symbolized in Fig. 2, B and C, is, however, a relatively minor one, and Fig. 2C should definitely not be considered as an allopatric model in the classical sense of the dumbbell diagram.

The concepts presented here are put forward in the hope that they may stimulate renewed interest in the cytogenetic processes involved in animal speciation. It is beginning to appear that there are more different kinds of mechanisms involved than was suspected a few years ago. Differences in modes of speciation are clearly related to differences in population dynamics and population structure; but they may also depend on differences in the genetic system.

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Sleep Disorders: Disorders of Arousal?

Enuresis, somnambulism, and nightmares occur in confusional states of arousal, not in "dreaming sleep."

Roger J. Broughton

Nocturnal enuresis, somnambulism, the sleep terror, and the nightmare are important social and medical problems. Their social impact can be appreciated from the extent to which they appear in the literature of various cultures (examples are Lady Macbeth's sleepwalking and the nightmares of Dante's Souls in Purgatory). They pose medical problems because of their frequency, their unresponsiveness to treatment, and their similarity, in certain respects, to other, more dangerous sleep disorders, especially nocturnal epileptic seizures. Recent studies have helped clarify their pathogenesis. Data are presented here which support the hypothesis that they occur independently of typical periods of dream activity, a view in direct contradiction to widespread long-standing assumptions. The results of recent investigations of their physiological and psychological mechanisms are also presented. Until these mechanisms are understood, treatment will remain empirical.

I begin with a summary of the essential features of each of the four attacks.

1) Nocturnal enuresis, or bed-wetting, is a common symptom in children and young adults. Incidences have been reported as follows: 10 to 15 percent for "nervous" children and 30 percent for institutionalized children (*1*); 1 percent for U.S. naval recruits who have been previously screened for the disorder (*2*); and 24 percent for naval recruits discharged on psychiatric grounds (*2*).

Typically the subject awakens to find himself in soaked bedclothes. An observer usually notes movement succeeded by several seconds of tranquillity, with apparent continuation of sleep, at which time the enuresis occurs. The subject is difficult to arouse, confused, disoriented even to the extent of denying that the bed is wet, and completely unable to recall any dreams.

When no organic cause (for example, pathology of the genitourinary system, epilepsy, and so on) is known, enuresis often is described as "idiopathic" or "essential." It has been interpreted as an expression of aggression, Oedipus fixation, or pathologically deep sleep (*3*). The unmotivated nature of the episode and the social ostracism incurred have been well described by George Orwell (*4*), who suffered from childhood enuresis:

> I knew that bed-wetting was (a) wicked and (b) outside my control. The second fact I was personally aware of, and the first I did not question. It was possible, therefore, to commit a sin without knowing you committed it, without wanting to commit it and without being able to avoid it.

2) In a typical attack of somnambulism or sleepwalking the individual sits up quietly, generally an hour or two after falling asleep, gets out of bed, and moves about in a confused and clumsy manner. Soon his behavior becomes more coordinated and complex. He may avoid objects, dust tables, go to the bathroom, or utter phrases which are usually incomprehensible. It is difficult to attract his attention. If left alone, he

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goes back to bed. A great deal of stimulation is required to awaken him. And when he is awakened he has little if any recollection of his sleepwalking activities and no recollection of dreaming. Nevertheless, these remarkable attacks are almost universally interpreted as an "acting out of dream activity" (*5*).

3) The sleep terror or pavor nocturnus attack of children is very different. The child abruptly sits up in bed and screams. He appears to be staring wide-eyed at some imaginary object; his face is covered with perspiration and his breathing is labored. Consoling stimuli have no effect. After the attack, dream recall is rare and usually fragmentary, and the dreams recalled resemble daytime fantasies. The child has no recollection of the episode the following morning.

These three disorders are often found in combination. All three may be manifested by a single subject at different times. Or all may appear in the family history; thus, 34 percent of enuretic naval recruits have a personal history, and 25 percent have a family history, of sleepwalking (*6*). Finally, they may be combined in a single attack, a sleep terror or an enuretic episode being followed, for example, by prolonged somnambulism.

The nightmare or incubus of adults has received particular attention over the centuries (*7*). During an attack the adult suddenly cries out in his sleep and shows all the signs of intense anxiety: sweating, a fixed facial expression, dilated pupils, difficulty in breathing. The essential symptoms of the nightmare are respiratory oppression, paralysis, and anxiety, usually occurring in that order. The anxiety is believed by many to be the most intense ever experienced by man (*7*). Well-structured dream activity is rare. [This is in contrast to the terrifying dream (see cover) which contains complex imagery, remains a sequence in time, and is not accompanied by respiratory oppression (*7*).] Reports of apparent dreaming are usually poor descriptions of oppressive situations, such as being locked up in a tomb, or having rocks piled on one's chest. The following morning the subject has absolutely no recollection of the attack. Subjects typically show intense daytime anxiety.

Writers of antiquity, the Middle Ages, and even the late 18th century believed that nightmares were caused by a nocturnal demon pressing upon the sleeper's chest. This is beautifully illustrated in Fig. 1, and in the etymology. The root word in the case of the English *nightmare,* the German *Nachtmar,* and the French *cauchmar* is the ancient Teutonic *mar*, meaning devil. Thus *nightmare* and *Nachtmar* mean nocturnal devil, and *cauchmar*, which derives also from *caucher*, an ancient French verb meaning "to press," means "pressing devil." The Italian *incubo* comes from the Latin *in* and *cubare,* signifying "to lie upon."

Freud (*8*) believed that both the incubus attack and the night terror of children express "repressed sexual impulses." But the four types of attack have more generally been interpreted as being due to dream activity, despite the infrequency of dream recall. And the amnesia has often been considered a result of "repression."

Recently, the "dream hypothesis" of the pathogenesis of the sleep disorders has become verifiable, as true dreaming has been shown to occur in association with a recurrent and distinct physiological state.

In this article I examine a number of questions. What are the known physiological correlates of dreaming? Do the sleep disorders occur when dreaming is taking place? If not, when do they occur? To what extent might some or all of the symptoms of these apparently "psychological" disorders be explicable by physiological factors associated with changes in the waking-sleeping cycles? I review investigations that have provided some answers to these questions and report new studies concerning others. What neurophysiological mechanisms underlie the common physiological correlates of the disorders? How relevant are they in explaining the symptoms of the attacks? To what extent, if any, is the attack pattern in a given individual determined by preceding physiological rather than psychological events? I conclude with a discussion of the possible relationships of the attacks to various types of mental activity.

Physiological Correlates of Dreaming

For over a decade it has been known that dreaming is statistically related to a distinct and cyclically recurring type of sleep characterized briefly by a low-voltage electroencephalogram lacking "sleep spindles" or K-complexes, and by bursts of rapid eye movements (*9, 10*). The incidence of dream recall (that is, the probability that a subject will recall having dreamed) following arousal from this state has been found to vary between 70 and 90 percent (*10, 11*). Since publication of these initial reports, other biological changes have been documented as characteristic of this state. Excellent reviews are available, by Dement, Jouvet, Snyder, Roffwarg, and others (*12, 13*).

The neurophysiological changes (*12, 13*) typifying rapid-eye-movement sleep include cerebral activation affecting at least the neocortex, specific sensory relay nuclei, and the so-called limbic system and require participation of brainstem structures, including a restricted pontine area (the nucleus reticularis pontis caudalis and nucleus coeruleus). The state has been called rapid-eye-movement (or REM) sleep, stage-I REM sleep, desynchronized sleep, paradoxical sleep, and rhombencephalic sleep.

After infancy, the sleep of normal subjects always begins with a state qualitatively different from REM sleep and divisible by electroencephalographic criteria into stages I to IV in the Dement and Kleitman classification (*10*). The first sign of decreasing vigilance is usually a slowing and diffusion of the alpha rhythm (stage I^{A1}) (*14, 15*), which becomes fragmented (stage I^{A2}) before being replaced by diffuse theta activity (stage I^{B}). Soon sharp waves with phase reversal around the vertex appear, followed by 12- to 14-cycle-per-second spindle activity occurring synchronously in both central regions (stage II), and by K-complexes representing cerebral potentials evoked by various stimuli.

As vigilance continues to diminish, background slow-wave delta activity (1 to 3 cycles per second) increases. When this is present in less than 50 percent of the tracing, the electroencephalogram is classified as stage III; when it is present in more than 50 percent, the classification is stage IV (*9, 14, 15*). Stages III and IV are often referred to together as "slow-wave sleep"; stages I to IV, as non-rapid-eye-movement or non-REM sleep. Such sleep is characterized also by a subsequent low incidence of dream recall, generally given as 5 to 10 percent (*9–11*), although higher figures have been reported.

The physiological correlates of non-REM sleep include decreased function

of the brainstem "reticular activating system" (resulting in the slow waves of cortical origin) and synchronization of unspecific thalamocortical connections (responsible for the sleep spindles). Non-REM sleep therefore has been described as synchronized or telencephalic sleep.

In the adult, a night of normal sleep consists of a series of cycles, each containing non-REM, then REM, sleep interspersed with occasional awakenings. The first cycle usually has much slow-wave activity and may lack a rapid-eye-movement stage. As the cycles pass, the proportion of REM sleep increases and that of non-REM sleep, especially slow-wave sleep, decreases. The average duration of the sleep cycles increases with age, from 45 to 55 minutes in the infant to 75 to 90 minutes in the young adult (*13*).

If the sleep disorders are related to, and perhaps even generated by, dream activity, they would be expected to occur mainly or exclusively during REM sleep. Sleep studies of the actual attacks therefore are of particular interest.

Sleep Studies of Sleep Disorders

Nocturnal enuresis has been studied more carefully than any of the other sleep disorders considered here. I therefore discuss some of the features common to all four in the context of this disorder.

In 1955 Ditman and Blinn (*15*) reported that micturition in young children coincided with electroencephalographic patterns of slow-wave sleep, and in young adults, with patterns resembling wakefulness. Pierce *et al.* (*16*) showed that nocturnal enuresis in children and in young naval recruits occurred during slow-wave sleep, was not associated with dreaming, and, in fact, actually retarded the appearance of the first rapid-eye-movement period. These observers nevertheless interpreted enuresis as a "dream equivalent"—that is, a phenomenon replacing the initial "dream period."

Bental then published a study of two teen-age brothers having almost nightly enuresis, both of whom passed into long (about 2.5-hour) "dissociated states" (states in which electroencephalograms typical of wakefulness were associated with behavioral sleep) before one wet his bed and the other awoke to void (*17*). Saint-Laurent *et al.* (*18*), in a pilot study at Gastaut's laboratories in Marseilles, demonstrated that, in epileptic children, nocturnal enuresis takes

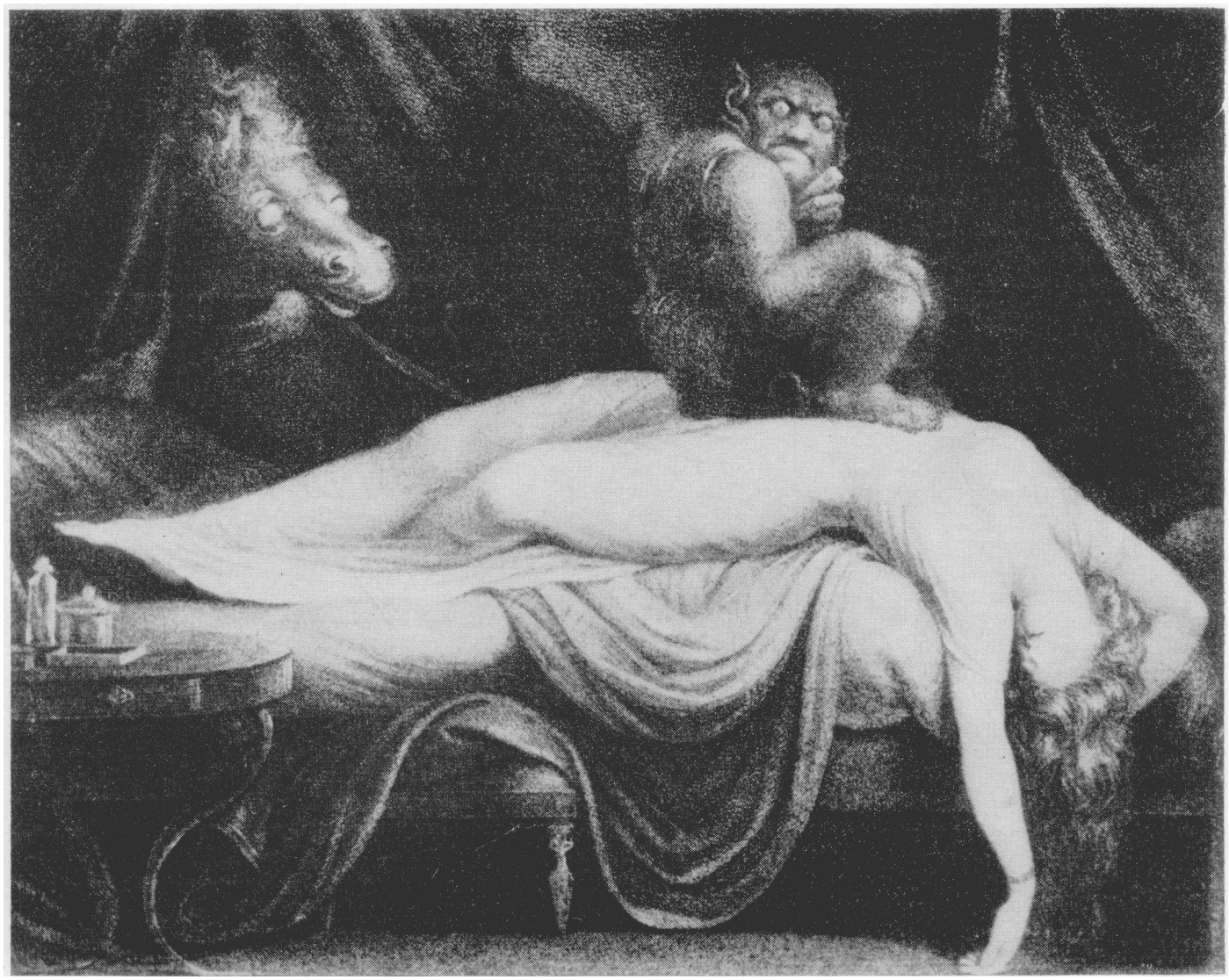


Fig. 1. "The Nightmare," painting by Johann Heinrich Füssli (Henri Fuseli), 1741–1825. The monster sitting on the patient's chest and the female horse leering in the background refer to the ancient Teutonic word *mar*, meaning devil, from which *nightmare* is derived, and the English word *mare*, which it suggests. [Courtesy of the Detroit Institute of Arts]

place during arousal from slow-wave sleep and is almost always nonepileptic in nature.

In order to clarify the contradictions in the literature, a program of enuresis research was initiated in 1962, under the supervision of Gastaut, in which more sophisticated recording techniques were employed: use of specially devised bikini-type shorts housing electrodes which were short-circuited by the first drops of urine at the meatus and, in later studies, continuous direct-current recording of intravesicular pressure by means of an indwelling catheter.

These studies (*14*, *19*, *20*) showed that enuresis in childhood and in adolescence almost always occurs during a partial or complete arousal from slow-wave sleep as a part of a more or less constant sequence of events which we called the "enuretic episode." The episode usually began with body movement associated with increased muscular tone, tachycardia, tachypnea or apnea, a decrease in skin resistance, and other signs of arousal. Micturition itself took place when electroencephalographic patterns were still those of stage-IV sleep or, much more frequently, had become those of stage III, II, or I or even wakefulness. The period of latency from the first electroencephalographic or polygraphic indication of arousal to the beginning of the micturition was found to increase more or less linearly with increase in vigilance, as judged by electroencephalographic criteria. Thus the average periods of latency from the initial sign of arousal during stage-IV sleep (bursts of higher-voltage delta-wave activity, tachycardia, or other sign) to micturition associated with an electroencephalogram typical of stage III, II, or I or wakefulness were 37, 68, 100, and 140 seconds, respectively.

Few enuretic episodes (three of 22) were related in any way to REM sleep. One occurred during passage from stage II into REM sleep, and two occurred after REM sleep had been well established. The "wet dreams" so often mentioned in the literature of enuresis were present *only* if the bedclothes were left wet and the subject awakened during subsequent REM sleep—a situation which allowed the incorporation of exteroceptive stimuli into dreaming.

The finding that enuresis occurs at some point along a *continuum* of increasing vigilance (arousal) from slow-wave sleep to a stage of lighter sleep or even wakefulness reconciled the divergent views on whether it is synchronous with electroencephalographic patterns of slow-wave sleep, light sleep, or even wakefulness. To a considerable extent this depends upon the recording method. That is, if the recorder is far from the meatus (it is often placed under the bed sheets), micturition which started in slow-wave sleep may not be signaled until considerably later in the arousal. More recent results (*21*) again indicate an age factor: in children nocturnal enuresis occurred during slow-wave sleep, but in adults it occurred 12 to 45 minutes after electroencephalographic patterns of non-REM sleep had been replaced by patterns of wakefulness associated with behavioral sleep (*22*). Again it did not take place during rapid-eye-movement periods, and there was no related dream recall.

In subsequent studies, sleepwalking (limited by the length of the electrode wires to a distance of about a meter) was recorded in 18 of 30 subjects said to be suffering almost nightly from these attacks. Like enuresis, it was found to be related to arousal from slow-wave sleep (*14*, *19*) except for one episode that occurred during transition from stage-II REM sleep (*14*, *19*, *23*). It was never related to dream recall. The association of sleepwalking with arousal from slow-wave sleep has been confirmed by Jacobson *et al.* (*24*).

Seven episodes of night terror or pavor nocturnus were recorded in seven children. These attacks occurred during sudden and intense arousal, again from slow-wave sleep. The electroencephalogram became that of wakefulness (*14*, *19*).

Finally, ten typical nightmare attacks were recorded during similar intense arousal from slow-wave sleep in five adults and one adolescent. A further nightmare in an adult also followed arousal from stage-IV sleep, but only after stage-I^{A} patterns had been recorded for 25 minutes. None of these attacks were associated with REM sleep or with detailed dreams (*14*, *19*). Apart from a case report (*25*) and incidental mention, in two instances, of apparent nightmare attacks during investigation of other phenomena (*26*), I have found no other reports of studies of either the night terror of children or the nightmare in the literature.

An interesting feature of the investigations of these sleep disorders was the finding that the incidence of the attacks during recording sessions was lower than the incidence when the subject was in his home milieu.

The Confusion of Awakening

The most striking aspect of our initial studies of these sleep disorders was the finding that *all* four took place during arousal from slow-wave sleep rather than in, or during arousal from, REM sleep. Furthermore, they were associated with body movement, autonomic activation, and the following six symptoms: (i) mental confusion and disorientation; (ii) automatic behavior; (iii) relative nonreactivity to external stimuli; (iv) poor response to efforts to provoke behavioral wakefulness (in the sense of full and lucid contact with the environment); (v) retrograde amnesia for many intercurrent events (an entire sleepwalking episode, the activity of the investigators, and so on); (vi) only fragmentary recall of apparent dreams, or none at all.

It was hypothesized that these six common symptoms might be attributable to the arousal process itself. We therefore investigated normal subjects and sleepwalkers during abrupt arousal from the various stages of sleep (*14*, *19*). We paid particular attention to comparisons between arousal from slow-wave sleep (stages III and IV) and from REM sleep. Telemetric recordings of the subject's electroencephalogram patterns and eye movements, filmed recordings of his behavior, and magnetic-tape recordings of his speech were made. Arousal was achieved by suddenly standing the subject up and questioning him.

In the case of REM sleep the threshold for arousal was high. Nevertheless, the subjects became lucid almost immediately and generally (70 percent) recalled dream activity. The threshold for arousal in slow awakening from slow-wave sleep was also high. But forced arousal from slow-wave sleep, in striking contrast to arousal from REM sleep, frequently produced periods of mental confusion with poorly coordinated automatic behavior, distant or slurred speech, relative nonreactivity, retrograde amnesia, and a lack of dream recall—in short, all the common symptoms described for the sleep attacks. This was true whether the forced arousal occurred during continuous slow-wave sleep or following the body movement that frequently "spontaneously" terminates such electroencephalographic patterns. The intensity and duration of confusion were greatest in children and in sleepwalkers. Kales *et al.* (*27*) recently reported similar findings.

Confusional episodes associated with awakening from an apparently "deep" sleep have been known for centuries; they are referred to in the German literature as *Schlaftrunkenheit* (*28*) and in the French literature as *l'ivresse du sommeil* (*29*). They were induced experimentally in man as early as 1897, by de Manacéine (*30*).

We went on to induce attacks of somnambulism experimentally in sleepwalkers or, sometimes, normal children by giving the subjects a basic biological drive for micturition (a full bladder produced by forced intake of water before retiring) and suddenly arousing them in stage-IV sleep (*14*, *19*). The subjects walked about (usually to a distant toilet), voided, and returned to bed. All had amnesia when forcibly awakened at the end of the episode. The electroencephalographic record made during these confusional periods generally showed a diffuse and rather high-amplitude alpha rhythm which reacted poorly or not at all to intense light stimuli, or else showed patterns of low-voltage delta and beta activity, lacking spindles. That forced arousal from slow-wave sleep can produce sleepwalking has been confirmed by others (*27*, *30a*).

The belief that arousal from stage-III or stage-IV sleep is the optimum, if not the necessary, condition for the confusional syndrome common to all the sleep disorders was the basis of recent investigations performed at the Montreal Neurological Institute.

Physiology of the Confusion of Awakening

If the confusional state following arousal from slow-wave sleep were, as our results implied, one of impaired cerebral responsiveness or of "functional deafferentation," this would at least partially explain *all* the common symptoms. The hypothesis is amenable, within certain limitations, to investigation by scalp recording of the brain's responses to controlled stimulation.

The visual evoked potential was studied during a presleep period of relaxed wakefulness following 20 to 30 minutes of dark-adaption with the eyes closed, during various stages of sleep, and during and following arousal from slow-wave sleep and REM sleep. No hypnotics were given, and the subject's pupils were not dilated by means of drugs. Ten subjects, aged 6 to 30 years, were studied. Only findings relevant to the present discussion are given here.

Stimulation was provided by means of a sound-attenuated Grass stroboscope (peak intensity, 10^6 lumens per square meter) placed 25 centimeters from the bridge of the nose. The electroencephalogram was amplified by four Grass P511 preamplifiers (50-percent attenuation at 0.3 cycle and 500 cycles per second), then stored on an Ampex 1200 tape recorder, and the responses ($N = 100$, occasionally 50) were summated by a Mnemotron CAT 400B digital computer and written out on an X–Y plotter. The reference electrode was on the earlobe. Superficial electromyograms of scalp muscles were recorded.

Arousal from slow-wave sleep and from REM sleep had markedly different effects on the visual evoked potential. Following one-fourth of the arousals from slow-wave sleep, the visual evoked potential at the standard occipital or vertex electrode initially contained apparent carry-over of components typical of slow-wave sleep despite the return of electroencephalographic patterns characteristic of the waking state. These persisting components were either a surface positivity peaking at 75 ± 15 milliseconds or a large negativity peaking at 250 ± 50 milliseconds, or both; they gave rise to a visual evoked potential intermediate between that of slow-wave sleep and that of wakefulness (Fig. 2). Even when there was no such carry-over, the visual evoked potential regularly showed decreased amplitudes and increased latencies of later components (Figs. 2 and 3), despite marked mydriasis, before reverting to the amplitudes and latencies of presleep wakefulness (*31*). Following arousal from REM sleep, on the other hand, the visual evoked potential was essentially that of presleep wakefulness (Fig. 3), and no changes similar to those in subjects awakened from non-REM sleep were observed.

The effects of the two types of arousal on the visual evoked potential paralleled the behavioral changes. After arousal from slow-wave sleep, mental confusion was associated with altered cerebral reactivity to visual (and probably also other) stimuli despite the return of an alpha rhythm characteristic of wakefulness. The nature of this alteration indicates either a continuation of responsiveness typical of non-REM sleep or changes in later visual-evoked-potential components suggestive of inhibition or occlusion of visual pathways. [Interestingly, Shakespeare, during Lady Macbeth's sleepwalking (*32*), has the Doctor of Physic say, "You see, her eyes are open," to which the Waiting-Gentlewoman responds, "Ay, but their sense is shut."] It should be noted that in experimental studies of the cat by Walsh and Cordeau (*33*), arousal from slow-wave sleep produced long periods of inhibition of the primary visual cortex, usually preceded by transient facilitation. Furthermore, such arousal is associated with reduced rates of background firing of unit cells in the visual cortex (*34*, *35*) and with a decrease in evoked unit activity (*35*).

Following arousal from REM sleep, however, cerebral reactivity to visual stimuli returns quite directly to that of the waking state and, not surprisingly, there is no mental confusion. It is in fact disputable whether the term *arousal* should be applied to awakenings from REM sleep, where, contrary to the case for awakening from non-REM sleep, the brain shifts from one complex pattern of intense activity to another and some cerebral functions might even be viewed as becoming less active (*12*, *13*).

The importance of these findings would appear to be as follows. If the altered cerebral responsiveness to exteroceptive stimuli in arousal from slow-wave sleep indeed represents a carry-over of non-REM sleep reactivity or some degree of inhibition or decrease in integration of sensory systems, confusion would certainly be expected. Confusion from any etiology (drug intoxication, epilepsy, head trauma, and so on) is associated with a "liberation" of behavioral automatisms, the organism reverting to, or adapting by means of, simpler and less conscious activity. The decreased behavioral reactivity to other environmental stimuli during the confusional episode can reasonably be compared to the altered cerebral reactivity to at least visual stimuli. And when one considers that the movement, increased tone, and autonomic phenomena are part of the arousal itself, it becomes evident that arousal from slow-wave sleep can produce all the symptoms common to the four major sleep disorders.

The postarousal state following slow-wave sleep would therefore appear to be the necessary, but not sufficient, condition for the occurrence of the confusional sleep disorders, and to explain the common symptons, although *not* the initiating or triggering mechanisms. But what determines the nature of the attacks?

Pathophysiology of the Attack Type

Continuous recordings of the bladder pressure of enuretic and normal children during sleep had previously shown quantitative differences between the two groups during and, interestingly, independently of the attack (*20*). Specifically, the enuretic group had an increase in the number and intensity of spontaneous and evoked primary detrusor contractions during non-REM sleep. Bladder contractions could be evoked by such stimuli as clicks, hand clapping, or other noises, even when these caused no observable electroencephalographic changes. Occasionally a "spontaneous" bladder contraction would precede a K-complex, indicating that these potentials can be evoked by interoceptive as well as exteroceptive stimuli. Any arousal during non-REM sleep provoked primary bladder contractions.

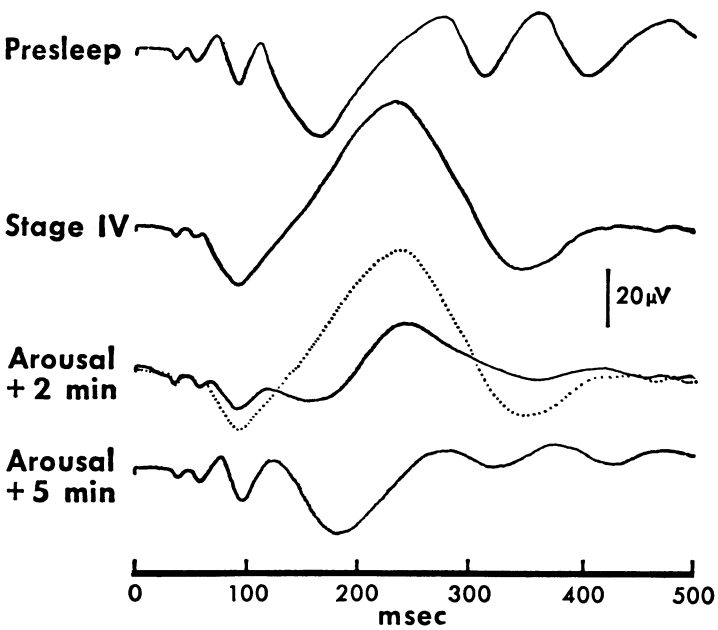
During stages III and IV, the spontaneous increases in bladder pressure were of the order of 3 to 15 cm-H_2O, but they sometimes reached 50 cm-H_2O or even more. The enuretic episode itself consisted of an increase in the frequency and magnitude of these contractions, culminating in micturition, at which time the peak pressure attained values up to 10 cm-H_2O before dropping to near-zero levels (*14, 20*). In REM sleep, however, there were few spontaneous bladder contractions, and, of course, there was no series of contractions leading to enuresis.

A few of the recordings of bladder pressure of enuretic subjects were, it turned out, made on nights when there were no enuretic episodes. Excessive bladder contractions erved throughout these nights. con- tractions never reached ude of those observed durin hen such episodes occurred, ever culminated in a series o ions such as generally precedes an retic episode.

Normal subjects, by compa on, showed very few primary detrus on- tractions during sleep, and the in ases in pressure were almost never above 25 to 30 cm-H_2O. Baseline pressu , moreover, were generally lower.

The differences in bladder physiology between enuretic and nonenuretic children observed *throughout* sleep suggested that other aspects of sleep, and especially of automatic function, might be affected. A statistical study of the sleep cycles and of cardiac and respiratory functions was therefore made from data accumulated in Marseilles. In this study, data for ten enuretic children on 13 nights on which enuretic episodes occurred were compared with data for ten nonenuretic children on 13 nights; the experimental and control groups were matched for age (6 to 16 years) and sex (six males, four females).

During the sleep of normal subjects the periods of slow-wave sleep (stages III and IV) are often terminated by movement, autonomic discharge, and other signs of arousal. (Direct passage from slow-wave REM sleep is also observed, but it is rare.) This recurrent phenomenon was designated the "slow-wave arousal episode." In the study, each enuretic episode was compared to the corresponding slow-wave arousal episode that terminated the identi-



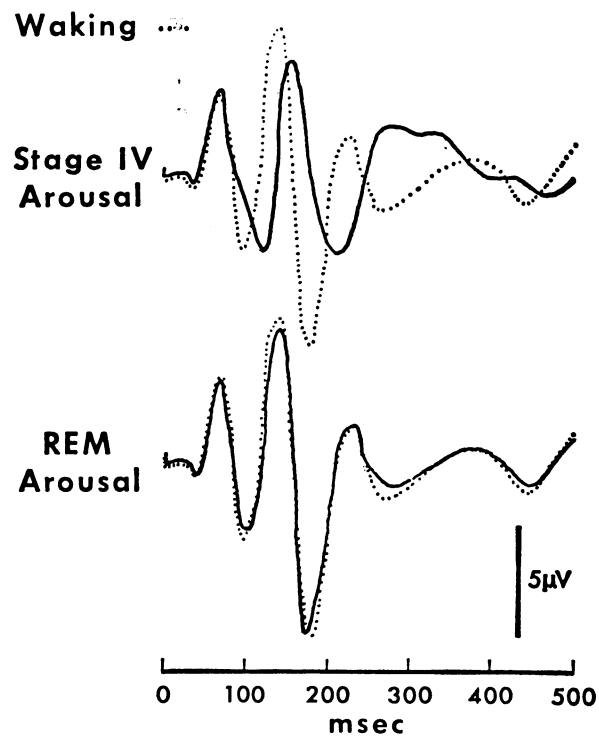


Fig. 2 (above). A comparison of the visual evoked potential during presleep wakefulness, stage-IV sleep, and the confusional period following arousal from stage-IV sleep for a 16-year-old subject. (The recording is from the occipital electrode to the earlobe; 100 responses were summated.) During slow-wave sleep the visual evoked potential shows consistent marked positivity at the occipital electrode (positivity is in the downward direction), with a peak latency of about 85 milliseconds and a later negativity at 250 milliseconds. The visual evoked potential 2 minutes after arousal in this case shows definite carry-over of components of the wave form of stage-IV sleep (superimposed dotted line), producing a potential intermediate between sleep and wakefulness despite the presence of an alpha rhythm in the electroencephalogram. The visual evoked potential 5 minutes after arousal has a wave form similar to that of wakefulness, although various components have slightly greater latency and lower amplitude than are typical of wakefulness for this subject. Fig. 3 (right). Visual evoked potential for a 27-year-old subject during a confusional period 4 minutes after arousal from slow-wave sleep (stage IV) and during a lucid state 4 minutes after arousal from REM sleep, relative to presleep wakefulness (dotted line). (The recording is from the occipital electrode to the earlobe; 100 responses were summated.) Note the decrease in amplitude and especially the increase in latencies of the later components of the potential after arousal from slow-wave sleep. The potential after REM sleep is within the variability of that of wakefulness.

Table 1. Comparison of relative intervals (group means) to different sleep stages and to the slow-wave arousal episode for enuretic patients (children) and matched control subjects. The means values for the two groups are essentially identical [$P > .05$ (Student's t-test)] The only aspect of sleep cycles approaching significant levels is the longer interval to initial REM sleep in enuretics ($P = .08$).

Subject	Interval to stage onset (minutes)					
	Recording onset–last I^A	Last I^A–stage II	Last I^A–stage III	Last I^A–stage IV	Last I^A–slow-wave arousal episode	Last I^A–1st REM
Enuretics	38.89	2.96	8.82	15.62	72.13	174.96
Controls	38.51	3.92	9.65	15.96	65.44	141.01

cal cycle for the matched control subject.

The intervals to the onset of the various stages of sleep and to the slow-wave arousal or the enuretic episode were measured (see Table 1). Also, the average rates and percentile variability (degree of arrhythmia) were determined for cardiac and respiratory function (Tables 2 and 3). Variability was calculated for each minute as the longest minus the shortest relevant interval (R-wave and peak inspiration, respectively) expressed as a percentage of the shortest interval. Average variability was the mean of the minute-by-minute results for the period concerned.

The data analyzed were restricted to pre-episode and related post-episode sleep and to later REM-sleep stages. Cycles which occurred much later in the morning were not included, due to the laborious nature of the calculations. No studies were made to determine whether there were differences in sleep patterns and physiological function of (i) normal children and (ii) enuretic children on nights when the latter experienced no enuretic attacks.

The results showed that the only aspect of sleep cycles (Table 1) which approached significance was the considerable increase in mean latency to the first REM-sleep period in enuretic subjects (13 minutes later in these subjects, $P = .08$). Rapid-eye-movement sleep always followed enuresis, usually after 5 to 15 minutes of further non-REM sleep.

The heart rate of enuretic children (Table 2) was significantly higher than that of normal children before sleep, in stage-IV sleep, and during the arousal episode. Five of the 13 studies of enuretics actually showed tachycardia after the subject had fallen asleep, and only three showed bradycardia. Because of this, further analysis was made, which indicated that the mean heart rate in stage-IV sleep of enuretics was not significantly different from the rate during wakefulness, whereas the heart rate of normals during such sleep was significantly lower (at the 2-percent level) than the rate during wakefulness. Moreover, the increase in heart rate during the arousal episode over the rate during stage-IV sleep was greater in enuretics ($P < .05$), despite the higher base line. The heart rate of enuretic children remained higher (Table 2) even during the 5-minute period following enuresis (usually stage-II sleep) and in subsequent REM sleep. Cardiac variability (arrhythmia) was correspondingly significantly lower in these children. Respiration (Table 3) showed a decrease in variability only in stage IV for the enuretic group. The data for the control group of normal children as a function of stages of sleep are qualitatively similar to data for adults reported by other workers (*36*, *37*).

This statistical investigation of the sleep of enuretic children confirmed the hypothesis suggested by the all-night studies of bladder function that autonomic changes occur throughout sleep, independent of the enuretic episode itself, and, moreover, are of the type that would predispose the subject to micturition. The enuretic episode in fact appears to represent simply an increase of these changes to a clinically overt level. And by interrupting the normal flow of sleep cycles, the episode retards but does not replace the first REM period. The enuretic episode therefore can in no way be considered a "dream equivalent," as Pierce suggested (*16*).

Patients who suffer from other sleep disorders, such as somnambulism, nightmares, and sleep terrors, manifest other constellations of physiological changes which differ from patterns in normal subjects, which occur both during the attack and at other times, and which have not yet been completely studied. Individuals subject to nightmares, for instance, have relative tachycardia during slow-wave sleep and hyperreactive heart rates during arousal episodes throughout the night. Sleepwalkers make many movements independent of the attack of somnambulism itself which are complex and gestural in nature and may occur in association with continuous electroencephalographic slow-wave activity (*14*, *19*, *30a*); they also have more marked and longer confusional episodes following forced awakening from slow-wave sleep (*14*, *19*, *27*).

Subjects with a classical sleep disorder therefore appear to manifest physiological changes which predispose them to attacks of a given type. These changes may be enhanced during some nights, culminating in an attack.

It should be noted that very similar attacks may occur in other subjects during intense arousal from virtually any background level of vigilance. Attacks during wakefulness consist of micturition during stress (stress incontinence); acute anxiety attacks with confusion, automatic behavior, or even fugues, and with more or less complete retrograde

Table 2. Comparison of heart rates (group means, in beats per minute) for enuretic patients (children) and matched control subjects at various stages before, during, and after the slow-wave arousal episode. Values in parentheses indicate variability (in percentage).

Subject	Presleep	Stage IV	Slow-wave arousal episode (peak)	Post-episode	REM sleep
Enuretics	94.13* (9.51)*	93.05† (11.77)†	128.10† (41.12)	86.75* (15.20)	92.20‡ (16.17)‡
Controls	84.47 (17.03)	72.96 (26.85)	101.67 (35.36)	74.69 (19.06)	78.77 (30.91)

* $P < .05$ (Student's t-test). † $P < .001$. ‡ $P < .01$. In all other cases, $P > .05$.

Table 3. Comparison of respiration rates (group means, in respirations per minute) for enuretic patients (children) and matched control subjects at various stages before and after the slow-wave arousal episode. Values in parentheses indicate variability (in percentage). All the comparisons but one are insignificant at the 5-percent level. No data are given for the period of slow-wave sleep arousal, because movement artifacts made respiratory measurements obtained at that time unreliable.

Subject	Presleep	Stage IV	Slow-wave arousal episode (peak)	Post-episode	REM sleep
Enuretics	21.21 (19.85)	19.76 (19.95)*	—	17.96 (22.77)	17.31 (28.90)
Controls	21.03 (22.39)	17.91 (28.55)	—	16.94 (22.06)	16.42 (39.16)

* $P < .01$ (Student's t-test).

amnesia; episodes of overt terror, rage, and so forth. Even when arousal is not as intense as it is during such attacks but is still supraoptimal, measurable degrees of impairment of performance are observed (*38*), which can be compared to the lowered psychomotor functioning following arousal from slow-wave sleep. I therefore emphasize the point that the sleep disorders are best considered *disorders of arousal.*

The conclusions, then, are that the attacks are essentially disorders of arousal; that the slow-wave sleep arousal episode is a normal cyclic event setting the stage for them; that preexisting constellations of physiological changes predispose a subject to a particular type of attack during the arousal episode; and that the postarousal confusional state explains the many common symptoms. These conclusions lead to a more fundamental and perhaps unanswerable question. What, during slow-wave sleep, actually provokes the *onset* of the attacks? This brings us back to consideration of the relationship of the disorders to preceding, and therefore possibly causative, mental activity.

Relation of Attacks to Mental Activity

There is no evidence whatsoever that REM sleep and its concomitant dreaming play any role in the genesis of these attacks. The various theories of the biological function of REM sleep (*13*, *39*) are therefore considered irrelevant.

Non-REM sleep itself has been shown to have an incidence of dream recall usually varying from 5 to 10 percent (*10*, *11*). But incidences as high as 54 percent have been reported (*40*). Important factors affecting the degree of recall appear to be the abruptness of awakening (*41*) and the strictness with which dreaming is defined (*42*). Kales *et al.* (*42*), in a recent study directed toward explaining these divergent figures, found only a 4-percent recall following awakenings from non-REM sleep in the first half of the night and the least recall following awakenings from stage-III and stage-IV sleep. Dreaming is therefore reported least often by normal subjects following periods when the four sleep disorders are typically most frequent. No studies have been made to determine whether such low percentages of recall for normals hold true for subjects with these sleep disorders.

There is some evidence that mental activity occurs which is as qualitatively different from dreaming as the physiological features of non-REM sleep are from those of REM sleep (*43*). Such mental activity lacks the panoramic hallucinatory features of true dreaming and appears to be much more closely related to daytime concerns and to the environmental situation. Subjects usually describe it as "thinking," and they may even deny having slept. Rechtschaffen *et al.* (*44*) have shown that this mental activity is much more common than true dreaming in episodes of talking during non-REM sleep, where the effects of arousal are much less of a contaminant than they are in full awakenings.

It is therefore conceivable that, in the deepest stages of non-REM sleep, when the risk of subsequent recall is minimal, protective barriers are lowered, allowing such mental activity to express emotional conflicts which otherwise are repressed, and that such activity leads to the attacks. But the interpretation of any data on mental activity that are gathered following arousal from non-REM sleep is in fact fraught with the difficulty of controlling a possible postsleep genesis of this activity during intermediate stages of impaired consciousness before the subject is fully awake.

The possibility must even be entertained that such disorders arise from a "psychological void," and that even the symptoms specific to the attack types are due to abnormally marked physiological responses during the recurrent slow-wave arousal episode (bladder contraction, producing enuresis; sensorium impairment, leading to somnambulism; cardiorespiratory changes, giving rise to terror).

In fact, a serious and unresolved paradox exists. Although mental activity of any one of a number of types (complex hallucinations, illusions, "thinking," and so on) might or might not exist in slow-wave sleep before the attacks, arousal would almost certainly erase any recollection of such activity (as it erases, indeed, most recollection of the attacks themselves) and thereby make detailed study of its nature, or even formal proof of its occurrence, impossible. We may be faced with a biological example of the indeterminacy principle. But certainly sleep and arousal (as evidenced by activation of memory traces in REM sleep, physiological amnesia of recent events following passage into non-REM sleep or following arousal from non-REM, but not REM, sleep) appear highly relevant to recent research (*45*) into memory consolidation and retrieval.

Summary

In summary, the classical sleep disorders of nocturnal enuresis, somnambulism, the nightmare, and the sleep terror occur preferentially during arousal from slow-wave sleep and are virtually never associated with the rapid-eye-movement dreaming state. Original data are reported here which indicate that physiological differences from normal subjects, of a type predisposing the individual to a particular attack pattern, are present *throughout* the night. The episode, at least in the case of enuresis, appears to be simply a reinforcement of these differences to a clinically overt level.

A number of features are common to all four sleep disorders. These had been shown previously to be attributable to the arousal itself. New data obtained by means of evoked potential techniques suggest that these common symptoms of the confusional period that follows non-REM sleep are related to alterations of cerebral reactivity, at least of the visual system.

The symptoms which distinguish the individual attack types (that is, micturition, prolonged confusional fugues, overt terror) appear to be based upon physiological changes present throughout sleep which are markedly accentuated during arousal from slow-wave sleep. These changes may in some way be related to diurnal psychic conflicts. But, to date, it has proved impossible to demonstrate potentially causal psychological activity, dreaming or other forms of mental activity, or even a psychological void in sleep just preceding the attacks.

The presence of all-night or even daytime predisposing physiological changes and the difficulty in obtaining any solid evidence of a preceding psychological cause explain, no doubt, why the results of efforts to cure the disorders at the moment of their occurrence (for example, by conditioning procedures in nocturnal enuresis) have been far from satisfactory.

I stress the points that the attacks are best considered disorders of arousal and that the slow-wave sleep arousal episode which sets the stage for these attacks is a normal cyclic event. Indeed it is the most intense recurrent arousal that an individual regularly experiences.

The most fruitful possibilities for future research would appear to be more detailed studies of those physiological changes that predispose individuals to certain types of attacks when they un-

dergo intense arousal or stress; the reversal of these changes by psychological or pharmacological means; and more refined investigations of the physiological and psychological characteristics of the process of cyclic arousal from non-REM sleep.

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Sleep Disorders: Disorders of Arousal?: Enuresis, somnambulism, and nightmares occur in confusional states of arousal, not in "dreaming sleep."

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