawski MJ, Bartholomay AF, et al: Adrenal steroid excretion patterns in eighteen healthy subjects. Psychosom Med 23:33–40, 1961
- ö. Friedman SB, Mason JW, Hamburg DA: Urinary 17-hydroxycorticosteroid levels in parents of children with neoplastic disease. Psychosom Med 25:364-376, 1963
- Sachar EJ, Mason JW, Fishman JR, et al: Corticosteroid excretion in normal young adults living under "basal" conditions. Psychosom Med 27:435-445, 1965
- 8. Rose RM, Poe RO, Mason JW: Psychological state and body size as determinants of 17-OHCS excretion. Arch Intern Med 121:406–413, 1968
- 9. Katz JL, Ackman P, Rothwax Y, et al: Psychoendocrine aspects of cancer of the breast. Psychosom Med 32:1-18, 1970
- 10. McGhie A: The subjective assessment of sleep patterns in psychiatric illness. Brit J Med Psychol 39:221-230, 1966
- 11. Mendels J, Hawkins DR: Sleep and depression. Arch Gen Psychiat 19:445–452, 1968
- 12. Feinberg I, Braun M, Koresko RL, et al: Stage 4 sleep in schizophrenia. Arch Gen Psychiat 21:262–266, 1969
- Johns MW, Egan P, Gay TJA, et al: Sleep habits and symptoms in male medical and surgical patients. Brit Med J 2:509-512, 1970
- 14. Broughton R, Gibson W: Effects of environmental and internal stress upon sleep and wakefulness in the rat. Paper read at Association for the Psychophysiological Study of Sleep, Annual Conference. Santa Fe, 1970
- 15. Hawkins DR, Mendels J: Sleep disturbance in depressive syndromes. Amer J

- Psychiat 123:682-689, 1966
- 16. Kupfer DJ, Wyatt RJ, Scott J, et al: Sleep disturbance in acute schizophrenic patients. Amer J Psychiat 126:1213–1223, 1970
- 17. Meltzer HY, Kupfer DJ, Wyatt R, et al: Sleep disturbance and serum CPK activity in acute psychosis. Arch Gen Psychiat 22: 398–405, 1970
- 18. Johnson LC, Burdick JA, Smith JS: Sleep during alcohol intake and withdrawal in the chronic alcoholic. Arch Gen Psychiat 22:406-418, 1970
- Monroe LJ: Psychological and physiological differences between good and poor sleepers. J Abnorm Psychol 72:225–264, 1967
- Johns MW: Methods for assessing human sleep. Arch Intern Med 127:484–491, 1971
- 21. Mattingly D, Dennis PM, Pearson J, et al: Rapid screening test for adrenal cortical function. Lancet 2:1046–1049, 1964
- 22. Cope CL: Adrenal steroids and disease. London, Pitman, 1965
- 23. Dennis PM: Personal communication
- 24. Mandell MP, Mandell AJ, Rubin RT, et al: Activation of the pituitary-adrenal axis during rapid eye movement sleep in man. Life Sci 5:583-587, 1966
- 25. Weitzman EDS, Schaumburg H, Fishbein W: Plasma 17-hydroxycorticosteroid levels during sleep in man. J Clin Endocr 26:121-127, 1966
- 26. Hellman L, Nakada F, Curti J, et al: Cortisol is secreted episodically by normal man. J Clin Endocr 30:411-422, 1970
- 27. Bellet S, Roman L, Barham F: Effect of physical exercise on adrenocortical excretion. Metabolism 18:484–487, 1969
- 28. Katz FH: Adrenal function during bed rest. Aerospace Med 35:849-851, 1964
- 29. Kollar EJ, Slater GR, Palmer JO, et al: Stress in subjects undergoing sleep deprivation. Psychosom Med 28:101-113, 1966
- Mason JW: The CNS regulation of ACTH secretion, Reticular Formation of the Brain. Boston, Little, Brown 1957, pp 645-670