The Derangements Affecting Sleep

by VIVIEN BONIUK '64

Sleep is a physiological necessity for normal metabolism and functioning, ranking in importance with the "basic needs" of food, shelter, and clothing, as well as being a subjective phenomenon of considerable interest to all thinking beings. Complaints referable to sleep pervade the realm of almost all fields of Medicine—General Practice, Internal Medicine, Surgery, Psychiatry, Pediatrics.

CLASSIFICATION.

Like any other physiological phenomenon, the derangements related to sleep may be classified as:

I. Too much — Hypersomnias.
II. Too little — Hyposomnias and Insomnia.
III. Disturbances of — Dyssomnias.

It is interesting to reiterate here the theory that individual sleep requirements form a spectrum, which, like biological measurements of any other kind takes the shape of a "bell curve". The degree of functioning of the Reticular Activating System (RAS), that part of the CNS most closely associated with consciousness, determines the "need for sleep". Most of the population falls within the range of "needing" 7-8 hours per night. It has been suggested that true idiopathic narcolepsy (to be discussed later) involves the portion of the populace within the outermost range of the curve—that is, they "need" much more sleep. Conversely, there are recorded cases of fortunate people, like Thomas Edison, who subsisted very well on 3-4 hours per night. A person with a hypo-functioning RAS would be less responsive to the constant barrage of awakening stimuli, and so be susceptible to falling asleep at the least provocation—that is, in "drowsy situations", for example a warm room, a boring lecture, or a movie.

I. TOO MUCH SLEEP.

A. Prolonged Somnolence.

This has been most commonly seen following encephalitis lethargica. An epidemic began in Europe in 1915, in the United States in 1918, and remained sporadic until 1926—since which time, very few cases, if any, have been seen. The disease was best described by von Economo in 1923. Although named for the somnolent effects, the disease causes a varying and complex disturbance of the sleep-wakefulness rhythm, acutely, chronically, or acutely with chronic after-effects. The etiology is viral, but the virus has never been isolated. Pathologically, there is a localized inflammatory lesion in the periventricular gray matter and floor of the third ventricle (hypothalamus) and neighbouring parts of the third ventricle.

Clinically, von Economo recognized three stages:
(1) Initial—Psychic symptoms, paresthesias, and fever (lasting a few days to weeks).
(2) Hyperkinetic—Marked restlessness (lasting up to six weeks).
(3) Lethargic—Psychic symptoms and restlessness disappear and there may result an apathetic, adynamic stage.

This picture was, of course, not invariable; many deviations and unusual after effects occurred. In most patients, the ocular mechanisms were often affected so that sleep could be induced by fixation on an object, or closing the eyes, as well as by hyper-ventilation, which tends to lower the "level of consciousness". The mortality was 30 - 40 per cent and sequelae were suffered by 20 per cent including "pseudopsycho-neuroses (headaches, irritability, loss of mental powers), athetosis, salivation, spasticity, and Parkinsonism."
B. Narcolepsy—literally, this means “sleep seizure”.

This disorder was first described by Gelineau in 1880. It consists of sudden, short-lasting attacks of sleep, occurring during the customary waking hours of the individual, which may be associated with attacks of muscular tonelessness, known as cataplexy, in which the patient is aware but helpless, and he cannot move.

Etiologically, the disorder may be:

(1) Idiopathic.

(2) Symptomatic—postencephalitic, and secondary to diseases involving certain anatomical areas.

As mentioned before, RAS hypoactivity, on an hereditary basis is postulated as being the prime factor in the idiopathic variety.

Adolescence, or young adulthood is the period of onset; 70 per cent of cases have their onset before age 26. It is more common in males, and can occur in any racial group.

Clinically, True Idiopathic Narcolepsy comprises a tetrad:

(1) Persistent drowsiness—induced by boredom and monotony.

(2) Cataplectic attacks occurring with any emotion, especially laughter and excitement. (The patient with Narcolepsy finds himself in a paradoxical position—that is, avoiding the factors which precipitate the drowsiness may result in accentuation of the cataplectic phase.)

(3) Sleep paralysis.

(4) Hypnagogic hallucinations.

There may be as many as 10-15 attacks a day, precipitated by boredom and monotony, or any drowsy situation. The sleep is normal, and it comes on as an irresistible impulse, but may be so deep that there are no tendon reflexes elicited, and
a positive Babinski flexor response may appear. Sleep lasts an average of 10-20 minutes.

EEG—It has been noted that the pattern is normal when standing, but as soon as the person lies down, the normal alpha waves give way to delta waves (sleep pattern).

Treatment. A word on treatment is pertinent here, since this disorder is very amenable to therapy, enabling normal activity to be resumed. CNS stimulants, especially caffeine, amphetamine, and Ritalin are most helpful.

II. TOO LITTLE SLEEP.

A. Experimental Deprivation of Sleep.

In experiments carried out where subjects were deprived of sleep for 90 hours, it was shown that although they could still perform many functions at normal levels, there was diminished ability at any task requiring a span of concentration, or use of the higher levels of cortical integration. It was found that there was decreased sensory acuity, quickness of reaction, motor speed, and memorizing ability, associated with increased susceptibility to pain (otherwise, these effects are very similar to those experienced following the ingestion of a large quantity of alcohol). Psychic symptoms include a nasty disposition and visual hallucinations. Laboratory measurements indicate a fall in body temperature, and an increase in polymorphonuclear leucocytes, with a decrease in lymphocytes.

Pathologic studies of dogs experimentally deprived of sleep (done in 1913 and repeated in 1937) show lesions in the CNS, liver, and adrenals. In the CNS, there are typical cellular degenerative changes in the pyramidal cells of the cortex, Purkinje cells of the cerebellum, and the damage is proportional to the sleepiness of the dogs. If the cerebrospinal fluid from the sleepy dogs is injected into normal dogs, it induces sleepiness without degenerative changes.

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The ability to withstand the effects of loss of sleep, and maintain normal cerebral function can be prolonged with the aid of CNS stimulants such as amphetamine. Recovery from loss of sleep can be obtained with less than 35 per cent of the original amount of sleep lost.

B. Insomnia and Hyposomnia.

Since the effectiveness of sleep is a function of both depth and length, lessening of the duration of either of these, or both, acutely or chronically, may be defined as hyposomnia.

ETIOLOGICAL CLASSIFICATION OF HYPOSOMNIA.

(1) Exogenous—Environmental or climatic. This may be occupational, or due to excesses of tobacco or coffee, too much humidity in the air, or a party in the apartment overhead.

(2) Endogenous: (a) Essential—there is presumably an hereditary predisposition, as mentioned before, involving the hyperactivity of the RAS. These people soon accustom themselves to their individual “saturation point” of sleep.

(b) Secondary to mental, psychic nervous, or organic causes. These need no extensive elaboration, since examples of exogenous hyposomnia have been experienced personally by all, and examples of the endogenous type are frequently encountered in medical practice.

Chronologically, in relation to sleep, the hyposomnia may be:

(1) Initial—Usually to nervous activity, such as the worried person, who is unable to fall asleep.

(2) Intermittent—Usually the result of unpleasant dreams or organic disturbances. For example, a person who suffers from gastric discomfort with flatulence,
will awaken some time during the night, relieve his symptoms with sodium bicarbonate, and fall asleep again.

(3) Terminal—This occurs in the elderly, who need less sleep, but nevertheless, go to bed at the same hour as usual, and wake up early. This is also seen in the psychotic depression, and is an important diagnostic point.

In the normal individual, a sense of fatigue leads to a desire to sleep, for which muscular relaxation is essential. If muscular relaxation is delayed, various mechanisms are utilized, according to the individual’s experience, to attain this state of relaxation. In psychic fatigue, muscular relaxation cannot be achieved easily, the person cannot sleep; this produces more anxiety, which perpetuates the cycle.

Insomnia, however, is a complaint which accompanies a great many organic ailments, and cannot merely be accepted as an indication of neuroticism, although this is the most common cause.

III. DYSSOMNIAS — DISTURBANCE OF SLEEP.

A. Aberrations in sleep-waking rhythm.

This encompasses conditions of prolonged somnolence, alternating with periods of wakefulness, and occurs most commonly following encephalitis, meningitis, or with trauma or tumors or areas related to maintaining the normal sleep-waking rhythms.

B. Nightmares and Night Terrors.

Nightmares are due mainly to digestive, and occasionally circulatory disturbances, as experienced by those who indulge in certain spicy delicacies before retiring; these tend to distend the stomach with food or gas, pressure against adjacent structures produces afferent impulses, interpreted centrally as somatic.

Night terrors occur in children between three to eight years of age, who awake in a fearful state, proclaiming the presence of terrifying enemies. Usually, they are
neither aware, nor remember the incident the next day, and this condition generally fades after eight years of age. These night terrors may also be caused by organic disturbances, such as infections, intestinal parasites, or CNS lesions, but most commonly occur in children with a hereditary predisposition (i.e.—a nervous temperament), often complicated by environmental difficulties.

C. Somnambulism and Sleep Automatism.

In these conditions, there is a separation of the behaviour patterns from the patterns of awareness. The site for the correlation of these patterns is believed to be in the diencephalon.

Automatic movements and patterns of action often occur with an associated dream. The patient does not easily regain consciousness even with the stimulus of a loud noise or pain-producing stimuli. The personality of these individuals described as being immature, inadequate, and non-aggressive, with repressed unconscious conflicts. Several cases have been reported of somnambulistic homicide, which is never premeditated or willful; legally, they can plead a state of unawareness at the time of the deed, and this is grounds for acquittal.

D. Disturbances in Transitional Periods of Sleep.

(1) Somnolescent Starts (or myoclonic twitches). In some individuals who are tense or nervous, sleep comes on slowly, and maintained nervous activity in motor areas provoke a sudden movement of an arm or a leg. These immediately pass off and are not indicative of organic dysfunction.

(2) Sensory Paroxysms. A very similar mechanism is operative in the sensory system. There may be experienced an auditory hallucination of a clanging or crashing sound, vestibular hallucinations of being lifted, and dashed to earth, or visual
hallucinations of apparitions. These are often termed hypnagogic hallucinations, and are not indicative of organic dysfunction.

(3) Nocturnal Paralysis. The patient awakens, aware, but unable by any effort of will to move an arm or leg; however, he is easily aroused by an external cutaneous stimulus of any sort. (One may postulate a similar explanation as for 1 and 2 above, relating to the inertia of nervous activity; however, in this case, the resting state is maintained.)

E. Sleep Palsies and Acroparesthesias.

In sleep, the immobility of limb, below the level of awareness, may maintain pressure on a superficial nerve (ulnar, radial, peroneal). If this pressure is maintained for more than 30 minutes, a paralysis may result, which is usually not serious, and only lasts a few days. A common example of this is the “Sunday morning palsy” of the Saturday night drinker, who falls asleep with his arm over the back of a chair, producing direct pressure on the brachial nerve. The afferent impulses which are usually operative in producing changes of position on normal sleep, do not produce such changes in these individuals due to an intoxication of central mechanisms.

Acroparesthesias include numbness, tingling, prickling, “pins and needles” sensations, most commonly along the course of sensory distribution of the median nerve. This occurs in individuals who do heavy work, and may be relieved by a period of hospitalization or a holiday. Relief may also be effected by sectioning the flexor retinaculum, since these sensations may be due to compression of the median nerve in the carpal tunnel at the wrist.

SUMMARY.

A classification and brief discussion of the disorders affecting sleep has been presented. Such a classification may be useful to the practicing physician in understanding this common patient complaint—a disturbance of sleep.

REFERENCES:


Kleitman, N.: Sleep and Wakefulness, 1943.
