Primary Sleep Disorders: A Review

An Honors Thesis (ID 499)

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Muncie, Indiana

February, 1978
For most people, sleep is a regular and welcome period of rest and unresponsiveness, sometimes punctuated by dreams, which occurs daily and lasts about eight hours. However, about one in five Americans have one form or another of sleep disorder. For them, sleep can be terrifying, insufficient, overabundant, or, in some cases, fatal. The purpose of this paper is to take an in-depth look at conditions in which disturbances of sleep are the primary and often only symptoms. Karacan calls these primary sleep disorders, and lists them as insomnia, narcolepsy, chronic hypersomnia, the Klein-Levin syndrome, the Pickwickian syndrome, sleep paralysis, and nightmares. In addition, sleep apnea will also be discussed.

Many sleep disorders involve a complaint of excessive daytime sleepiness (EDS). The most common of these is narcolepsy, which is defined to be "an illness involving excessive daytime sleepiness, sleep episodes, and cataplexy, with or without sleep paralysis, frightening hypnagogic hallucinations, and disturbed nocturnal sleep" (Guilleminault and Dement, 1977, p. 17). Automatic behavior can also be symptomatic of narcolepsy.

Excessive daytime sleepiness usually consists of sudden, irresistible periods of sleep, usually about fifteen minutes long (although they may range from five to thirty minutes), from which the narcoleptic awakens feeling alert and refreshed. There is usually a refractory period of one to five hours between sleep attacks, and they most often occur in boring or monotonous situations. Zarcone reports on a patient who fell asleep "during a bombing run in a B-17" (Zarcone, 1973, p. 1158).
Somewhat more common are reports of falling asleep during intercourse or while driving a car. One study reported that 40% of the narcoleptics surveyed had fallen asleep at the wheel, and another study found that more than 10% of the narcoleptics they questioned had had at least one automobile accident due to EDS. In general, narcoleptics also report feeling sleepy between sleep attacks.

Although the presence of cataplexy, as well as EDS, is now crucial to a diagnosis of narcolepsy, it has not always been so. Yoss and Daly reported that about one-fourth of all patients diagnosed as narcoleptic suffered from excessive sleepiness alone, and about 8% more of all narcoleptic patients exhibited sleep attacks with hypnagogic hallucinations and/or sleep paralysis, but without cataplexy. Roth has used the term "independent narcolepsy" for the cases of excessive sleepiness only, and, although independent narcolepsy is not a form of narcolepsy proper, Roth's terminology will be used in this paper.

Cataplexy consists of sudden, full or partial loss of muscle tone, almost always triggered by some strong emotion, such as laughter, anger, surprise, fright, exultation, or elation. The attack may range in severity from a feeling of subjective weakness to total paralysis, and may affect only certain groups of muscles, or virtually all muscles. The extraocular muscles are occasionally affected; cataplexy usually occurs only in the voluntary muscles, and therefore the respiratory system (as well as other involuntary muscular systems) is almost never disrupted.
The narcoleptic is most often fully conscious during the cataplectic attack and able to remember sounds heard during cataplexy, although unable to speak or move. Electroencephalographic recordings taken during cataplexy are normal. A cataplectic attack may last only a few seconds, is rarely longer than two minutes, and may be terminated immediately if the narcoleptic hits something when he falls down. An exception to this may occur when a narcoleptic is sitting or reclining during a cataplectic attack, which then develops into a sleep attack. The onset of cataplexy rarely occurs before the onset of EDS, and most often happens either the same year as the first sleep attacks or in the five years immediately after EDS starts.

Like cataplexy, sleep paralysis is usually experienced as full consciousness coupled with normal electroencephalographic recordings and the inability to move or speak, most often lasting only a few seconds, although it may last as long as twenty minutes or develop into sleep. Unlike cataplexy, however, sleep paralysis may be accompanied by hallucinations and a feeling of helplessness and intense fear. Also unlike cataplexy, sleep paralysis occurs only during the transition between sleep and wakefulness, and may include respiratory arrests, although these are rare. Sleep paralysis occurs in about 36% of all narcoleptics, in about 15% of all independent narcoleptics, and fairly often in normal people. The rate of such occurrence is unknown, although Dement reports that about half of a large group of Stanford students have experienced sleep paralysis at least once.
Hypnagogic hallucinations often occur simultaneously with sleep paralysis, although they can also occur separately. They happen, like sleep paralysis, in the state between wakefulness and sleep, usually just before the onset of sleep. Hypnagogic hallucinations are false perceptions, predominantly visual, although often auditory or tactile, that occur both in normals and in narcoleptics. The content of these hallucinations may be benign or frightening, although in narcoleptics they are most often terrifying. They typically involve the bedroom or sleeping area, and are often experienced as an imminent attack by some intruder (human, animal, or monster) from which escape is impossible because the limbs are paralyzed. Hypnagogic hallucinations occur in about 37% of all narcoleptics and in about 17% of all independent narcoleptics. Incidence rates in normals are unknown, although the frightening type are often associated with emotional stress.

Disturbed nocturnal sleep may take any one of several forms. An estimated 60% of all narcoleptics suffer from repeated awakenings throughout the night, from which they have no trouble returning to sleep. Nightmares also occur frequently in narcoleptics, as do body movements during sleep. A less common disturbance is sleep apnea, which occurs in approximately 20% of all male narcoleptics, although not in female narcoleptics. Mitchell and Dement have found that disrupted nocturnal sleep occurs in most cases of narcolepsy and usually precedes or occurs with the onset of EDS.

Automatic behavior, estimated to occur in about half of
all cases of narcolepsy, involves performance of an action which cannot be recalled afterwards. Such behavior may last moments or hours and mostly occurs late in the day. It is sometimes preceded by rolling eye movements and blinking, although the narcoleptic is unaware of this. The automatic nature of the behavior may be unnoticed, since such behavior is often a continuation of the previous behaviors.

It has been estimated that there are about 250,000 narcoleptics in the United States, which yields an incidence rate of approximately .1% of the general population, although other estimates are as low as 50,000 or .02% of the population. Narcolepsy occurs equally often in men and women. It most often onsets at about puberty, with about half of all cases showing the first symptoms between 10 and 20 years of age. In women, onset may occur along with pregnancy. Onset of symptoms seldom occurs after the age of forty.

Proper treatment of narcolepsy is, of course, dependent on correct diagnosis. If a person has a distinct history of sleep attacks and cataplexy, no testing is needed to confirm a diagnosis of narcolepsy, although such tests may be used as a guide to treatment. When the history of cataplexy is not distinct, the usual diagnostic procedure involves a sleep recording. Whenever possible, the person is withdrawn from all medication for several days before the test, and is awake during the entire morning of the test. In the afternoon, electrodes are attached to the person so that the electroencephalographic, electromyographic, electrooculographic, and, on some occasions,
the electrocardiographic responses may be recorded. The person is then asked to take a nap. Approximately 90% of all narcoleptics (excluding independent narcolepsy) show evidence of a sleep-onset REM period in this nap, and all narcoleptics show at least one sleep-onset REM period in a 24-hour polygraphic recording. These sleep onset REM periods are not seen in normal people, where there is a delay of 70 to 100 minutes between sleep onset and the first REM period, nor are they seen in people with independent narcolepsy.

Although this diagnostic procedure seems straightforward, the average time between the onset of narcoleptic symptoms and the diagnosis of narcolepsy is fifteen years, and is often more than twenty years. Many narcoleptics will visit four or five different doctors before receiving a correct diagnosis. Part of this is undoubtedly due to the gradual onset of narcoleptic symptoms. Narcoleptics often do not seek medical attention until the symptoms become severe, even incapacitating. Another factor, often leading to incorrect diagnoses, is that many narcoleptics either wrongly report their symptoms (e.g., tiredness or fatigue instead of EDS) or do not report all of their symptoms. The third reason that accurate diagnosis may take so long is that many doctors are unable to identify and treat narcolepsy.

In many cases, narcolepsy is mistakenly diagnosed as something else. The most frequent mis-diagnosis is hypothyroidism. Basal metabolism tests may indicate hypothyroidism simply because the narcoleptic falls asleep during the test,
even though other measures of thyroid functioning show no
abnormality. Even so, many narcoleptics report having been
unsuccessfully treated with thyroid extract prior to correct
diagnosis. Hypoglycemia may be diagnosed instead of narco-
lepsy if the narcoleptic reports fatigue instead of sleepiness.
Although a glucose tolerance test should reveal the falsity of
this diagnosis, the problem may be compounded by the presence,
in some cases, of functional hyperinsulinism, along with sleep
attacks.

In some cases, narcolepsy may be diagnosed as another of
the sleep disorders, especially chronic hypersomnia, the Pick-
wickian syndrome, or apnea. The differentiation between
narcolepsy and apnea is complicated by the occurrence of apnea
in narcoleptic males, so that even a correct diagnosis of apnea
does not rule out the possibility of narcolepsy.

Narcoleptics may report their sleep attacks as 'blackouts',
or cataplexy may be confused with petit mal seizures, leading
to a diagnosis of epilepsy. Ocular symptoms, such as double
vision and ptosis, at the onset of a sleep attack may lead to
a false diagnosis of multiple sclerosis or myasthenia gravis.

Although there is no evidence that narcolepsy is symptomatic
of a psychological disturbance, it may be diagnosed as a
psychological disorder. Especially if thyroid extract is unsuc-
cessful in treating EDS, narcolepsy may be incorrectly diagnosed as
a neurotic fatigue state. If the narcoleptic reports only sleep
paralysis and/or hypnagogic hallucinations, an erroneous diagnosis
of schizophrenia may be made. Such diagnoses have been noted
in children, and differentiating between narcolepsy and schizo-
Phrenia can be complicated by the presence of disordered sleep in schizophrenics. The narcoleptic may also show signs of paranoia and/or schizophrenia after prolonged amphetamine therapy.

A diagnosis, during childhood, of hyperactivity has been reported by about one-tenth of all narcoleptics. This is probably due to the narcoleptic child's attempts to resist EDS. In children, narcolepsy may also be confused with viral encephalitis, although the only similarity between the two is that the term 'sleeping sickness' is often used to describe both conditions.

When proper diagnosis does not occur soon after the onset of symptoms, the narcoleptic may be thought to be stupid, uncooperative, irresponsible, lazy, bored, unable to concentrate, depressed, or mentally ill. The narcoleptic's abilities to learn, to read, and to study are often impaired. These labels and impairments can seriously disorganize the narcoleptic's social and economic life, and result in a distorted self-image. The narcoleptic may be unable to participate in sports (and other activities that may involve surprise or exultation) due to cataplexy, and narcoleptics may restrict their emotional responses in order to help control the cataplexy. They may also withdraw from relationships and situations that provoke cataplectic attacks, thus generating emotional disturbances. Group therapy or family therapy may be helpful in treating these disturbances.

Once narcolepsy has been diagnosed, treatment may be instituted. If the narcolepsy is mild and cataplexy causes few problems, several short naps per day may be all that is needed to
control the sleepiness, enabling the narcoleptic to function normally\(^5\). This type of treatment is also recommended for narcoleptic children, as the effects of other treatments on development are unknown\(^5\).

The most common treatment for more severe narcolepsy involves the use of analeptic drugs. The preferred medication for treating EDS is methylphenidate hydrochloride, 20-80 mg/day\(^4,5,9\). Amphetamines and methamphetamines can also be used to treat EDS\(^4,8\), especially methamphetamine hydrochloride\(^8\) and dextroamphetamine sulphate\(^6\), although they have many side effects. If none of these analeptics can control the symptoms, monoamine oxidase (MAO) inhibitors may be used\(^20\). They control not only EDS but also cataplexy, hypnagogic hallucinations, and sleep paralysis; however, MAO inhibitors have a large number of side effects, including possible optic damage, and interact with many drugs. Also, consumption of any food rich in tyramine (an intermediate stage in the synthesis of epinephrine from tyrosine) while being treated with MAO inhibitors may result in intracranial hemorrhages and death\(^21\). Withdrawal from MAO inhibitors can result in insomnia, extreme anxiety, depression, and suicidal thoughts; for this reason, MAO inhibitors are best used only when all other treatments fail\(^8\).

In order to control cataplexy, imipramine or imipraminic compounds (desipramine or chlorimipramine) are usually used\(^4,5,6,8\). Although they have little or no effect on EDS, they are very effective treatments for cataplexy, sleep paralysis, and hypnagogic hallucinations\(^4,5,6,8\). The usual dosage of imipramine for treat-
ment of cataplexy is 75-100 mg/day\(^5,8\), although chlorimipramine is more effective than either imipramine or desipramine\(^4\). Side effects and interactions are less common with imipraminic compounds than with MAO inhibitors\(^21\). The combination of amphetamines and imipramine is generally considered unsafe\(^4,5\), and therefore seldom used. However, methylphenidate hydrochloride may be used together with imipraminic compounds without deleterious side effects\(^4,5,21\), although dosages must be carefully adjusted since each drug tends to potentiate the other\(^21\).

Primidone has also been used to treat cataplexy\(^22\). To treat disrupted nocturnal sleep and/or sleep paralysis, 10 to 15 mg of diazepam at bedtime may be effective\(^8\), apparently due to the sedative action of diazepam shortening the transition between wakefulness and sleep\(^9\).

No drug treatment currently in use is completely safe or completely effective. Long-term treatment is usually required, due to the fact that narcolepsy only rarely remits\(^3,9\), and tolerances to most of these drugs may develop with long-term usage\(^5,8\). This tolerance may be offset by withdrawing the medication for a period of time and then readministering the same medication at a lower dosage level\(^5\). Gamma hydroxybutyrate, although still being studied and tested, seems to be effective in treating EDS, cataplexy, and disrupted nocturnal sleep\(^5\). There is also the possibility that narcoleptics may be able to learn to modify their EEG responses and thus control narcoleptic symptoms without drugs\(^23\).

Yoss and Daly consider narcolepsy to be an hereditary
disorder, caused by an autosomal dominant gene. The evidence supporting this hypothesis comes from the observation of several families in which more than one member has narcolepsy. However, an early analysis of 250 to 300 reported cases of narcolepsy showed that approximately 81% of these cases were not familial, i.e., these narcoleptics did not have any relatives who had narcolepsy. When several cases of narcolepsy were reported in the same family, the number of cases ranged from 2 to 8, with an average of 2.8. These familial cases usually consisted of parents and children or of siblings. This is consistent with recent research, which indicated that narcolepsy occurs about 200 times more frequently (a rate of occurrence of 4% to 19.4%) in the immediate relatives of narcoleptics than in the general population. However, if Yoss and Daly are correct and narcolepsy is caused solely by an autosomal dominant gene, all reported cases of narcolepsy should cluster in familial groups, and the rate of occurrence of narcolepsy in the immediate relatives of narcoleptics should be approximately 50%. Therefore, one may conclude that, although there is probably a genetic predisposition to narcolepsy, the cause of narcolepsy is not genetic.

There is no evidence that narcolepsy is caused by tumors or lesions of the brain, severe head injuries, or viral encephalitis, and significant pathology in the central nervous system does not appear to be a factor in narcolepsy.

The observation that sleep onset REM periods occur in all cases of narcolepsy proper, when combined with other observations, leads to the conclusion that narcoleptic symptoms are manifestations of a disturbance in the system controlling REM sleep.
The typical duration of narcoleptic sleep attacks is the same as the typical duration of REM sleep episodes (5 to 30 minutes), and narcoleptics show a much greater incidence of REM sleep in afternoon naps than do those with independent narcolepsy. Catalepsy is strikingly like the motor inhibition that occurs with REM sleep, although it occurs during wakefulness. Sleep paralysis and hypnagogic hallucinations can be considered premature expressions of REM sleep's motor inhibition and dream imagery. Disrupted nocturnal sleep may also be a manifestation of a disturbance in the system controlling REM sleep. Many authorities on narcolepsy agree that narcoleptics suffer from some disturbance of REM sleep, but there is disagreement as to whether or not non-REM sleep is involved. Roth et al. have concluded that non-REM sleep disturbance, as well as REM sleep disturbance, occurs in most cases of narcolepsy.

The question of what neural centers are structurally or functionally abnormal and thus create the narcoleptic symptoms does not have a simple answer. Early research implicated noradrenaline circuits in the pontine reticular formation and the locus ceruleus as the neurological areas controlling REM sleep. A report of a patient with some symptoms similar to narcolepsy and severe neurological damage in the pontine tegmental nuclei may be taken as partial support for involvement of the pontine area in narcolepsy. However, many other areas of the brain influence the pontine reticular formation, and a dysfunction in any one of these may cause narcolepsy. Other researchers have suggested that some mechanism of wakefulness fails to inhibit the reticular formation areas controlling REM sleep.
lepsy has also been characterized as a disturbed circadian cycle or as a regression to infantile sleep patterns. Passouant has hypothesized that the narcoleptic either has regressed to the polyphasic sleep pattern of sleep onset REM periods found in infants or has not progressed beyond this pattern.

Jouvet has postulated that REM sleep is controlled by the caudal part of the pontine reticular formation and the locus ceruleus. These areas are activated by cholinergic mechanisms which in turn are activated by mechanisms in the caudal part of the raphe system. The raphe system controls non-REM sleep, while the pontine reticular formation controls REM sleep. The locus ceruleus seems to be involved in REM motor inhibition. Although other areas in the central nervous system are also part of the sleep system, the raphe system, the pontine reticular formation, and the locus ceruleus seem to be the central mechanisms. Peripheral areas of the sleep system include the septal-basal forebrain area, which "exerts descending facilitatory influences on the brainstem structures responsible for the various characteristics of paradoxical sleep" (Knauss, 1966, p. 1699). The basal forebrain also appears to influence the non-REM system.

It is my contention that a disturbance in this system is the cause of narcolepsy. More specifically, in normal humans there must be a functional link between the raphe system and the pontine reticular formation such that inactivity in the former inhibits activity in the latter and the locus ceruleus. In narcoleptics, this link does not function normally, i.e., does not always inhibit REM phenomena, although it sometimes does. Thus,
descending facilitatory influences from the septal-basal forebrain area are sometimes not counterbalanced by inhibitory influences from the raphe system, yielding REM phenomena during wakefulness (i.e., cataplexy, sleep paralysis, and hypnagogic hallucinations). In particular, the septal area (which is part of the limbic system and therefore involved in emotional functioning) is functionally connected to the locus ceruleus, so that strong emotion can trigger a disassociated state of REM motor inhibition, otherwise known as cataplexy. Furthermore, influences which in a normal person would facilitate the raphe system and thus inhibit the pontine reticular formation, producing non-REM sleep, have other effects in narcoleptics. These influences, while facilitating the raphe system, would also facilitate the pontine reticular formation, instead of inhibiting it. If there were simultaneous influences from the septal-basal area, a sleep-onset REM period or a REM nap would result. This dysfunction in the link between the raphe system and the pontine reticular formation could result from a failure of the structure to mature, from damage to the structure, from a genetic weakness of the structure, or from any combination of these.

Independent narcoleptics may or may not develop cataplexy. One study found that EDS onset preceded the first cataplectic attack by more than five years in about 28% of all cases of narcolepsy. Thus, some independent narcoleptics are actually narcoleptics whose full symptomatology has not yet developed. They may or may not show sleep onset REM periods. Other cases of independent narcolepsy actually be cases of apnea, as about two-thirds of all cases of apnea report excessive sleepiness.
In a recent study, no cases of independent narcolepsy without sleep onset REM periods were reported, indicating that, as a diagnosis, independent narcolepsy is no longer adequate.

Apnea may be either central, obstructive, or mixed, and affects an estimated 50,000 Americans, mostly male. All apnea victims actually stop breathing during their sleep as many as 400 to 900 times per night. In central apnea, movement of the diaphragm stops during sleep, while in obstructive apnea the muscles in the upper airway collapse. In either case, the apnea victim is usually unaware of his nocturnal cycle of falling asleep, ceasing to breathe, awakening, breathing, and falling asleep again. The most common sign of apnea is a history of extremely loud snoring. Other symptoms include high blood pressure, hypersomnia, insomnia, morning headaches, a feeling of disorientation and drunkenness upon awakening, and frequent body movements during sleep. Apnea may result in hypertension, hypoxia, cardiac abnormalities, chronic heart disease, and even death.

Apnea resulting in EDS may be differentiated from narcolepsy because narcoleptics awaken from daytime sleep episodes feeling refreshed, while the apnea victim awakens feeling unrefreshed. In most cases, the root cause of apnea is unknown, although Ondine's Curse (a form of central apnea) is caused by a tumor of the brain or spinal cord. Treatment for central apnea usually includes medication. Tricyclic antidepressants (including the imipramine compounds) are sometimes effective in central apnea, and carbamazepine has also been used with
some success. A recent development in the treatment of obstructive apnea is the tracheostomy, which places a valve in the patient's throat. This valve is closed during the day to permit normal functioning and opened at night to permit breathing. Hypnotic drug treatments for insomnia, which also depress the breathing mechanism, should not be used with apnea, as they can be fatal.

The Pickwickian syndrome is characterized by periodic apnea, as well as hypersomnia and obesity. The apnea episodes, usually 20 to 30 seconds long, are both preceded and followed by several deep breaths, with EEG arousal occurring just prior to the end of the episode. Increased muscle tone, myoclonic jerking in the arms, and cardiac acceleration may accompany the EEG arousal. The duration of these apnea episodes appears to be directly related to the depth of the sleep they occur in. Frequency of occurrence of the Pickwickian syndrome is unknown; however, Guilleminault and Dement found no cases of it in their study. Apparently, the symptoms of the Pickwickian syndrome are an exaggeration of a normal sleep phenomenon called Mayer's waves. Perhaps this exaggeration is triggered by obesity.

Chronic hypersomnia also appears to be an exaggeration of normal sleep phenomena. Hypersomniacs, unlike narcoleptics, do not find their sleep attacks irresistible, but they usually last much longer than the narcoleptic's sleep attacks, with a duration of several hours to a few days. The hypersomniac may exhibit extended nocturnal sleep in addition to (or in place of) daytime sleep attacks. Other symptoms include difficulty in awakening,
postdormital confusion, and increased heart and respiration rates during sleep\textsuperscript{2,6,37}. The hypersomniac has a normal, though pro-
longed, cyclic pattern of nocturnal sleep, and the proportions of sleep spent in each stage of sleep are also normal\textsuperscript{2,6,37}. Guilleminault and Dement report several cases of hypersomnia associated with increased levels of 5-hydroxyindoleacetic acid, and have successfully treated some of these cases with methysergide\textsuperscript{3}. Analeptic drugs are often used to treat hypersomnia\textsuperscript{6}. Hypersomnia seems to be associated with injuries or damage to the central nervous system or with a family history of hypersomnia\textsuperscript{6,37}. Hypersomnia has also been reported in patients with manic-depressive illnesses\textsuperscript{38}.

The Kleine-Levin syndrome, first reported in the 1920's, consists of periodic hypersomnia and excessive eating, associated with alterations in the victim's mental state, sometimes even schizophrenic or confusional states\textsuperscript{2,39}. Episodes are usually preceded by physiological stresses, such as illness, and many are associated with neurological or somatic abnormalities and/or elevated levels of sexual fantasy and behavior\textsuperscript{39}. Guilleminault and Dement found no cases of the Kleine-Levin syndrome in their study\textsuperscript{3}, but other sources report that most patients are adolescent boys\textsuperscript{39}. Onset of the disorder is often preceded by febrile illnesses such as leukocytosis or encephalitis\textsuperscript{40}. The Kleine-Levin syndrome is generally thought to result from a dysfunction in the thalamus or hypothalamus\textsuperscript{2}.

Nightmares, or dreams "in which fear is of such intense degree as to overwhelm the dreamer and force at least partial
awakening" (Boller et al., 1975, p. 1026) may be divided into three categories. Two of these three types are relatively normal phenomena of sleep, namely, REM anxiety dreams and those associated with sleep paralysis. The third type, called night terror, pavor nocturnus, or incubus, is associated with non-REM sleep; more specifically, with arousal from stage 3 or 4 sleep. These nightmares most often occur in the first non-REM period of the night, although they may also occur in the second. Night terrors are characterized by an alpha EEG pattern, loud screams, tachycardia, increased respiratory amplitude and rate, motility, and somnambulism, lasting only a few minutes, often coupled with subjective anxiety, a rapid return to sleep, and, usually, amnesia for most of the episode. When amnesia is incomplete, recalled themes are usually of dying, falling, choking, being crushed, abandoned, or enclosed, or fearing the aggressive actions of others. The amount of delta sleep prior to the night terror episode appears to be positively correlated with the intensity of the night terror. The etiology of night terrors is unknown, but they may be associated with epilepsy, temporal lobe lesions, certain types of hypnotic drug treatment, conflict, external crises, or head injury. Although some psychoanalysts have thought that the nightmare expressed internal conflict over incestuous impulses, there is little, if any, evidence that night terrors are associated with the buildup of instinctual forces. Night terrors are estimated to occur in three to six percent of all young children, and less frequently in adults. Children tend to eventually outgrow
night terrors, although removal of the adenoids, if they are enlarged, may eliminate the night terrors. Night terrors may be treated with imipramine, diazepam, systematic desensitization, or implosive therapy.

Insomnia, or "the inability to obtain adequate sleep" (Karacan and Williams, 1971, pp. 274-275) may stem from difficulty in getting to sleep, multiple awakenings throughout the night, early awakening in the morning, or any combination of these. Due to these disturbances of nocturnal sleep, the insomniac's daytime activities may be impaired. The total amount of sleep per night, however, is not a criterion for diagnosis of insomnia; Jones and Oswald report two men who normally sleep about three hours per night, and other researchers have found an elderly woman who sleeps only 67 minutes per night, but these people find their sleep adequate and thus are not insomniacs.

Insomnia may be divided into five types, based on causal factors. One type is physical insomnia, the result of physical symptoms preventing sleep. Apnea, arthritis, asthma, ulcers, kidney disease, heart disease, and migraines are examples of physical symptoms causing insomnia. A second type is physiologic insomnia, caused by jet lag, disruptions of the circadian cycle, tea, or coffee. A third type is psychologic insomnia, which is secondary to psychological disorders. The primary disorder associated with insomnia is depression. Comparative psychological testing of insomniacs and controls has found that scores measuring depression, fear, hypochondriacal concerns, somatic concerns, dependent rejection, obsessive worrying, and decreased control were elevated in insomniacs.
High levels of anxiety may or may not be associated with insomnia. The type of depression associated with insomnia changes with increasing age, and the proportion of insomniacs who are depressed increases. Yet another type of insomnia is iatrogenic, or caused by drugs such as amphetamines, antidepressants, or alcohol. Iatrogenic insomnia may be associated with either chronic use or withdrawal from hypnotic drugs.

The fifth type, idiopathic insomnia, consists of all cases of insomnia whose causes are unknown. In general, physiological arousal, including sexual arousal, is associated with any kind of insomnia.

In physical or physiological insomnia, the logical treatment is to alleviate or eliminate the cause of the insomnia. For iatrogenic insomnia, gradual and medically supervised withdrawal of the drug is recommended. Idiopathic and psychologic insomnia may be treated with drugs. The most effective drug for treatment of insomnia is flurazepam, 15-30 mg. at bedtime. This drug decreases both sleep latency and nocturnal awakenings, and insomniacs do not develop tolerances to flurazepam.

Flurazepam also does not disrupt REM sleep patterns. Diazepam, 10 mg. at bedtime, is also effective in reducing sleep latency and decreasing nocturnal awakenings. Some success has also been reported with the tricyclic antidepressants doxepin hydrochloride and desipramine hydrochloride.

Some hypnotic drugs have been found to be effective in reducing nocturnal awakenings and/or sleep latency over short periods of time, but lose effectiveness when used for extended
periods of time. These drugs include methaqualone, glutethimide, chloral hydrate, and secobarbital. Extended use of any hypnotic drug may lead to tolerance, increased use of the drug, and, eventually, iatrogenic insomnia.

In some cases, electrosleep treatments may be helpful in treating chronic insomnia; however, this does not always help and usually yields only short term effects. The use of an air-fluidized bed may temporarily reduce insomnia. The classical conditioning paradigm has also been used to treat insomnia. It has been suggested that biofeedback may be used to modify the EEG pattern and reduce insomnia. Attribution theory has also contributed possible treatments for insomnia.

Various forms of relaxation training have been generally found to be effective in treating insomnia. These include self-relaxation, metronome-conditioned relaxation, hypnotic relaxation, progressive relaxation, progressive relaxation with muscle-tension release, desensitization, and autogenic training. Relaxation training may be done in a group setting, and positive results were reported using tape-recorded instructions instead of a therapist.

Some authors recommend psychotherapy as a treatment for insomnia. The insomniac, however, often denies having any psychological problems and resists any form of psychiatric treatment, making such treatment difficult.

Although much research has been done in the field of sleep and sleep disorders in the past twenty-five years, much is yet unknown. What we do know is difficult to apply so that it may "make the widest possible contribution to the diagnosis, prog-
nosis, and treatment of patients" (Karacan et al., 1973, p. 77).

One of the reasons for this is that our basic questions about the function of sleep in general, and of the various sleep stages in particular, remain to be answered. And if we do not yet have answers to these questions, it is obvious that we cannot fully appreciate the significance of sleep disturbances. Furthermore, many of the sleep disturbances have yet to be completely described, much less examined from some more mechanistic point of view. (Martin et al., 1973, p. 75)

Much work remains to be done.
References


