Review

Methods for Assessing Human Sleep

Murray W. Johns, MB, BS, Melbourne

Over the last two decades there has been a great increase in interest and experimentation in the physiology and pathology of sleep. This has involved many different disciplines such as neurophysiology, general physiology, biochemistry, pharmacology, endocrinology, psychology, and psychiatry. The description of changes in the electroencephalogram during sleep by Loomis et al in 1935[1] and the later definition of sleep stages by Dement and Kleitman in 1957[2] greatly facilitated these investigations. Similarly, the discovery of a relationship between rapid eye movements (REM), a low-amplitude fast-frequency EEG, and subjective reports of dreaming has been followed by many objective investigations into the nature of the dreaming state which previously had not been possible. The nature of sleep disturbances in psychiatric illness and the effects of hypnotic drugs, which are so widely used but so little understood, are now the subject of intense research. Normal sleep habits and the epidemiology of sleep disturbances in the community are being investigated both by subjective methods in large groups and the objective techniques of sleep laboratories with smaller groups. Lack of sleep has been implicated as an etiological factor in the acute psychosis which occurs fairly commonly in patients in intensive-care wards, especially after cardiac surgery.[3] Dreaming sleep has been associated with abnormally high levels of gastric acid secretion in patients suffering from duodenal ulcers[4] and with the onset of angina pectoris and transient electrocardiographic charges in patients with ischemic heart disease.[5] Plasma levels of growth hormone have been found to be relatively high during delta-wave sleep[6] when levels of corticosteroids and catecholamines are

Received for publication Aug 12, 1970; accepted Sept 10.

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

Reprint requests to Sleep Laboratory, Department of Surgery, Monash University Medical School, Alfred Hospital, Commercial Road, Prahran, Victoria, Australia 3181 (Dr. Johns).

very low,[7,8] the latter changes probably playing a part in the restorative and anabolic functions attributed to sound sleep. The increasing relevance of sleep research to general medicine and surgery is thus clear.

Because of differences in experimental objectives, subjective bias of the experimenters, and availability of equipment, a wide variety of experimental methods have been used in sleep research. The similarities as well as the differences and limitations in the information available from these various methods have not been well understood. The great emphasis on the EEG and electro-oculogram (EOG) in recent years has sometimes involved a vain hope that these methods alone will give all of the relevant information about a night's sleep. By contrast, evaluation of the effect of hypnotic drugs in humans has sometimes been crude and inaccurate for the want of expensive and time-consuming EEG methods.

In this review the different methods currently used in sleep research are briefly described. The information which can be derived from each and the advantages and disadvantages are outlined. Examples of the use of particular methods will be cited without any attempt being made to review all of the relevant literature.

Subjective Methods

Sleep Questionnaires.—The questions asked in self-administered sleep questionnaires have varied considerably between different investigators, but most have included enquiries relating to the duration and quality of sleep and the nature of dreams. In the simplest case, information about the duration of sleep has been derived from answers to a question such as "How long do you like to sleep at night?"[9-11] A range of possible answers has sometimes been provided, with the experimental subject selecting the most appropriate. In more complicated questionnaires the subjects have been asked their usual time of retiring to bed, how long it takes them to fall asleep, how often they awake during the night, and what time they usually wake up in the morning.[12,13] Webb[14] found satisfactory reliability on test-retest administrations of such a questionnaire.

We have found that considerably more detail about sleep habits can be obtained if separate questions are asked about sleep at weekends and on weekdays in that the latter may be quite different, especially in young adults.[15] The duration of sleep over a whole week is much more constant than for single nights in any individual subject. One may calculate the usual total duration of sleep (day and night) for a week, making allowances for sleep lost as a result of night awakenings. Separate values may be calculated for the delay in falling asleep, the number and duration of night awakenings, and the delay before getting out of bed in the mornings. The total of these figures gives an estimate of the time spent awake in bed at night, which, together with the total duration of sleep, has been used to define the severity of sleep disturbances.[15] Normal subjects who differ only by small amounts in such factors derived from a sleep questionnaire have significant differences of personality and corticosteroid excretion throughout the day and night (Johns MW, Gay TJA, Masterton JP, Bruce D, unpublished data). Similarly, Monroe[16] has shown that such subjects selected as "good" or "poor" sleepers on the basis of a sleep questionnaire have objective differences in their sleep pattern as measured by EEG methods in the laboratory.

The accuracy of subjective estimates of the delay to sleep onset, number of night awakenings, and total sleep duration have been studied recently in normal subjects in the laboratory. Lewis[17] found that subjective reports tended to overestimate the delay to sleep onset and number of awakenings during the night, and to underestimate the total sleep time. Nevertheless, the subjective and objective results were significantly correlated. Baekeland and Hoy[18] reported greater accuracy than Lewis in their subjects' accounts of sleep latency and number of awakenings lasting longer than four minutes each. We have found that over 13 nights four normal subjects estimated the time of falling asleep to within two minutes (range, −14 to +29) and the total duration of sleep to within eight minutes (range, −30 to +29).

However, studies of psychiatric patients suggest that depressed patients overestimate their degree of sleep disturbance to a greater extent than do normal subjects[19] whereas manic patients underestimate their degree of sleep disturbance.[20] Likewise, there is a tendency for elderly subjects, especially those with chronic brain syndrome, to underestimate the duration of their sleep or to deny that they have slept at all during the night.[21] These problems arise in such patients because it is difficult for them to distinguish subjectively between brief periods of light sleep and wakefulness which characterize their sleep pattern.[22] They may also have more difficulty in estimating the passage of time when awake than healthy young subjects.[23]

Comparison of the efficacy of hypnotic drugs on the basis of subjective reports can be made quite sensitive to small differences, if instead of asking for an absolute rating of a night's sleep the subject is asked simply to decide whether a night's sleep with one drug is better or worse in several respects than his sleep with another drug. A double-

blind technique is used with an active drug and placebo, each patient acting as his own control.[24] There are some aspects of a night's sleep which demand subjective descriptions. Only the subject can tell us how he feels when he wakes up, whether refreshed or tired. We can measure objectively the duration of dreaming periods, but only the subject can tell us what he dreamed about.

Daily Sleep Charts.—Instead of asking a series of questions relating to a subject's sleep habits, one may ask him to record on a chart each morning for several days or weeks the particular times of going to sleep, night awakening, and final morning awakening. This method was successfully used by Lewis and Masterton[25,26] to ascertain the sleep habits of several different occupational groups, and has been used to describe the sleep and wakefulness in adults at different ages.[27-29] Daily sleep charts, like sleep questionnaires, depend on the subject's estimates of time, which under most circumstances are sufficiently accurate to give information about group differences, if not small individual differences. Sleep charts have the advantage over questionnaires of giving information on day-to-day variations in sleep over a prolonged period. However, a questionnaire is less trouble to complete. The similarity in most aspects of the sleep patterns reported by groups of normal subjects using sleep questionnaires and daily sleep charts would seem to enhance the reliability of both methods.

Objective Methods

Electroencephalogram.—The EEG has formed the mainstay of most recent attempts to measure sleep objectively. The usual arrangement is for an EEG to be derived from either bipolar frontooccipital or parietooccipital electrodes, or from a unipolar parietotemporal electrode referred to the opposite mastoid process. The EEG signal is amplified and may be recorded on a frequency-modulated tape recorder and replayed later, or may be recorded directly by a pen-writing polygraph running at chart speeds of 10 or 25 mm per second Other channels of the polygraph may be used to record additional EEG information, the electromyogram (EMG), or other variables such as the electrocardiogram or respiration rate. Because most sleep investigations involve all-night recordings, polygraph methods use thousands of feet of recorder paper.

The EEG is usually scored visually in segments of 20 or 30 seconds which can be assigned to stages of sleep. The proportion of time taken up by high amplitude, low frequency waves (delta waves) increases from stage 1 to stage 4 sleep (Fig 1). The Association for the Psychophysiological Study of Sleep has produced a manual with standardized nomenclature and methods for recording and scoring sleep EEGs which should lead to greater uniformity of sleep staging than has hitherto been possible,[30] especially for nondreaming stages.[31]

The percentage and the total time spent in particular stages and the pattern of change from one stage to another have been used to characterize a night's sleep[32] (Fig 2). The percentages of sleep in various stages have been shown to vary with age[33] and in certain psychiatric conditions such as psychotic depression,[34] acute schizophrenia,[35] and chronic brain syndrome.[21] Other characteristics of the EEG which have been described under various conditions include the delay to the first epoch of stage REM or stage 3 sleep, the number of brief arousals during the night, the mean duration and number of stage changes, and the intensity of spindle activity. Under stress, and hence during nights of subjectively poor quality sleep, the delay to the first epoch of stage REM increases and the total duration of this stage decreases. Similarly, the frequency of stage changes and the number of arousals increase and suggest a lack of ability to sustain long periods of any particular sleep process.[36] Results from the first night's recordings in a sleep laboratory are usually disregarded because of the unusually disturbed sleep, the "first-night effect" as it has been called.[36] However, even when sleep in the laboratory has improved somewhat after the first night it may still continue to be more disturbed than sleep at home, in most if not all subjects.[18]

Unfortunately there has been a tendency to equate each EEG stage with a particular depth and quality of sleep. The depth of sleep at any time depends on the particular method used to measure it, as well as the experimental subject and other factors.[37,38] In general terms, the degree of difficulty in arousing someone from sleep increases from stage 1 to stage 4 with stage REM being intermediate. But this arousal threshold apparently decreases with age within the same sleep stage and may vary from night to night in the same individual. For example, the arousal threshold in stage 3 is not invariably higher than in stage 2. The arousal threshold in stage 4 or stage REM is higher than usual if that sleep is taken at night rather than during the day,[39] and is higher than usual in all stages during recovery from sleep deprivation.[40]

There are other dangers in relying too heavily on the EEG to describe sleep. Certain drugs such as atropine sulfate and hyoscyamine sulfate produce alert behavior but an EEG characteristic of sleep.[41,42] After cardiac surgery without complications the waking EEG sometimes has greatly increased amounts of slow activity which seem to relate to hypo-

natremia.[43] Elderly patients often show diffuse slow wave activity in their waking EEG despite the fact that the amplitude of their delta waves during sleep is lower than in young adults. Also, there are variations in the EEG derived simultaneously from electrodes on different parts of the head which make sleep staging less reliable.[44] One must be cautious in interpreting sleeping EEGs in the absence of other measures of sleep.

Electro-oculogram.—The potential difference between the cornea and retina of each eye is such that electrodes placed at the orbital margins can record accurately the position of the eyeball.[45] Such monitoring of eye movements has become the method most commonly used for the detection of REM which, when associated with a stage 1 EEG, indicate dreaming periods of sleep.[2] The REMs are similar to the saccadic eye movements of wakefulness and involve conjugate movements with horizontal and vertical components. The horizontal components are easily detected as signals of up to several hundred microvolts between electrodes at the outer canthus of each eye (Fig 3). The vertical components can be detected at the same time by having an electrode above one outer canthus and the other electrode below the opposite outer canthus. Alternatively an electrode at each outer canthus can be referred to a third electrode at the nasion or mastoid process so that the position of each eye is monitored separately. The raw EOG is often recorded along with the EEG on a polygraph running at chart speeds of 10 to 25 mm/sec. However, the EOG can be interpreted quite readily at much slower chart speeds.

A completely different method of monitoring eye movements has been described by Baldridge et al.[46] A miniature ceramic strain gauge is at-

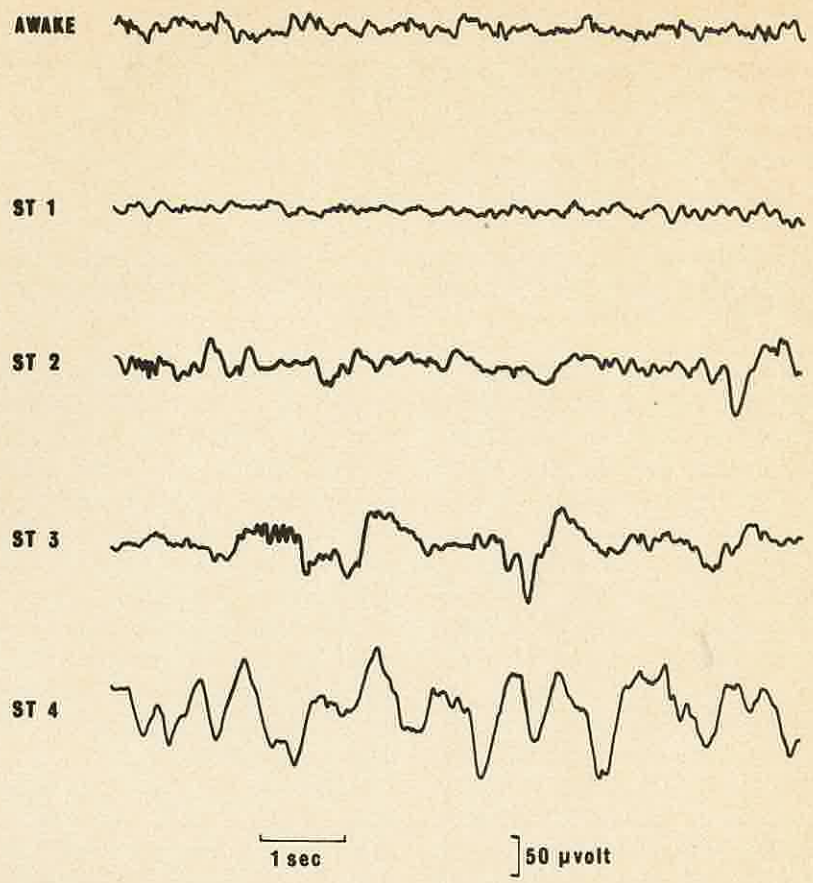
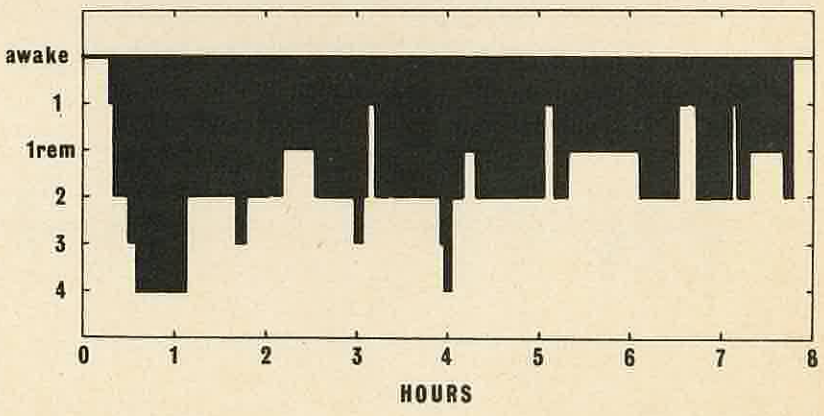


Fig 1.—The EEG of wakefulness and various stages of sleep recorded from a unipolar parietotemporal electrode. Average amplitude increases and the frequency decreases from stage 1 to stage 4. Spindles are present in stage 2 and stage 3.

Fig 2.—The pattern of stage changes during a typical night's sleep in a young adult.



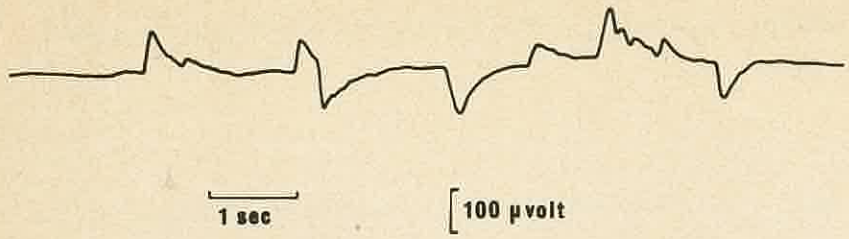


Fig 3.—Rapid eye movements as recorded by the electro-oculogram during stage REM or dreaming sleep. Very little activity or slow pendular eye movements are seen during nondreaming sleep.

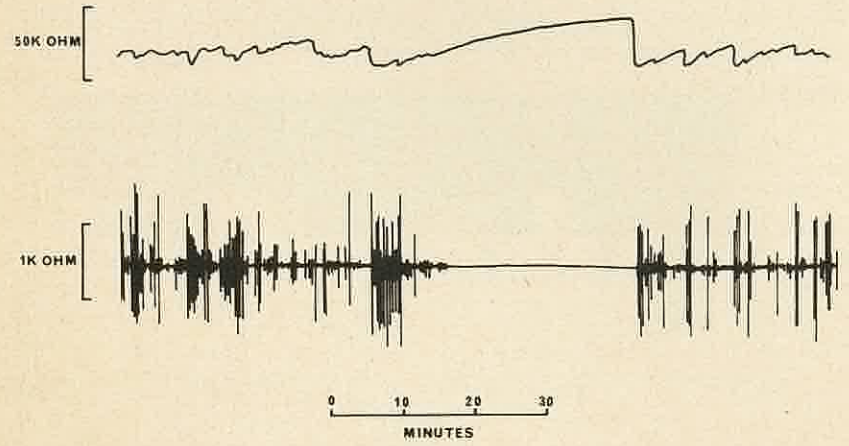
tached to each upper eyelid to give a signal of a millivolt or more when movement of the cornea beneath the closed eyelid deforms the strain gauge. This method has not yet been widely used.

It seems that the frequency of REMs during stage REM sleep is an indication of the intensity of sleep and dreaming processes. Under conditions of psychological stress the frequency of REMs increases whereas the duration of stage REM periods decreases.[47] The degree of mental retardation in patients with Down's syndrome, phenylketonuria, or brain damage is negatively correlated with the degree of eye movement activity during dreaming.[21]

Electromyogram.—An electrical assessment of muscle activity in the submental region is recommended by some as an additional source of information to the EEG and EOG in monitoring the stages of sleep.[30] The amplitude of the EMG measured over the suprahyoid muscles decreases slightly when the subject goes to sleep. Just before and during each stage REM epoch the muscle tone decreases still further.[48] This hypotonia is fairly general throughout head and neck muscle groups but paradoxically is associated with brief spikes of muscle activity which follow periodic discharges in the pons during stage REM sleep.

Because of the very small amplitude of the EMG, electrical interference is a common problem, but this can be reduced by the use of large flexible stainless steel mesh electrodes moulded over the skin.[30] At the conventional recording speed it is the envelope width of the EMG record which is important rather than individual wave forms. This record also provides an indication of body movements and so enables ready identification of brief arousals, the number and duration of which are related to the quality of sleep.

Fig 4.—The pattern of basal skin resistance (upper) and galvanic skin responses (lower) during wakefulness and a 27-minute period of sleep when the resistance steadily increased and fluctuations ceased.



Electrical Resistance of Skin.—Johns et al[49] have described a method for monitoring sleep and wakefulness with continuous measurements of the electrical resistance of skin. A small constant current is passed between electrodes attached to the fingers, the potential difference which is developed being proportional to the resistance of the skin. The basal level of skin resistance (BSR) undergoes large slow variations over periods of minutes or hours. In addition there are small, rapid fluctuation in resistance occurring over five or ten seconds, the so-called galvanic skin responses (GSR). These two measurements of skin resistance can be recorded separately at slow chart speeds (Fig 4). The pattern of BSR and the frequency of GSRs is such that one can determine accurately the onset of sleep, the time of any arousals no matter how brief, and the time of finally waking up. The frequency of GSRs occurring spontaneously during wakefulness can be used as a measure of arousal and anxiety. Their frequency decreases markedly in drowsiness and stage 1 sleep but, paradoxically, increases again, especially during stages 3 and 4 sleep, when low-amplitude GSRs occur up to 10 and 12 times per minute. Presleep psychological stress increases their frequency during all sleep stages.[50] This method therefore introduces an objective measure of the duration of sleep and probably also a measure of sleep quality which is independent of the EEG and EOG. Where it is thought to be difficult or unnecessary to carry out a full analysis of sleep stages using the EEG and EOG, skin resistance methods have proved simple and reliable and can be applied to hospital patients

who are seriously ill.

Body Motility.—Measuring body motility was one of the first objective methods used in sleep research. Kleitman[51] used mechanical apparatus connected to a special bed to measure the frequency and total duration of body movements during the night. He showed that normal subjects move between about 20 and 60 times during a night, each movement period usually lasting a few seconds. The frequency of movements increases during the night. However, because of the wide variation in the pattern of body movements in normal subjects, Kleitman concluded that "One is certainly not justified in judging the quality of sleep entirely or mainly on the basis of how many movements per hour or per night the sleeper made."[51] Such a statement may have influenced many investigators against measuring body motility, Oswald et al[52] included this measurement in their detailed study of the sleep of patients suffering from melancholia. Neither body motility nor the frequency of stage changes significantly differentiated the patients from controls, but both factors were decreased by the hypnotic drug heptabarbital. The frequency of movements decreased from stage 1 to stage 4 sleep and increased in REM stage. Monroe[16] used a piezoelectric crystal attached to the bedsprings to show that poor and good sleepers had significant differences in their body motility, especially in stages 3 and 4 sleep. Baekeland et al[18] found that normal subjects gave ratings of their own body movements and quality of sleep which were related significantly to the total number of body movements, especially to those associated with waking alpha waves in the EEG.

It seems, therefore, that measurements of body movements may provide worthwhile information about the quality of sleep, especially when combined with other measurements. We have found that major body movements are associated with a decrease in basal skin resistance and a high amplitude GSR which suggests a partial arousal at that time. This change is not due simply to electrode artefact because it occurs gradually over several seconds and is much less dramatic than the sudden decrease in BSR associated with waking up.[49] The presence of body movements may also be indicated by muscle artefacts in the EEG record, so it is doubtful if special equipment such as a piezoelectric crystal in the mattress is necessary.

Personal Observation.—Attempts have been made, particularly with children, to evaluate both the duration and quality of sleep by intermittent observation.[53] This method is clearly limited in that a subject may frequently wake up for only a few minutes without this being observed. The time of initially falling asleep and finally waking up usually could not be obtained with a resolution of less than about 15 minutes. Nevertheless, Platman and Fieve[20] showed that the total time which psychiatric patients spent awake in bed attempting to sleep, estimated by nurses' observations every half hour, distinguished periods of depression from recovery periods. Some of the objections to this method can be overcome by continuous observation of the sleeper, for instance, by watching on closed-circuit television from another room.[49] However, it remains difficult to distinguish periods of quiet wakefulness from sleep.

Other Methods.—Many other physiological factors have been shown to vary during sleep. Heart rate, blood pressure, and respiratory rate all tend to decrease during NREM sleep and to increase and show greater variability during REM sleep.[54] Penile erection occurs during most periods of REM sleep in males.[54] Variations such as these have mainly been of interest in relating psychological aspects of sleep and dreams to bodily changes.[55] There is some evidence to suggest that subjectively poor sleepers continue to have relatively high levels of activation of the central nervous system compared to good sleepers, even in delta-wave sleep.[16] This view is supported by the finding of a significant elevation in the level of adrenocortical activity, day and night, in subjects with even minor degrees of sleep disturbance (Johns MW, Gay TJA, Masterton JP, Bruce D, unpublished data). Some such biochemical measure may provide a routine method for assessing the restfulness or otherwise of sleep, at least for comparisons within individuals.

Routine measurements of arousal threshold during the night by presenting graded stimuli and eliciting behavioral responses have not been widely carried out, presumably because they are difficult and involve disturbing the very sleep which one is trying to assess. However, an unusual method of testing hypnotic drugs in humans has been based on the resistance to being waked by increasing bladder pressure as a result of drinking a large volume of water before sleeping.[56]

Automatic Methods of Sleep Analysis

We have seen that there are many different measurements which can be used to assess objectively the duration and quality of human sleep. In some instances these measurements involve equipment which is complicated and expensive. A common approach in recent years has been to use a polygraph to record several measurements at the one time on paper, the speed of which has been determined largely by the need to run the EEG at fast chart speeds to facilitate visual scoring. This leads to

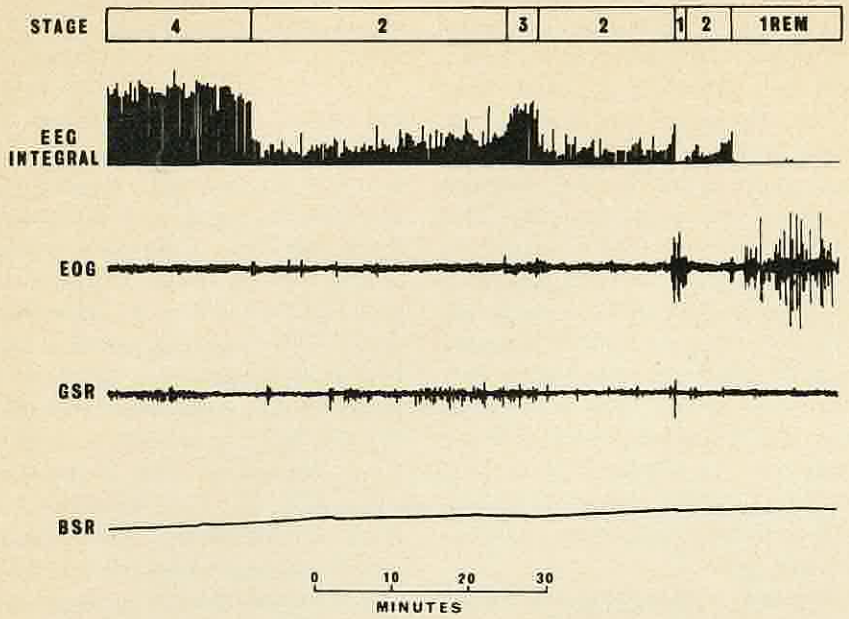


Fig 5.—Simultaneous recordings during various stages of sleep of (1) the integral over 20 sec periods of EEG activity passing a 2 Hz filter, (2) EOG showing greatest activity during stage REM, (3) GSRs which decrease on going to sleep initially but reappear in bursts during the night, (4) BSR which increases gradually until near the end of the night's sleep. It decreases sharply upon waking.

the accumulation of thousands of feet of paper for every few patients studied.[52] Having produced such voluminous records, the reproducible and accurate scoring of sleep stages from the EEG and EOG remains difficult and time consuming.

Several attempts have been made to make sleep analysis simpler and more reliable by using automatic EEG analysis. Use has been made of analogue systems to integrate the amplitude of the raw EEG or some portion of it passing particular bandpass filters which may be of electromechanical or electronic type.[57-59] Digital computers have been used to provide frequency analysis by counting base line crossings of the raw EEG or its first derivation.[60] Fourier analysis has provided spectra of power or amplitude versus frequency for short segments of EEG, the shape of which can be recognized by multiple regression or discriminant analysis to be characteristic of particular sleep stages.[61,62] Most of these methods have provided criteria by which some of the sleep stages may be distinguished by using on-line or off-line automatic analysis. However, analysis based on the EEG alone has not proved very reliable in distinguishing drowsiness from stage 1 or stage REM.[61] In addition, the high cost of digital computer analysis of all-night sleep EEGs and the relatively large volume of information which it produces have decreased its value. No such system of analysis has yet proved sufficiently reliable or simple to justify its frequent or routine use in all-night sleep monitoring.

A complex system for sleep analysis has been designed by Kripke et al[63] and is based on EEG, EMG, EOG, ECG, and respiration data. This system has bandpass filters, rectifiers, and integrators for the EEG and EMG. Digital output is produced for each channel of information and then a computer is used with a stepwise discriminant analysis program for data analysis.

We have developed an analogue method for sleep analysis based on the EEG, EOG, and skin resistance measurements. The integral over-20-seconds segments of the rectified EEG signal passing a 2 Hz band-pass filter provides an analogue signal which is proportional to the number and amplitude of delta waves in the raw EEG. This readily distinguishes stages 2, 3, and 4 sleep but does not reliably distinguish drowsiness from stage 1 or stage REM sleep. However, continuous skin resistance measurements provide a simple method for distinguishing wakefulness from sleep. Similarly, the EOG distinguishes stage REM periods at the time when skin resistance recordings indicate sleep rather than wakefulness and the EEG analysis indicates stage 1-type EEG (Fig 5). This combined information can be recorded on-line at very slow chart speeds so that the details of an entire night's sleep are available on a few feet of paper. Much paper, time, and effort are saved by this method when compared with the usual paper write-out of the EEG. A disadvantage of the automatic analysis equipment which has been used so far is that it must be calibrated for each experimental subject's EEG in known sleep stages. This involves visually scoring small samples of the raw EEG and relating the stage of sleep to the amplitude of the analogue signal derived from the EEG analysis. Further work is in progress to develop analogue analysis equipment which does not require such calibration for each night's recording.

Comment

The best method to use for a particular investigation of sleep will depend on the specific objectives, the number of experimental subjects, and the equipment which must be used. It now seems clear that no single method of sleep assessment, whether subjective or objective, can give enough information to permit

neglect of other methods.

Subjective responses by groups of subjects to a detailed sleep questionnaire or daily sleep records can in most cases give estimates of such parameters as delay to sleep onset, number of night awakenings, and total duration of sleep which will be accurate to within a few minutes. These estimates may involve larger errors when individual reports in normal subjects are considered, and especially so in the elderly and in patients with functional or organic psychiatric illness. There is some evidence that subjective reports give more reliable information about certain aspects of sleep at home than do detailed EEG studies in the sleep laboratory which tend to make sleep more disturbed than usual. Also, people often sleep for short periods during the day, and may catch up on a minor sleep loss by sleeping longer during the weekend, variations which only subjective methods can describe.

In those circumstances where accurate and detailed information about sleep in individual subjects is required, the EEG forms the basis of the method of choice but it must be combined with the EOG and perhaps also the EMG, skin resistance, or other measurements. The EEG is required to give the stage of sleep, but must be interpreted with the reservation that drugs and metabolic disturbances may cause dissociation between behavioral and EEG signs of sleep and wakefulness. Therefore it is unwise to rely too heavily on the EEG alone as the indicator of sleep. Skin resistance measurements have proved valuable alone or in combination with the EEG and EOG, especially when the latter recordings involve analogue signals derived from sleep analysis systems.

In future, sleep research should pay more attention to information about the quality of sleep other than that inferred from EEG stages. Johnson et al[64] have reported that the clinical and psychological ratings of chronic alcoholics were related less to the amount of any particular stage of sleep than to the overall quality of sleep as measured by latency to onset, number of awakenings, and total duration of sleep. All of these measurements could have been made easily from skin resistance recordings alone.[49] The use of better automatic and integrated systems for sleep analysis which are based on several factors and a better understanding of biochemical changes eventually may enable us to explain the function of sleep and the effects of its disturbance on the physical and psychological well-being of patients.

Summary

There has been a great increase in sleep research in recent years, the results of which are of increasing relevance to general medicine and surgery. In this review, most of the subjective and objective methods used at present for assessing the duration and quality of sleep are briefly discussed. These include sleep questionnaires, daily sleep charts, the electroencephalogram, electrooculogram, electromyogram, electrical resistance of skin, body motility, and biochemical factors. No single method can give enough information to permit neglect to others. The best methods of use under particular circumstances depend on experimental objectives and the number of subjects to be studied. In future, automatic systems for sleep analysis based on several physiological parameters may provide a better understanding of the functions of sleep and dreams.

This investigation was supported by a National Health and Medical Research Council of Australia Medical Postgraduate Research Scholarship awarded to the author.

Nonproprietary and Trade Names of Drug

Heptabarbital—*Medomin.*

References

1. Loomis AL, Harvey EN, Hobart GA: Cerebral states during sleep, as studied by human brain potentials. *J Exp Psychol* **21**:127-144, 1937.

2. Dement W, Kleitman N: Cyclic variations in EEG during sleep and their relation to eye movements, body motility, and dreaming. *Electroenceph Clin Neurophysiol* **9**:673-690, 1957.

3. Kornfield DS, Zimberg S, Malm J: Psychiatric complications of open-heart surgery. *New Eng J Med* **273**:287-292, 1965.

4. Armstrong RH, Burnap D, Jacobson A, et al: Dreams and gastric secretions in duodenal ulcer patients. *New Physician* **14**:241-243, 1965.

5. Nowlin JB, Troyer WG Jr, Collins WS, et al: The association of nocturnal angina pectoris with dreaming. *Ann Intern Med* **63**:1040-1046, 1965.

6. Honda Y, Takahashi K, Takahashi S, et al: Growth hormone secretion during nocturnal sleep in normal subjects. *J Clin Endoc* **29**:20-29, 1969.

7. 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.

8. Weitzman ED, Schaumburg H, Fishbein W: Plasma 17-hydroxycorticosteroid levels during sleep in man. *J Clin Endocr* **26**:121-127, 1966.

9. Cohen EL: Length and depth of sleep. *Lancet* **2**:830-831, 1944.

10. Chrysanthis K: The length and depth of sleep. *Acta Med Orient* **5**:152-155, 1946.

11. Webb WB, Stone W: A note on the sleep responses of young college adults. *Percept Motor Skills* **16**:162, 1963.

12. McGhie A: The subjective assessment of sleep patterns in psychiatric illness. *Brit J Med Psychol* **39**:221-230, 1966.

13. Haider I: Patterns of insomnia in depressive illness: A subjective evaluation. *Brit J Psychiat* **114**:1127-1132, 1968.

14. Webb WB: Sleep characteristics of human subjects.

Bull Brit Psychol Soc **18**:1-10, 1965.

15. 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.

16. Monroe LJ: Psychological and physiological differences between good and poor sleepers. *J Abnorm Psychol* **72**:255-264, 1967.

17. Lewis SA: Subjective estimates of sleep: An EEG evaluation. *Brit J Psychol* **60**:203-208, 1969.

18. Baekeland F, Hoy F: Reported vs. recorded sleep patterns. Read before the Annual Conference of the Association for the Psychophysiological Study of Sleep, Santa Fe, NM, 1970.

19. Samuel JG: Sleep disturbance in depressed patients: Objective and subjective measures. Brit J Psychiat **110**:711-719, 1964.

20. Platman SR, Fieve RR: Sleep depression and mania. *Brit J Psychiat* **116**:219-220, 1970.

21. Feinberg I, Braun M, Shulman E: EEG sleep patterns in mental retardation. *Electroenceph Clin Neurophysiol* **27**:128-141, 1969.

22. Oswald I: *Sleeping and Waking*. Amsterdam, Elsevier, 1962.

23. Orme JE: Time estimation and personality. *Acta Psychol* **20**:285-303, 1962.

24. Jick H, Slone K, Shapiro S, et al: Clinical effects of hypnotics: II. An epidemiologic study. *JAMA* **209**:2016-2020, 1969.

25. Lewis HE, Masterton JP: Sleep and wakefulness in the arctic. *Lancet* **1**:1262-1266, 1957.

26. Masterton JP: Patterns of sleep, in Edholm OG, Bacharach AL (eds): *The Physiology of Human Survival*. London, Academic Press Inc, 1965, pp 388-397.

27. Tune GS: Sleep and wakefulness in normal human adults. *Brit Med J* **2**:269-271, 1968.

28. Tune GS: Sleep and wakefulness in 509 normal human adults. *Brit J Med Psychol* **42**:75-80, 1969.

29. Tune GS: The influence of age and temperament on the adult human sleep wakefulness pattern. *Brit J Psychol* **60**:431-441, 1969.

30. Rechtschaffen A, Kales A: *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects*, bulletin 204. Public Health Service, Bethesda, Md, 1968.

31. Monroe LJ: Inter-rate reliability and the role of experience in scoring EEG sleep records: Phase 1. *Psychophysiology* **5**:376-384, 1969.

32. Webb WB, Agnew HW: Measurement and characteristics of nocturnal sleep. *Prog Clin Psychol* **8**:2-27, 1968.

33. Feinberg I, Carlson VR: Sleep variables as a function of age in man. *Arch Gen Psychiat* **18**:239-250, 1968.

34. Mendels J, Hawkins DR: Sleep and depression: Further considerations. *Arch Gen Psychiat* **19**:445-452, 1969.

35. Feinberg I, Braun M, Koresko RL, et al: Stage 4 sleep in schizophrenia. *Arch Gen Psychiat* **21**:262-266, 1969.

36. Agnew HW Jr, Webb WB, Williams RL: The first night effect: An EEG study of sleep. *Psychophysiology* **2**:263-266, 1966.

37. Rechtschaffen A, Hauri P, Zeitlin M: Auditory awakening thresholds in REM and NREM sleep stages. *Percept Motor Skills* **22**:927-942, 1966.

38. Williams HL: The problem of defining depth of sleep: *Res Publ Assoc Nerv Ment Dis* **45**:277-287, 1967.

39. Corvalan JC, Hayden MP: Depth of sleep and auditory thresholds during 24 hour recording. Read before the Annual Conference, Association for the Psychophysiological Study of Sleep, Santa Fe, NM, 1970.

40. Williams HL, Hammack JT, Daly RL, et al: Responses to auditory stimulation, sleep loss and the EEG stages of sleep. *Electroenceph Clin Neurophysiol* **16**:269-279, 1964.

41. Bradley PB, Elkes J: The effect of some drugs on the electrical activity of the brain. *Brain* **80**:77-117, 1957.

42. Mirsky AF, Pragay EB: The relation of EEG and performance in altered states of consciousness. *Res Publ Assoc New Ment Dis* **45**:514-534, 1967.

43. Harden A, Glaser GH, Pampiglione G: Electroencephalographic and plasma electrolyte changes after cardiac surgery in children. *Brit Med J* **4**:210-213, 1968.

44. Kahn E, Fisher C: The sleep characteristics of the normal aged male. *J Nerv Ment Dis* **148**:477-494, 1969.

45. Shackel B: Eye movement recording by electro-oculography, in Venables PH, Mortin I (eds): *A Manual of Psychophysiological Methods*. Amsterdam, North Holland Publishing Co, 1967, pp 299-334.

46. Baldridge BJ, Whitman RM, Kramer M: A simplified method for detecting eye movements during dreaming. *Psychosom Med* **25**:78-82, 1963.

47. Baekeland F, Koulack D, Lasky R: Effects of a stressful presleep experience on electroencephalograph-recorded sleep. *Psychophysiology* **4**:436-443, 1968.

48. Jacobson A, Kales A, Lehmann D, et al: Muscle tonus in human subjects during sleep and dreaming. *Exp Neurol* **10**:418-424, 1964.

49. Johns MW, Cornell BA, Masterton JP: Monitoring sleep of hospital patients by measurement of electrical resistance of skin. *J Appl Physiol* **27**:898-901, 1969.

50. Lester BK, Burch NR, Dossett RC: Nocturnal EEG-GSR profiles: The influence of presleep states. *Psychophysiology* **3**:238-248, 1967.

51. Kleitman N: *Sleep and Wakefulness*. Chicago, *University of Chicago Press* 1963.

52. Oswald I, Berger RJ, Jaramillo RA, et al: Melancholia and barbiturates: A controlled EEG, body and eye movement study of sleep. *Brit J Psychiat* **109**:66-78, 1963.

53. Parmelee AH, Wenner WH, Schulz HR: Infant sleep patterns: From birth to 16 weeks of age. *J Pediat* **65**:576-582, 1964.

54. Snyder F: Autonomic nervous system manifestations during sleep and dreaming. *Res Publ Assoc Nerv Ment Dis* **45**:469-487, 1967.

55. Hobson JA, Goldfrank F, Snyder F: Respiration and mental activity in sleep. *J Psychiat Res* **3**:79-90, 1965.

56. Isaacs B: A method for evaluation of hypnotics. *Lancet* **1**:556-558, 1957.

57. Koga E: A new method of EEG analysis and its application to the study of sleep. *Folia Psychiat Neurol Jap* **19**:269-278, 1965.

58. Maulsby RL, Frost JD Jr, Graham MH: A simple electronic method of graphing EEG sleep patterns. *Electroenceph Clin Neurophysiol* **21**:501-503, 1966.

59. Agnew HW Jr, Parker JC, Webb WB, et al: Amplitude measurement of the sleep electroencephalogram. *Electroenceph Clin Neurophysiol* **22**:84-86, 1967.

60. Itil TM, Shapiro DM, Fink M, et al: Digital computer classifications of EEG sleep stages. *Electroenceph Clin Neurophysiol* **27**:76-83, 1969.

61. Lubin A, Johnson LC, Austin MT: Discrimination among states of consciousness using EEG spectra. *Psychophysiology* **6**:122-132, 1969.

62. Larsen LE, Walter DO: On automatic methods of sleep staging by spectra of electroencephalograms. *Electroenceph Clin Neurophysiol* **28**:459-467, 1970.

63. Kripke DF, Clark C, Merritt JA: *A System for Automated Sleep Staging and Physiological Data Reduction*. Holloman Air Force Base, New Mexico, 1969.

64. Johnson LC, Burdick JA, Smith J: Sleep during alcohol intake and withdrawal in the chronic alcoholic. *Arch Gen Psychiat* **22**:406-418, 1970.