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Diagnostic Classification of Sleep and Arousal Disorders

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Introduction

Disturbed sleep and inadequate wakefulness are inestimable sources of human misery. Many individuals have had their chances for predictable social functioning, gratifying family life, and achievement in work wrecked by the symptoms of sleep and arousal disorders. The impetus for development of this Diagnostic Classification of Sleep and Arousal Disorders grew out of many years of requests from patients to medical practitioners for help with sleep problems, requests that for want of information traditionally fell upon deaf ears.

Medical causes of sleep disturbance do not customarily attract a great deal of interest or study, and the complaint of sleepiness is given only passing attention in most case presentations and records. The general physician is not alone, however, in turning aside or misunderstanding patient complaints about sleep. The workers who chose to enter the new research field of sleep physiology in the late 1950s and early 1960s by and large also believed themselves unable to interpret or help sleep symptoms, and all but a few stood apart from investigation and treatment of the clinical sleep disorders. However, this situation has recently undergone a dramatic change.

There can be little disagreement with the proposition that if a clinician is unable to diagnose or even suspect an existing sleep pathology, his treatment of the patient cannot be suitable. The practitioner, because of his customary uncertainty about the significance of sleep symptoms, has a tendency to treat sleep complaints as trifling or annoying. Characteristically, he both underestimates the etiology of sleep disorders and overtreats the symptoms with drugs.

Most doctors are astonished to discover that their accepted approach to exploring clinical problems is marked by an easily overlooked inconsistency, depending on the nature of the patient's complaint. Confronted with a traditional

medical problem, the doctor—whose training emphasizes a primary objective of uncovering the source of symptoms—typically initiates a thorough workup aimed at identifying underlying pathology. By means of examination and medical tests, he attempts to document a diagnosis or isolate one or more possibilities. Only if therapy may be specifically related to reversing or neutralizing pathological mechanisms does the physician feel prepared to prescribe treatment. In contrast, when dealing with a sleep disturbance, he acts in uncharacteristic fashion in terms of his usual procedural standards: typically without verifying the patient's description of symptoms—which may be grossly inaccurate—or undertaking a diagnostic protocol to discriminate the etiology of the symptoms, he will prescribe a drug for the symptoms. In this circumstance, the practitioner is in effect treating a condition he has not investigated with an agent whose pharmacokinetics, sites of action, and effects on mental and motor performance he likely does not know and whose actual impact on the sleep of the patient he lacks the means to determine with certainty. He generally also fails to arrange for follow-up observation. Accordingly, it must be acknowledged that the time-honored exhortation to physicians to "do no harm" has been trampled in the customary medical approach to the sleep disorders.

This situation—at the same time lamentable and understandable—results chiefly from ignorance and confusion, not from a disinclination on the part of medical practitioners to respond to the needs of their patients. If anything, the physician so wishes to provide relief that he tries to alleviate symptoms before he investigates their determinants.[1] Of course, the recipient of treatment also plays a role. The patient is commonly eager, often demanding, for help from the doctor, frequently preferring that assistance be undertaken solely at the symptom level.

Much is currently written about the attitudes and qualities of physicians, but one thing is clear—physicians are sensible pragmatists. Those who have had an opportunity to observe physicians in operation are repeatedly reassured that if practitioners are alerted to information they need to know in order to understand and treat their patients' illnesses more efficiently, and if the concepts are scientifically sound, meaningfully taught, and most of all salient to increased clinical effectiveness, the practitioners will lose little time in learning the information and applying it. There is no question that the findings now being made by sleep disorders specialists are beginning to be used in this manner by the general medical community.

It is therefore with humility that we recognize that a whole realm of intimately experienced human difficulties—the disturbances and disorders of sleep—has only desultorily played upon our attention for centuries. The recent advances in knowledge about sleep resemble the pattern of advance in other medical fields—it has waited until the very last decades to emerge. Be that as it may, these breakthroughs now make possible a coherent categorization of the sleep disorders and the construction of a rational system of diagnosis. We believe that the new diagnostic classification system contained in this volume will permit the sleep and arousal

[1] This is not uncommon with many medical problems; the difference in the sleep disturbances is that little consideration is ever given to efforts at diagnosis.

disorders to be finally "demystified" for the practitioner. The gain for the physician will be that rather than having to view sleep symptoms as inconsequential, obscure, or unfathomable phenomena, he can learn the elements that go into accurate diagnosis of sleep disorders. The physician will also be in a position to use findings of abnormal sleep patterns and symptoms to increase his awareness of the existence of related medical and psychiatric illnesses in his patients.

Antecedents of the Diagnostic Classification System

The mid- to late 1970s have witnessed a sharp expansion in the number and geographic spread of sleep disorder investigation-treatment facilities and a corresponding rise in the numbers of patients assessed. That the sleep disorders field has emerged as a true medical subspecialty—one in which research stands astride clinical work—is demonstrated also by the increase in practitioners working in the sleep disorders discipline, the inauguration of training programs and courses of instruction, the rapidly rising count of specific publications on clinical sleep problems, and the appearance of new scientific journals devoted to sleep studies.

The attention of sleep physiology research to clinical issues began slowly in the United States. In the 1960s a small group of investigators trained in electrophysiology began to be concerned with the nature of complaints about disturbed sleep, the physiological anatomy of sleeplessness, the patterns of excessive sleep and sleepiness, the pathophysiological disturbances linked to certain sleep stages and the process of arousal, and the effects of hypnotic agents. Since then a growing number of researchers have become clinician-investigators and have turned their efforts towards studying patients with sleep problems. Some began diagnosing and treating sleep pathologies in individual practice; others established hospital-based sleep disorders centers and clinics. Both sources have contributed heavily in recent years to the sharp increase in our data base for these conditions.

In 1972, at the tenth annual meeting of the Association for the Psychophysiological Study of Sleep (APSS), a workshop was organized on the "nosology and nomenclature of the sleep disorders." This panel marked the first, formal group attempt to share concepts concerning the "definition of the primary and secondary sleep disorders." The invitation to participate was sent to the thirteen international researchers who had a working interest in the area. The progress in our knowledge since this 1972 session has been remarkable, as indicated by the absence of sleep apnea as a condition in any of the diagnostic classification schemes submitted for discussion by the participants.

In 1975 two notable events occurred that spurred the growth of the sleep disorders discipline: one was the decisive Montpellier conference on narcolepsy, which established international conventions for the diagnosis of this condition; the other was the formation of the Association of Sleep Disorders Centers (ASDC), which has provided a continuing forum for scientific communication in the sleep disorders and has now set standards of certification for diagnostic–treatment units and clinical polysomnographers. During the February 1976 meeting, a "Nosology Committee" (later designated the Sleep Disorders Classification Committee) was appointed to begin the task of creating a diagnostic system for the sleep and arousal disorders, one that would include all conditions encountered clinically.

Classifications of the pathologies of sleep had been devised even in ancient times. The organization of these categorizations has varied widely. A number of recent, excellent classifications have been offered by individual investigators currently working in the field, to which the ASDC-APSS nosology is indebted.

Necessity for a Diagnostic Classification System

Optimization of understanding and investigative headway is only realized in a sphere of medical-scientific activity when colleagues share the same concepts about the constitution and terminology of presenting entities. They must also agree as to the lines of subdivision of clinical phenomena, how to group the conditions, and on common criteria of measurement. These agreements are *not* the end of knowledge in the field, rather somewhere near the beginning; they are simply a set of operating hypotheses and conventions, a working platform upon which to gain a foothold for efficient, future study.

The decision of the ASDC to develop a new classification system stems from the manifest need for an inclusive framework of sleep and arousal disorders, one that would be equal to ordering and recording the full spectrum of maladies presenting in general, as well as in specialized, clinical experience. In addition to inclusiveness, the classification and its contents, we hoped, would represent a true consensus among working specialists in the field as to the most heuristically valuable categorization of the disorders into major groupings. Another objective was that the characterizations of the diagnostic entities incorporate not only the best clinical descriptions in the scientific literature, but, when possible, also recent studies that throw light on the interrelationship of the character of the patient complaint, the clinical signs, and the invaluable physiological data furnished by polysomnographic recording.

A working assumption of the ASDC was that the nosology should be developed by a broad cross section of professionals. Varied inputs were sought from clinicians and clinician-investigators, many with strong roots in fundamental research, who served as committee members, contributors, and consultants. Moreover, all publications pertaining to the conditions were carefully reviewed and considered. Accordingly, both the overall structure of the classification system, as well as the material written on each disorder, represent amalgams of the best empirical data at hand and the shared judgments of experienced diagnosticians. Clearly, this classification system is a consequence, as well as a hopeful forerunner, of advances in our knowledge.

The value of a broad consensus is that accepted and, hopefully, the most valid, diagnostic conventions will now be standard in the evaluation of patients. Great constraints have existed on the inferences derived from needed case series investigations and other types of research owing to uncertainties and disagreements about diagnostic criteria. Only with concurrence in regard to essential diagnostic criteria can the status of clinical diagnosis, treatment, and future research in the sleep disorders be raised. Utilization of the nosology, we believe, will reduce the contamination in clinical studies introduced by data gathered from putatively identical, but in fact impure, diagnostic groupings. It is the faith of this enterprise

in nosology that intra- and interfacility research will increase and be more comparable across studies. In addition, since future study populations identified in accord with this nosological system should be more homogeneous, their responses to investigative manipulations and treatments may be expected to be more uniform. This will enhance the opportunities for research to acquire insights into the pathophysiology and etiology of the sleep disorders—the ultimate goal of this classification system and the final step before the sleep disorders can be eradicated.

Limitations of a New Sleep Disorders Nosology

It is well known that standardization of diagnostic criteria is not equivalent to diagnostic validity. The purpose of an inclusive and agreed-on set of diagnostic divisions is to establish concrete entities that may then be challenged and tested on validity grounds in future research. If standardized diagnostic criteria do not agree with the pathological features appearing in nature, nature will let us know. Diagnostic "variants" of certain conditions will soon reveal themselves. The appearance of many will suggest that the original diagnostic criteria were too narrow or aberrant. In short, a diagnostic classification system guarantees only that individuals who fit (and those who do not) are at least operationally specifiable and that research commentaries about groups of patients, categorized as within (or without) particular criteria, have a chance at consistent applicability to the defined populations.

As described above, the Sleep Disorders Classification Committee used the best evidence and judgments at its command to clarify and cluster diagnostic entities. But it must be remembered that a consensus arrangement of diagnoses simply establishes a focused synchronization of viewpoints, not validity. Diagnostic boundaries must continue to be appraised as research explores the mechanisms of disorders. It is to be hoped that many of the conditions proposed—and their diagnostic criteria—will prove valid, but we hold no brief for the permanence or organizational positioning of any diagnosis. The latter merely represent today's best judgments. Concepts of classification will surely change as new findings and improved conceptual frameworks evolve.

Undoubtedly, the wisest orientation to maintain towards the sleep disorders classification system is that it is a provisional, working construct. Many conditions will require revision or elimination; others will be added. The committee anticipates that more than a few of its judgments will have to be corrected. A case in point is the decision not to create two additional diagnostic entries: "primary" disorders of initiating and maintaining sleep (DIMS) in adolescence and in old age. Some evidence is accumulating that—because of biological factors—both periods are "at risk" for the development of disturbed sleep. The committee, however, concluded that the data were insufficient at this time to warrant these unique diagnoses. It judged other DIMS in the classification sufficient to explain the insomnias presenting in adolescence and in old age, as well as in other age groups. However, this decision may someday be seen as an instance in which the committee's choice lagged behind the evidence.

In other cases, our judgments may have led the evidence. For example, though the diagnoses of childhood onset DIMS (A.7) and advanced sleep phase (C.2.c) lack the specificity of more definitive diagnoses in the classification, we believe that, like Pirandello's six characters in search of an author, two pathological conditions—approximating the two diagnoses—are "out there" in nature, shadowing diagnostic actualization. Accordingly, it was decided to include these two diagnostic entities in the classification.

To restate our view, the committee believes that not all conditions described in the nosology can lay a sure claim to eventual verification, at least in their present form; some may be merely "holding operations." However, an approximate diagnosis is superior to no diagnosis, when one is needed. The approximate diagnosis also provides a lattice upon which additional findings can be hung, allowing for a process that often leads to eventual refinement of the diagnostic picture. In this way, the nosological system assists in its own upgrading at the same time that it permits better research into the biological underpinnings of the conditions.

Structure of the Classification System

The members of the classification committee were confronted with developing a meaningful arrangement of the diverse conditions that the field encompasses. Though the partitioning of the syndromes that was arrived at seems simple enough, the reasons for the ultimate divisions and diagnostic entries had to be thought through carefully.

A leading question was whether to segregate the disturbances of sleep and the excessive somnolence conditions at all. It is well known that abnormalities of either one affect the other; that is, insomnia and hypersomnia are both sleep–wake syndromes. The argument was made that they are, in fact, inseparable and that separate categorization amounts to an artificial distinction. Nevertheless, there are problems in not separating DIMS and disorders of excessive somnolence (DOES). One is that unless other criteria of categorization—which, as discussed below, have their own deficiencies—are employed, the classification consists only of a listing of disorders and not a nosological system. There is the benefit, however, of the one-list approach that each diagnostic entity need appear only once.

As to whether the DIMS and DOES conditions are conceptually separable, the committee believes that they are at least symptomatically separable. Classification into disturbed sleep and excessive somnolence conditions allows emphasis to be placed on the apparent sleep or wake period of initiation of symptoms. We also felt that the dangers of viewing DIMS and DOES syndromes too narrowly may be avoided by drafting the clinical descriptions of the conditions in the classification to span the 24 hour symptomatology and functioning of the patient.

The committee evaluated the partitioning of sleep problems also along the lines of primary and secondary disorders, functional and organic, and by means of polysomnographic variables. Primary versus secondary is a traditional discrimination and has the advantage of appearing to take etiology into consideration.

However, in virtually no sleep disorder are the true etiological antecedents of the clinical picture known. Hence, use of the term, primary, is either misapplied or confusing and is of little help in clinical evaluations. Similar arguments may be made in the case of partitioning along functional versus organic lines. It would certainly be ideal to construct a nosology of sleep disorders by etiology, as in infectious diseases. But, as mentioned, we are as yet nowhere near etiological understandings in our area of inquiry.

As to the use of the polysomnogram, the sleep research field underwent a paradigmatic change with the advent of multiple-channel electrophysiological recording and sleep-stage scoring. The committee would have been pleased to use the polysomnographic parameter, which has been so illuminating to our comprehension of the sleep disorders, as a tool in classification (similar to cardiological disease classification according to electrocardiographic findings). However, with the exception of its pattern in a few conditions, the polysomnogram is not sufficiently specific to be adequate as a diagnostic differentiator. It would also not be available in every clinical patient evaluation.

Turning to the classification schema that was developed, it is composed of four sections. (The prefatory notes to each section give additional information about features of these categories.) *Section A* classifies the disorders of initiating and maintaining wakefulness (DIMS), comprising types of disturbed and inadequate sleep; *Section B* covers the disorders of excessive somnolence (DOES), discussing types of excessive sleep and inappropriate sleepiness; *Section C* examines disorders of the sleep–wake schedule; and *Section D* describes the dysfunctions associated with sleep, sleep stages, or partial arousal, comprising abnormal behaviors and medical symptoms appearing in sleep. The classification system was designed to be reasonably comprehensive, though not encyclopedic, with respect to abnormal sleep. It emphasizes commonly observed conditions and omits very rare and unconfirmed diagnoses. The discussions of differential diagnosis are restricted to the major clinical situations requiring a discrimination of conditions.

Though it might be assumed from the classification outline that the DIMS and DOES divisions of the nosology were assigned by *type of pathology*, the ordering of diagnoses is actually according to *type of complaint*. For example, in obstructive sleep apnea, the polysomnogram informs us that the patient may be awakened more than a hundred times in the course of sleep. However, the individual complains only of sleepiness and has no recollection of the arousals. This syndrome—which by polysomnographic criteria should be viewed as a disorder of maintaining sleep, a DIMS—is rather classified as a DOES in the new classification system.

It would seem that categorizing by sign would be sufficiently regressive (akin to discussing certain pulmonary disorders under "the cough" or infections under "the fevers"), but categorization simply by symptom—especially since the patient may be unaware of important features of the condition—would appear to be wholly indefensible. What is the justification for this system?

The fact is that this seemingly antiquated approach has many benefits. We have determined that the greatest yield of data comes from the patient's expressed reasons for seeking help. The chief complaints of insomnia and notable daytime

sleepiness are found to predict reliably two nonoverlapping groupings of sleep pathologies.[2] Though this does not necessarily dictate a nosological partitioning along such lines, the committee decided that at the present stage of development of this field, a classification system that gives first priority to "listening to the patient" may be for now the most justifiable. It disposes to two superb logic-tree sets of inquiry about other attributes of the symptom picture, which offer a rational path for arriving at correct diagnosis and differential diagnosis. A symptom category classification—by providing such a means for the teaching of sleep disorders identification—has great potential at this time for upgrading diagnostic skills. Additional subcategorizations of the two symptom sets—e.g., sleep-onset DIMS versus early morning DIMS and obligatory napping versus sleepiness—are helpful in final specification of the diagnosis. (These types of symptom details are insufficient in themselves as a total basis for a classification system.)

This schema confesses rather than covers up its rudimentary nature. To summarize, it leaves the classification of DIMS and DOES as loose confederations of syndromes, whose only structural lines derive from empirical findings regarding the diagnostic utility of patient complaints. Clearly, the field is wide open for additional findings. It was our wish to let needed tests of concepts in the field and new data govern future subcategorizations, rather than have them imposed by intuition or dicta. We should, of course, be alert to any possibility. Whatever the current benefits of clustering by symptom, this system has its dangers—syphilis has taught us that several disparate symptom pictures may have a single etiology.

One other exception should be noted to the highlighting of symptoms in the classification system. Such an approach would have been counterproductive if applied to the sleep schedule disturbances, which have been isolated for separate attention in Section C of the nosology. Patients do not enter a clinic complaining of a sleep schedule or sleep–wake cycle disturbance. The currency of their presenting symptoms is, indeed, disturbed sleep, inappropriate somnolence, or both. However, with the sleep schedule disturbances, it is crucial that one not be confused by the apparent abnormalities of sleep or waking and that one learn how to collect the symptom data so as to identify, when present, the problem of *misplaced* (in circadian terms) sleep and waking.

Guidelines for the Coding of Diagnoses

Disorders may be coded according to two systems. First, the coding designations may be used that are inherent in the outline form of the ASDC-APSS classification system. For example, persistent psychophysiological DIMS (A.1.b) appear under A. Disorders of Initiating and Maintaining Sleep (DIMS), 1. psychophysiological, b. persistent. This method of coding has the advantage of

[2] Despite the fact that disturbed sleep (disorders of initiating and maintaining sleep, DIMS) leads to some daytime symptoms, these are not described by the patient in the same way as the "sleepiness" reported in disorders of excessive somnolence (DOES). If the clinician reserves the designation of the term sleepiness not for physical fatigue, lack of energy, poor concentration, etc., but for literal inability to remain awake even in stimulating circumstances, he will be in a position to differentiate the DIMS and DOES clusters.

utilizing the conceptual clusterings of the sleep disorders among and within the four major rubrics of the classification. It is likely to be used in facilities such as sleep disorders investigative units that plan to keep records on the flow of their clinical experience in terms of the whole spectrum of sleep disorders conditions. Second, a coding system has been devised that utilizes the coding numerals of the current *International Classification of Diseases, 9th revision, Clinical Modification* (ICD-9-CM) and which may be used concurrently or alternatively with the ASDC–APSS outline. ICD-9-CM is the official classification manual used by hospital record rooms in the United States. A section at the end of this classification system describes how ICD-9-CM codes may be used for the sleep disorders, that is, their correspondence to the ASDC-APSS rubrics. It is recommended that the ICD-9-CM designations be used in hospital records and in supplying diagnostic information to agencies and insurers.

The classification committee suggests that all clinically relevant and useful sleep disorder diagnoses be made, but parsimony is also to be encouraged. Those who have the responsibility for making diagnoses in general medical or psychiatric units should not feel themselves under an injunction to make *all* possible diagnoses relating to sleep. To illustrate, chronic schizophrenic patients commonly experience mild to moderate sleep disturbances. A formal diagnostic entry of a DIMS, separate from the diagnosis of schizophrenia, may be irrelevant (though notation of the symptom is not) unless a focus of interest exists in the facility concerning the status of sleep behavior in schizophrenia. In general, the decision to make the diagnosis of a sleep disturbance under such circumstances rests on grounds of the relationship of the symptom to the primary (non-sleep disorder) condition, the need for distinctiveness of the symptom apart from the major diagnosis, and the level of disability contributed by the symptom.

The issue of single versus multiple sleep disorders codings will be encountered with certain patients. In general, it is advisable to assign one diagnosis that can account for the entire spectrum of symptoms in the patient, but this is not always possible. *It is axiomatic that the condition, not the patient, is coded.* Though most patients seen for a sleep problem will yield only a single diagnosis for coding, some patients have multiple conditions requiring entry. For example, a few patients with narcolepsy will have sleep-related enuresis or sleepwalking as well.

There is one distinct situation in which multiple coding is required: when a parasomnia, in turn, disturbs sleep repeatedly, to the extent that the patient complains of insomnia. The parasomnia is first entered under section D, and then the DIMS induced by the parasomnia is coded under A.6.

In terms of the compatability of diagnoses, there may exist not only simultaneous presentation of conditions of different types, but also superimposition of conditions within the same group of disorders. To illustrate, a tense patient with a longstanding and stable difficulty in maintaining sleep, diagnosed as having persistent psychophysiological DIMS (A.1.b), hears of the unexpected death of his father. The patient now develops an inability to fall asleep in less than 2 hours for several nights, and, in addition, his repeated awakenings continue. The criteria for the diagnosis of transient and situational DIMS (A.1.a) are clearly present along with the persistent psychophysiological DIMS, and both should be coded (if relevant).

Though multiple diagnoses may be compatible, determining whether to use them is often complicated in clinical situations. The question of entering two diagnoses arises whenever features of one condition are present along with features of another. The decision to code one or more diagnoses rests, in part, on the determination of whether, in a specific case, the different clinical features are pathognomonic, or whether some of them may have less than diagnostic implication.

Sleep-related (nocturnal) myoclonus is an example of a condition that itself is an accepted cause of DIMS and DOES, but the leg jerks that typify this disorder also appear in conjunction with other conditions such as narcolepsy, sleep apnea, and drug abuse. It is not likely that the myoclonus—when it is observed with the other conditions—represents the myoclonus disorder so much as a secondary event. However, this question is controversial and is not at all well understood. Accordingly, the condition should be individually coded as a diagnosis whenever the criteria for its diagnosis are achieved (see A.5.a and B.5.a). The less-than-criteria episodes of leg twitching should also be noted, but not as a diagnostic entry. (Data such as these, if recorded, will ultimately help to unravel the role of sleep-related (nocturnal) myoclonus in symptom formation.)

A less complicated decision may be made in those cases in which an unambiguous case of sleep apnea DOES syndrome displays sleep-onset REM sleep. Though REM sleep at sleep onset is a diagnostic sign in narcolepsy, it can occur in other situations, e.g., heightened REM sleep "pressure" or unusual sleep scheduling, and is not an *absolute* indication of narcolepsy. The phenomenon has been described—without attribution of narcolepsy—in connection with other conditions. Accordingly, it would be inadvisable in this case to diagnose narcolepsy separately. However, the possibility of the existence of unique and independent conditions exists. If after resolution of the sleep apnea, repeat recordings continue to show sleep-onset REM periods in the presence of symptoms, the diagnosis of narcolepsy should be entertained.

Some Additional Points About the Nosology

The classification committee elected not to provide a listing of operational criteria for each diagnosis. However, the write-ups of the conditions contain the key diagnostic inclusion and exclusion points. It may be helpful to abstract, and more tightly codify, the diagnostic features of particular conditions for the purposes of some research projects.

The acronyms DIMS and DOES are generally used as nouns in the body of the classification but may also be found as adjectives, e.g., DIMS symptoms. This is admittedly infelicitous. At times it is resorted to in order to reduce use of the terms insomnia and hypersomnia—designations that have distorted meanings because they have lost all specificity and, worse, signify homogeneous symptoms in some people's minds. DIMS and DOES may eventually suffer the same fate, but at least when they are decoded, they remind us that a group of heterogeneous disorders is being referred to.

In an effort to acknowledge that people are increasingly unconfined to sleeping

at night and waking by day, and because many symptoms are actually related to sleep, not night, the terms "nocturnal" and "night" (e.g., night terrors) have been replaced by "sleep-related." Exceptions and transitional terms are used in certain cases to avoid confusion, e.g., sleep-related (nocturnal) myoclonus.

Similarly, modifiers of sleep—such as "nighttime" and "night"—used to indicate the major sleep period of the 24 hour day are no longer justifiable. With some exceptions, the appellations "major sleep period" and "major wake period" have been employed.

Whenever possible, psychiatric conditions are described with the designations employed by the forthcoming *Diagnostic and Statistical Manual, 3rd edition* (DSM III), of the American Psychiatric Association.

The outline of the classification system makes use of the phrase "not otherwise specified" (see, for example, A.8.c. and C.2.f.), which is to be understood in the sense of "unspecified." This entry is intended to leave place in the classification for (1) undiagnosed ("don't know") conditions and (2) additional—as yet undocumented (i.e., new)—conditions that may be described in the future.

A Note On the Bibliography of the Classification System

Literature references are grouped under the individual disorders in the classification. In addition, a number of excellent books and reviews are included under the general bibliography heading because they contribute carefully annotated summaries and syntheses of a broad range of syndromes.

The data contained in the descriptions of disorders that were written for this classification understandably derive from many sources. It should be emphasized that the data in the descriptions are not drawn solely from the citations given for each condition. These references were selected only to provide additional detail and to support certain key facts. The reader will note that a few conditions have no references listed simply because none are as yet available in print.

A related matter that warrants mention is our inability—in view of the need to limit the references for most of the conditions to two or three—to cite for many of the disorders the primary or "classical" sources. This is unfortunate, but it is not our intention to slight the critical, seminal efforts of the past. However, we were frequently forced to make the alternative choice, that is, to refer to more recent and comprehensive articles in the faith that they will not only inform the reader about the disorder, but also lead him to the excellent, earlier reports in the literature.

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* This entry is intended to leave place in the classification for both undiagnosed ("don't know") conditions and additional (as yet undocumented) conditions that may be described in the future.

A. DIMS: Disorders of Initiating and Maintaining Sleep (Insomnias)

The disorders of initiating and maintaining sleep (DIMS) are a heterogeneous group of conditions that are treated in this section because they are considered to be responsible for inducing disturbed sleep or diminished sleep. Even the disorders described in other medical, toxic, and environmental conditions (A.6) represent instigators, and not consequences, of DIMS. Accordingly, when a parasomnia (Section D) causes a symptomatic disturbance of sleep, it should be coded a second time in this section under A.6.

Past, long-term use of the blanket term "insomnia" should not blind us to the fact that although sleeplessness may be a final, common pathway, it has many sources, both psychological and somatic. In fact, there is little uniformity even in the symptom. Practitioners are well advised to elicit from the patient the quality as well as the quantity of an insomnia complaint. A great deal of information will be inexpensively obtained by means of a few questions that bring out the details of the symptom.

Though considerable overlap exists among underlying conditions in terms of the character of the sleep disturbances they present, it is nevertheless typical for a depressed individual to be able to fall asleep fairly quickly, but be unable to maintain the continuity of sleep, so that premature morning arousal is a routine symptom. On the other hand, inability to fall asleep is often the picture in acutely anxious, worried, or guilty individuals, whether they are essentially normal or in a psychotic state.

The mental state that appears to be the most powerful disrupter of the sleep-onset process is the internal arousal related to conscious and excessive "trying" to fall asleep (see A.1.b), whereas the early night inability to sleep in delayed sleep phase (c.2.b) represents a sleep–wake cycle shift and not a true abnormality of the sleeping process. A pattern of periodic awakenings all through sleep, devoid of early or late night emphasis, may connote a repeated REM sleep interruption DIMS (A.8.a).

The reader should be aware that certain possible causes of insomnia have not been included in the current compilation of DIMS because of insufficient data. However, their inclusion in revisions of the nosology will be reconsidered. Several persistent forms of psychophysiological arousability may exist that covertly cause DIMS. Individuals who are subject to such overactivity of the mechanisms of arousal might be the same individuals who report excessive sensitivity to caffeine. A "primary" insomnia of adolescence may fit conceptually into a psychophysiological arousal paradigm.

There may also be DIMS representing neurochemical or neurophysiological insufficiencies of the mechanisms of maintaining sleep. (A few patients with disturbed sleep seem to have low levels of cerebrospinal fluid 5-HIAA.) Mechanisms of this sort might underlie the association of insomnia and aging.

As discussed in the Introduction to the classification system, because of the

segregation of disorders according to the patient's subjective report of symptoms, it is impossible to avoid some duplication of syndromes in the classification system. The most notable example of this is sleep-related (nocturnal) myoclonus and "restless legs," which are included in this section (A.5) as well as in DOES (B.5). The syndrome can present with a complaint of disturbed sleep or as excessive drowsiness during waking hours, without memory of disturbed sleep. The same is true of the sleep apnea syndromes (A.4.a and B.4.a), though in sleep apnea the balance of central and obstructive components seems to govern the subjective symptomatology.

A. 1. Psychophysiological

a. Transient and Situational

Key words and phrases: transient reaction, situational, episodic, acute stress, emotional shock, acute emotional arousal, loss or threat, conflict, unfamiliar sleep environment, less than 3 weeks.

Essential features: *Transient and situational DIMS represents a brief period of sleep disturbance usually provoked by an acute emotional arousal or conflict caused by a loss or perceived threat.*

Depending mainly on age, premorbid personality, and precipitating events, this sleep disturbance may be characterized by any combination of sleep disruptions: difficulty in falling asleep, intermittent awakenings, and premature morning arousal. To be classified as transient DIMS, the sleep disturbance may not last longer than 3 weeks following termination of the precipitating event.

During serious situational reactions, emotional arousal is often unremitting, resulting in "round-the-clock" insomnia. Though substantial loss of sleep may have occurred, affected individuals often are not truly sleepy by day; rather, they are fatigued, achy, and "washed out," though unable to nap despite missed sleep. Transient and situational DIMS is the purest example of the effect of psychological factors on sleep. With resolution of the emotional reaction, sleep returns to normal.

Associated features and other information: Most transient insomnias are triggered by an emotional shock or immediate fear of threat to one's security such as the death of a close one, divorce, change of job, or an examination. Occasionally, a period of temporary sleeplessness is precipitated by intense positive emotions such as the exhilaration felt in response to a marriage proposal or the joyful anticipation of a child on Christmas Eve. In the case of either a distressing or happy reaction, the emotional components underlying the insomnia are manifest and also distinctly linked to a recent event. Perhaps the most common instance of a transient and situational DIMS is the poor sleep experienced in an unfamiliar sleep environment.

All people are subjected to situational insomnia at least several times in their lifetimes. Individuals who are insecure and have a low threshold to emotional arousal are most vulnerable.

Because transient and situational DIMS usually has a short course and is af-

filiated with a real precipitating event, it is not likely to be associated with the severely dysphoric thoughts characteristic of a clinical psychiatric syndrome. Serious medical or psychological complications are rare unless transient DIMS is superimposed on a preexisting medical or psychiatric condition.

Differential diagnosis: Sudden and sharp episodes of sleeplessness more likely represent transient and situational DIMS than any other condition. However, such an onset of sleep disturbance may retrospectively be viewed as the commencement of a persistent and serious psychiatric disturbance (see A.2.a–c), which at times is set in motion by disturbing real-life events.

It should be noted that the diagnosis of transient and situational DIMS may be properly employed even when a preexisting psychiatric condition exists but is not contributory to the insomnia. For example, a previously sound-sleeping, chronic schizophrenic patient may become insomniac for a short period on learning of the death of a parent. If the DIMS persists less than 3 weeks, it should be classified under transient and situational DIMS, not under other functional psychoses (A.2.c).

Temporary periods of insomnia also have their origins in medical, toxic, and environmental conditions (see A.6). If a rapidly developing sleeplessness has a physical source in pain, discomfort, metabolic disturbance, irritating physical treatments, or other somatic or environmental causes, it should be classified under A.6.

However, sleeplessness at the time of hospitalization is very commonly a reflection solely of apprehensiveness and removal from familiar (and "safe") home surroundings. Under such circumstances, and also in situations when DIMS accompanies medical illness without discomfort, or only develops when a serious diagnosis is revealed to a patient (without change in medical status), the insomnia should not be attributed to medical causes but rather considered as a transient and situational DIMS.

DIMS referable to emotional factors may also be inadvertently overlooked when somatic symptomatology is obvious and appears to account for the insomnia. A psychological component in the insomnia is suggested if, for example, complaints of insomnia become progressively more insistent at the same time that a patient is coping better with painful treatment or uncomfortable symptoms are improving.

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A. 1. b. Persistent

Key words and phrases: persistent, conditioned insomnia, negative reinforcement, internal arousal insomnia, chronic somatized tension–anxiety, chronic stress, psychophysiological arousal, greater than 3 weeks.

Essential features: *Persistent psychophysiological DIMS is a sleep-onset and intermediary sleep maintenance insomnia that develops as a result of the mutually reinforcing factors of chronic, somatized tension–anxiety and negative conditioning to sleep.*

In persistent DIMS the psychological elements responsible for the sleep disturbance are covert and not manifestly connected with recent events as in transient and situational DIMS (A.1.a). It not infrequently evolves from a situational insomnia and may be diagnosed when the difficulty in sleeping persists for more than 3 weeks after resolution of the precipitating event.

In middle-aged and older patients, who may fall asleep reasonably quickly due to exhaustion, persistent psychophysiological DIMS may present as prolonged awakenings toward the latter part of the sleep period. Patients may stay in bed excessively in an effort to resume sleep, and they frequently attempt to nap, generally without success. They are more fatigued than truly sleepy by day.

Though few systematic studies have been carried out, it has been observed clinically that resting muscle activity in many patients with persistent psychophysiological DIMS remains high in sleep. Pulse rate is often rapid, and alpha activity may infiltrate the record. Repetitive awakenings associated with worried thoughts and anxious dreams are frequent. When negative learning elements in the sleeplessness (see below) are relatively more dominant than "tension" factors, normal proportions of sleep stages 3/4 and rapid eye movement (REM) are maintained. Greater weighting of tension factors leads to frequent awakenings and reductions in these stages.

Associated features and additional information: It must be emphasized that persistent psychophysiological DIMS is a heterogeneous condition that is fueled from two separate but reciprocally amplifying sources of interference with sleep: *somatized tension-anxiety* and *conditioned association*. The two elements are additive in terms of arousal and persist owing to their interaction. Proportions of the two components vary among cases of this insomnia. An occasional patient will demonstrate only one factor, with little evidence that the other plays any role.

The chronic tension–anxiety sources of psychophysiological arousal in persistent DIMS are the prepotent elements in the majority of patients with this disorder. The anxiety is not generally sensed as such, but rather discharged into physiological channels. This somatized anxiety can present as restlessness, motor tension (often sustained at high levels during sleep), automatic hyperactivity, apprehensive expectation, ruminative thoughts, hypervigilance, and excessive visual scanning. The patient is not usually knowledgeable concerning the factors that maintain the sleeplessness. If aware of tension, the patient takes it for granted as part of his "makeup." Nevertheless, a clinical clue concerning tension factors in persistent DIMS is the experience of many patients that they sleep better at the start of the free period of their work or school week than on the night before their duties recommence. Accordingly, vacations away from home are times of relatively good sleep in patients with this disorder, not only because conditioned insomnia factors are diminished, as mentioned below, but because work and school stresses are also lowered.

The conditioned "reinforcers" of sleeplessness that contribute to persistent psychophysiological DIMS can be either external or internal. A *conditioned external factor* may develop from the continued association, during a temporary insomnia, of sleeplessness with objects and practices related to going to sleep, e.g., the furniture, aroma, and sounds in one's own bedroom, and the rituals before retiring. Patients in whom a conditioned association of bedroom and insomnia is significant report that they sleep better *away* from their bedrooms and usual routines—in a motel, on vacation, or when sitting on their living room couch or floor.

A conditioned internal factor is the apprehension about falling asleep that builds up in connection with unsuccessful and excessive efforts to sleep. A vicious circle develops: the more one strives to sleep, the less one can. Conscious efforts, or "trying to sleep," result in central nervous system (CNS) arousal. The existence of this factor in a persistent DIMS is suggested by a history of repeatedly falling asleep when not trying, as when reading, watching television or driving, but becoming fully aroused at the point of making an attempt to get to sleep.

Both external and internal conditioning are types of negative learning, corresponding to the formation of poor habits. Such learning may start insidiously in the course of a situational (A.1.a) or depression (A.2.b) DIMS. Because of this influence, the insomnia may persist long after the stress, conflict, or depression that triggered the original sleeplessness has disappeared. Extinction of the maladaptive sleep habits, once initiated, is prevented by occasional, naturally occurring nights of poor sleep.

Because the tensions underlying persistent psychophysiological DIMS are somatized and the conditioning elements self-perpetuating, the condition may continue unchecked for decades. It is observed in all age groups without regard to sex, including children, but may be distinguished from childhood-onset DIMS (A.7). In severe cases, the patient may be able to sleep only 3–5 hours a night, resulting in significant daytime impairment of functioning and mood and further exacerbation of tension and physical symptoms.

Persistent psychophysiological DIMS patients consider themselves "light sleepers." They often have multiple somatic complaints such as tension headaches, palpitations, and low back pain, leading to occasional abuse of alcohol, barbiturates, and minor tranquilizers. The patient may develop complications from such intakes and become subject to more pervasive medical or psychiatric disorders. Psychosocial and vocational stresses tend to exacerbate persistent psychophysiological DIMS. As pointed out above, the conditioning factors that perpetuate this disorder start in the course of insomnia from other causes. It is not certain whether the tension–anxiety components arise out of early, forgotten experiences or are in part constitutional.

Persistent psychophysiological DIMS is very common and probably accounts for a majority of all patients who present at sleep disorders clinics with insomnia due to psychological and psychiatric factors.

Differential diagnosis: In persistent psychological DIMS, the longstanding combination of negative, but occult, associations to sleep and emotional, but somatically embedded, sources of arousal renders the disorder some-

what difficult to clarify diagnostically. The picture is often additionally clouded by chronic use of hypnotics. Further, investigation of patients with persistent psychophysiological DIMS in the sleep laboratory is complicated by the tendency of many—though not all—patients to sleep unusually well there, as they do in other environments away from home. Though the laboratory may raise the apprehensions of certain patients with different types of DIMS and thereby worsen their sleeplessness, the conditioning factors, which are active at home and are specific to the insomnia in patients with persistent psychophysiological DIMS, are reduced in the sleep clinic. This may be due to several factors: poor generalization of conditioned elements to the laboratory setting, a lack of expectation by the patient that he can sleep well in the laboratory (leading to diminished "trying"), and diminished tension factors owing to the security inherent in the attendance of a trained staff member.

Though perplexing diagnostically when a professed poor sleeper sleeps well in the laboratory, it is helpful—and distinctive of this disorder—that the patient knows he has slept well after a good sleep night in the study unit. In contrast, patients with atypical polysomnographic features (A.8.b) and subjective DIMS complaints without objective findings (A.9.b) appear to sleep adequate amounts in the laboratory, yet do not feel they have enjoyed good quality sleep.

Persistent psychophysiological DIMS must be differentiated from many other DIMS conditions that share common features:

1. In DIMS with psychiatric disorders (A.2), the insomnia is associated with clinically definitive and classified psychiatric syndromes. Accordingly, patients complaining of insomnia that is symptomatically tied to a clear anxiety disorder (neurosis), panic "attacks," phobia, obsessive–compulsive disorder (neurosis), depressions, or acute schizophrenia should be classified under A.2. In these cases, the DIMS fluctuates directly with the waxing and waning of the other distressing symptoms of the condition. In persistent DIMS, anxiety and personality styles exist but are less syndromic than in A.2 conditions. Also in persistent DIMS, the insomnia is more fixed, though situational factors may affect its course to some degree. Early or mild organic mental syndromes (A.6) must be ruled out before the diagnosis of persistent psychophysiological DIMS is certain.

2. The externally conditioned features of persistent psychophysiological insomnia are separate from environmental or medical factors, A.6 (e.g., allergies, noise, pain), that lead to DIMS. Other conditions such as obstructive sleep apnea (A.4.a.) and paroxysmal nocturnal dyspnea (D.4.k.) should be considered along with psychophysiological DIMS when a patient claims that he falls asleep much easier when sitting up.

3. Patients whose main problem is "trying too hard to sleep," as noted, often fall asleep when not trying to sleep (e.g., when reading, listening to lectures, driving). This symptom is seen also in certain disorders of excessive somnolence (see section B). Persistent psychophysiological insomnia is usually distinguished by a long sleep latency (corresponding to an insistent complaint of sleep-onset insomnia) and by the absence of symptoms of sleep apnea (B.4) and narcolepsy (B.6), such as the obligatory nap pattern of the latter.

4. Patients with childhood-onset insomnia (A.7) may present a clinical picture

very similar to persistent psychophysiological DIMS. The distinction must be made on the basis of whether the insomnia is best explained by factors such as maladaptive learning, conditioning, and somatized anxiety. These features favor persistent psychological DIMS. In addition, in childhood-onset DIMS (A.7), the insomnia tends to be constant through a variety of emotional circumstances.

5. Sleep-related (nocturnal) myoclonus can mimic persistent psychophysiological DIMS insofar as sleep may seem to be disturbed at night without known cause, and the patient may be fatigued, mildly sleepy, and nap prone by day. Polysomnographic registration provides the information required to make a diagnosis.

6. Circadian rhythm disturbances are often associated with maladaptive sleep habits and may present with similar clinical descriptions of sleep-onset insomnia, sleeping late, and napping. Classification of a particular patient as persistent psychophysiological DIMS versus delayed sleep phase (C.2.b) may be exceedingly difficult. The constancy of the long sleep latency, the unbroken nature of sleep once achieved, the long length of sleep when allowed, and the consistently early onset, in combination, favor the latter condition.

7. Atypical polysomnographic features (e.g., spindle-REM sleep) are occasionally found in patients with persistent psychophysiological DIMS. In these cases, the aberrations in the sleep tracing are secondary to a long sleep-onset latency and broken sleep. In DIMS with atypical polysomnographic features (A.8.b), atypical features in the polysomnogram continue in the face of unbroken sleep. They disappear in persistent DIMS when sleep improves.

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A. 2. *associated with*

Psychiatric Disorders

a. Symptom and Personality Disorders

Key words and phrases: generalized anxiety, panic attacks, neurotic, phobias, hypochondriacal, obsessive–compulsive, personality disorder, "neurotic" personality, dissociative disorder.

Essential features: *DIMS associated with personality and symptom disorder is defined as a sleep-onset and intermediary sleep maintenance insomnia that is clearly related to the psychological and behavioral symptoms of the clinically well-known and classified, nonaffective, and nonpsychotic psychiatric dis-*

orders. These distinctive conditions—e.g., generalized anxiety, panic, and phobic disorders; hypochondriasis; obsessive–compulsive disorder; (various) personality disorders; and others (all in the formerly designated "neurotic" spectrum of psychiatric syndromes)—have traditionally been considered to be purely psychogenic and experiential in etiology.

Many, though not all, of the diverse symptoms in these disorders seem to represent an incompletely successful set of personality and behavioral attempts to control anxiety. Healthy adaptation to outer and inner needs or stress is insufficiently developed or has broken down in individuals with these symptomatic disorders. In many cases, it is clear that overt and unbridled anxiety—frequently and at times overwhelmingly experienced in manifest forms in anxiety and phobic attacks—or anxiety derivatives such as hypochondriacal concerns are responsible for the difficulties in falling asleep and the frequent awakenings (with and without anxiety dreams) that occur in the course of sleep. However, other psychiatric symptoms within this group of conditions, such as uncontrolled compulsive checking and ordering, or excessive pre-bedtime rituals may also interfere with the process of going to sleep.

Apart from the sleep-disturbing effects of anxiety and other manifest psychiatric symptoms, psychological conflict, whether conscious or unrecognized, and particularly if productive of guilt, may also have long-standing disruptive effects on sleep. Therapeutic resolution, or even enhanced understanding, of a conflict, such as one related to competitiveness, control, dependence–independence, or sexuality, is frequently observed to ameliorate an associated, psychogenic DIMS.

Associated features and additional information: Close parallels commonly exist between the severity and fluctuation patterns of the psychiatric symptoms and of the insomnia. The course of the DIMS may be chronic unless the anxiety or other distressing symptoms of these disorders are ameliorated.

Patients may present between adolescence and middle age with DIMS related to symptom and personality disorders. The sleep disturbance associated with this group of psychiatric conditions is not as prevalent as persistent psychophysiological DIMS (A.1.b) or DIMS associated with affective disorders (A.2.b). Gender ratio slightly favors females across the whole group of disorders, though in certain conditions such as Briquet's syndrome, the proportion of females is much higher.

Differential diagnosis: This insomnia cannot be easily differentiated on polysomnographic grounds from situational insomnia (A.1.a), persistent psychophysiological DIMS (A.1.b), DIMS with psychosis (A.2.c), and from the insomnia connected with various medical and environmental factors (A.6). Sleep fragmentation and reductions in deep non–rapid-eye-movement (NREM; stage 3/4) and REM sleep are general to all these insomnias with minor variations. Accordingly, classification of a DIMS as associated with symptom and personality disorders must rest on evidence of longer than 3 weeks coherence between the sleep disturbance and an unequivocal symptom of one of the conditions in this psychiatric category.

In contrast, situational DIMS (A.1.a) generally occurs against a background of relatively normal mental functioning (but may also occur in psychiatric disturbance without DIMS), and is limited to a duration of 3 weeks (see differential diagnosis in A.1.a). In the case of a chronically anxiety-ridden patient with a related DIMS, whose sleep disturbance increases after an event or experience *that makes the anxiety worse,* the diagnosis of DIMS with symptom and personality

disorder remains adequate without the addition of a diagnosis of situational DIMS (A.1.a).

Difficulties may be expected in some cases requiring distinction between DIMS with symptom and personality disorders and persistent psychophysiological DIMS (A.1.b), in which common features exist. For example, an insomniac person with compulsive symptoms may also have considerable somatized anxiety. In another individual, evidence may be uncovered that to some extent negative "learning" in regard to sleep (a feature of persistent psychophysiological DIMS) is operative in addition to the "neurotic" symptoms. Under such circumstances, if the symptoms are sufficient to make a categorical psychiatric diagnosis, the DIMS diagnosis favors symptom and personality disorders on the premise that psychophysiological arousal features are also part of such conditions.

DIMS associated with affective disorders (A.2.b) is distinguished clinically by the major affective symptoms (i.e., depression or mania/hypomania) and by premature morning arousal and short REM sleep latency.

Patients whose DIMS take the form of repeated REM sleep interruptions should be classed as A.8.a. However, the great majority of patients with manifest anxiety do not restrict their sleep interruptions to REM sleep. Patients with symptom and personality syndromes may regularly report dream anxiety attacks (D.4.a). On the other hand, few patients who have nightmares lose a sizable quantity of sleep.

The level of use and abuse of psychotrophic agents by patients with symptom and personality DIMS must be taken into account in the investigation of their insomnia. The sleep disturbance may have more relationship to drug ingestion or withdrawal (A.3) than to psychiatric status.

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A. 2. b. Affective Disorders

Key words and phrases: major depression, mania and hypomania, bipolar disorder, manic–depressive illness, primary and secondary depression, psychotic depression, suicide, "neurotic" depression, depressed mood, "masked" depression, sleep-onset insomnia (in manic state), sleep maintenance insomnia or sleep continuity disturbance (in depression), "early morning" (premature) arousal, shortened REM sleep latency.

Essential and associated features: *DIMS associated with affective disorders comprises two types of severe insomnias, which are characteristic of the disorders: in depression, the ability to fall asleep, but sleep maintenance disturbance, "early morning" (premature) arousals, and shortened REM sleep latency; in mania, sleep-onset insomnia and short sleep. (Bipolar depression may be associated with excessive sleep, see B.2.a.)*

The major affective disorders are divided into *major depressive* and *bipolar* (either depressive or manic–hypomanic episodes or both) *disorders.*[1] Both subtypes include patients with episodes of severity reaching psychotic proportions.

[1] Italicized terms indicate DSM III diagnoses as well as other accepted clinical designations.

Another group within the affective disorders is *secondary depression,* which is defined as depression occurring in connection with other psychiatric conditions or medical–neurological syndromes. DIMS associated with less severe depressions (formerly, reactive or neurotic depression) and depressed mood reactions are also included in this section, because the DIMS seen with them follows the form of the DIMS related to the major primary and secondary depressions.

The chief feature of the affective disorders grouping is the major and persistent disturbance in mood—either of a depressive or manic nature—that is evident. Depression and mania are usually recurrent disorders and may be incapacitating syndromes. They may be treated successfully with full return to the premorbid level of functioning. Although such episodes may develop within the context of a life stress, many—even in the same person—have no obvious precipitating factors.

The sleep disturbances connected with these psychiatric conditions can involve differing degrees of wakefulness occurring in the course of sleep. The severity of the DIMS is correlated with the severity of the affective pathology in psychotic depression. Most depressed patients subjectively complain of nocturnal restlessness and tired feelings, whereas patients with hypomania or mania, despite abbreviated sleep, awaken feeling refreshed. Despite the "achy," "washed out" feelings associated with the profound insomnia, most depressed patients of the unipolar type are not truly sleepy during the day. This probably relates to their high level of psychophysiological arousal, making it difficult for them to sleep easily at any time. In general, the more elderly the patient, the greater the sleep discontinuity in the second half of the night.

The sleep disturbance usually seen in *non-bipolar depressions* is most well known. (Until recently, it was mistakenly considered to be the classical sleep pattern in all depressions.) Whereas some difficulty falling asleep is not unusual, the most characteristic feature of the DIMS is the repeated awakenings, leading to the so-called "early morning" (or premature) awakening that foreshortens sleep. Waking up too early and not being able to return to sleep is the cardinal complaint of the unipolar depressed patient. The sleep tracings show a short REM sleep latency as well as reduced stages 3 and 4 NREM sleep in comparison to the patient's normal base-line amounts. (The short REM sleep latency is accepted as a biological marker of depression.) Sleep-onset disturbances indicate a component of agitation associated with depression.

It is controversial whether the high stage REM percents often seen in depression constitute a primary feature of depressive sleep or a compensation for REM sleep deprivation occurring early in the depression. Many acute and agitated depressions (including psychotic depression) are characterized by an extremely low REM sleep percentage. The characteristic DIMS associated with depression is frequently a very early sign of the affective change, often beginning before the clinical depression has become clearly established. This DIMS is also seen in what some clinicians refer to as "*masked*" *depression*—existence of a depression, though disguised by denial, coping attempts, and somatic symptoms.

The same pattern of sleep disturbance, though less marked, is found not only in middle-aged and elderly depressed people, but also to some extent in *depressed*

children and in *young adults with depression and depressed mood reactions.*

Bipolar depressions, in contrast to the non-bipolar type, frequently are associated with hypersomnia (see B.2.a). Nevertheless, these depressed patients also have the typical polysomnographic features of short REM sleep latency and reduced stages 3 and 4 sleep. Even though the patients sleep more than usual, they complain that they wake up unrefreshed.

A different sleep pattern is usually present in patients with *secondary depression,* whether it is part of a medical illness or preexisting psychiatric illness other than a primary affective disorder. These patients usually also show awakenings and sleep continuity disturbances, but they generally do not show as great a shortening of REM sleep latency. In addition, patients with secondary depression associated with medical illness continue to show reduced REM sleep.

The *hypomanic* or *manic patient* is distinguished by a profound inability to fall asleep. A severe manic, once asleep, will reawaken and be refreshed after only 2–4 hours of sleep. In manic conditions, REM sleep latency is also short, and there is often a decrease in stages 3/4. Sleep disturbances are of varying degrees in patients showing *hypomania* or even *cyclothymia*. Patients may alternate between DIMS with mania and DOES with depression when an alternating bipolar disorder is the underlying condition.

Differential diagnosis: In depressed patients, owing to the high incidence of somatic symptomatology (including disturbances in appetite, concentration, and fatigue), sleep disorders such as sleep-induced respiratory impairment (A.4) and sleep-related (nocturnal) myoclonus (A.5) must be ruled out. In both manic and depressed patients, the effects of drug and alcohol use must be considered in the differential diagnosis of the DIMS. These agents may alter the clinical presentation, particularly in regard to the type and degree of sleep discontinuity (see A.3).

Lability of affect, i.e., depressed and excitable phases, may be an early sign in presenile and senile dementia and in other organic mental syndromes (A.6) that are associated with sleep disturbances.

Depressed patients may have an insomnia related to repetitive awakenings occurring at intervals of 60–90 minutes and frequently associated with dreams. This unique sleep disturbance pattern should be classified under DIMS with repeated REM sleep interruptions (A.8.a).

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A. 2. c. Other Functional Psychoses

Key words and phrases: schizophrenia, psychotic decompensation, mental disorganization, delusions, hallucinations, anxiety, agitation, schizoaffective disorder.

Essential features: *DIMS associated with other functional (generally schizophrenic) psychoses consists usually of a severe sleep-onset insomnia and often sleep continuity difficulties, which develop in connection with acute psychotic decompensations or exacerbations. The components that usually underlie the sleep disturbance in the patients are anxiety, fear, suspiciousness, urgency of thought, and extreme guilt. Though usually reduced in intensity, DIMS often remains a significant problem in chronic psychosis, particularly when high levels of anxiety persist.*

Associative features and other information: The subjective sleep complaints of patients with functional psychoses (other than the depressive psychoses) usually revolve about difficulties in *falling* asleep. This often progresses to an almost total insomnia in acute cases that is associated with a partial or complete inversion of the day–night sleep cycle. With progression of mental decompensation, falling asleep is increasingly difficult. Extreme anxiety and preoccupation with delusional and hallucinatory phenomena result in motor hyperactivity. Until exhaustion supervenes, the agitated patient may be awake through the first part of the night. Once the patient is able to settle into sleep he may have 7 or 8 hours, though usually less, arising around midday. As the sleep latency becomes longer with progression of the psychosis, arising times correspondingly shift further into the afternoon. Sleep may be possible, but it is also subject to fragmentation by frequent arousals. Accordingly, given both the difficulties in initiating and maintaining sleep, partial (or total) sleep loss is commonly observed in eruptive functional psychotic conditions.

Acute schizophrenia and schizoaffective patients typically show reduced amounts of sleep stage 3/4 and, consistently, a reduced amount of REM sleep, especially during the period of greatest symptomatology. Many chronic schizophrenic patients complain of life-long poor sleep even when psychotic symptoms are low grade. However, other chronic schizophrenic, or remitted acute schizophrenic patients, show normal sleep parameters.

Differential diagnosis: When fragmentation of sleep continuity has been extensive and specific sleep stage losses considerable, the polysomnographic tracing may show many abnormal conjunctions of electrophysiological features and a lack of clarity in the demarcations of REM and NREM sleep periods. This should be differentiated from the sleep tracings in DIMS with atypical polysomnographic features (A.8.b). The most common conditions underlying DIMS to consider in relation to DIMS with other functional psychoses are drug-induced psychosis (A.6) and affective psychosis (A.2.b). Dementia and acute neurological disorders such as encephalitis must also be ruled out (see A.6).

Additional considerations concerning differential diagnosis relative to DIMS associated with other functional psychoses may be found in the sections on differential diagnosis in transient and situational (A.1.a) and persistent

psychophysiological (A.1.b) DIMS, and in symptom and personality disorders (A.2.a) and affective disorders (A.2.b) DIMS.

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A. 3. *associated with*

Use of Drugs and Alcohol

a. Tolerance to or Withdrawal from CNS Depressants

Key words and phrases: sleeping pills, hypnotics, sedatives, tranquilizers, bedtime use of alcohol, tolerance, dependence, withdrawal, overdosage, drug abuse, drug-dependency insomnia, REM sleep rebound, compensatory effect of agent.

Essential features: *DIMS is associated with tolerance to or withdrawal from CNS depressants. With sustained use of such agents—usually undertaken to combat DIMS arising from a different source—tolerance increases and the depressants lose their sleep-inducing effects, often leading to an increase in dosage. In addition, unrecognized periods of partial and relative withdrawal occur, which contribute to the development of the secondary, drug-related DIMS. With sudden discontinuance of drug, severe sleeplessness supervenes, often accompanied by the general features of a drug withdrawal syndrome.*

For this condition to be diagnosed, the drug must have been in daily employ as a bedtime hypnotic agent for a minimum of 30 days. The classes of agents that are implicated in this syndrome include the barbiturates; nonbarbiturate sleep agents such as glutethamide, chloral hydrate, methaqualone, and ethchlorvinal; beverage ethanol; "over-the-counter" and prescription antihistamine sedatives; bromides; and the rapid-metabolizing benzodiazepines (also intermediary, slow-metabolizing benzodiazepines taken in high doses).

These drugs—with the exception of the benzodiazepines—lose their pharmacological effect on sleep within 2 weeks, leading some patients to raise the dose at intervals, thereby inducing the development of tolerance and physical and psychological dependence on the agents.

During the period of chronic use of a hypnotic agent, nocturnal sleep is marked by frequent awakenings, each lasting more than 5 minutes, some of which include periods out of bed. Sleep continuity problems are frequent during the second half of the night because the drug rapidly loses its effect after a few hours in a tolerant individual.

Progressive prolongation of sleep latency may also occur as the period of drug ingestion lengthens. Occasionally, nights of extended and reasonably sustained

sleep are intermingled with nights of very disturbed sleep. If the agent is omitted at bedtime, it is likely that several awake hours will elapse before sleep is possible.

Patients on heavy doses of CNS depressants have considerable apprehension about having to depend on drugs in order to sleep. They are additionally concerned that the initially beneficial effects of the drugs have diminished or disappeared, although the patients may continue to experience, during the day, residual drug effects such as sluggishness, poor coordination, ataxia, slurred speech, visuomotor problems, and late afternoon restlessness and nervousness.

Many of the patients also show a range of psychopathological symptoms, including depression with suicidal ideation. (Much of the emotional pathology may derive from the predrug psychological profile; see differential diagnosis, below.) The patients characteristically remain focused on questions concerning the efficacy of their drug, convinced that many of their daytime symptoms are related to how well they slept the previous night. Generally, the patient has seen many doctors, tried a variety of sleep-inducing compounds, and has prescriptions from several physicians.

Polysomnographic recordings in chronic sleeping pill takers, *on drug,* show a disruption of the sleep architecture: decreased sleep stages 3/4 and REM; increases in stages 1 and 2; fragmentation of NREM and REM sleep periods; loss of clear stage demarcations; frequent stage transitions; reduced sleep spindles, K complexes, delta waves, and REM sleep eye movements; and increased 14–18 Hz "pseudo spindles" and alpha and beta frequencies. [Individuals on substantial doses of barbiturates show characteristic fast frontal electroencephalographic (EEG) activity.]

During *gradual withdrawal,* under supervision, from habitually taken sleep drugs, many individuals begin to show improvement in subjective sleep and polysomnographic parameters. This finding indicates that a major component of the presenting problem with sleep may be the effects of a relative withdrawal state, and perhaps also a direct CNS action of the pharmacological compounds.

In view of the sleep findings during the periods of tolerance and relative withdrawal, the term "drug-dependency insomnia," which has been used to describe the disrupted sleep of habitual users of sleeping agents, seems justified. However, even with a notable improvement in sleep after reduction of drug, the individual may not return to a fully normal sleep pattern in the drug-free state.

During a period of rapid reduction or abrupt withdrawal from daily ingested hypnotic agents (not infrequently self-imposed by a concerned patient), sleep is almost completely disrupted. What little sleep is possible contains a high proportion of REM sleep marked by a heavy density of phasic activity. This results from a compensatory rebound of sleep stage REM, which had been chronically suppressed by the heavy doses of hypnotic. The patient experiences a profound sleep-onset and sleep-maintenance insomnia coupled with severe dream anxiety attacks (nightmares) during the episodes of REM sleep. The sudden withdrawal gives rise to daytime symptoms of the drug withdrawal syndrome (sometimes not seen for 2–4 days after the last intake of drug): nausea, muscle tension, aches, restlessness, and nervousness. Such disturbing round-the-clock symptoms, following sharp lowering of the dose of a drug, influence many patients to believe

that they *must* take hypnotic substances in order to sleep. During withdrawal, temporary sleep-related myoclonus may appear (see A.5.a).

Associated features and other information: Some patients seem to have a psychological dependence on sleep-inducing agents and do not have an apparent physiological tolerance. They have little or no reported sleep disturbance so long as they continue to take the drug nightly. However, if the hypnotic substance is abruptly withdrawn, disruption of sleep occurs in these patients, as well as in the drug-tolerant group described above.

In regard to factors that predispose to the excessive utilization of sedating agents, patients typically give a history of an earlier period(s) when they had many disturbed nights of sleep, which led to exhaustion by day and the seeking out of pharmacological assistance with sleep. Some individuals provide a predrug history of leg jerks (see A.5.a) or snoring (see A.4.a); others describe an earlier (and still existent) picture of DIMS with tension–anxiety (A.1.b) or depression (A.2.a).

A substantial number of patients are first introduced to nightly use of sleeping agents during routine hospitalization, though the patient has given no indication on admission of a requirement for sleep medication. Other individuals describe being treated with a hypnotic for a brief insomnia. The prescriptions were renewed by their physician though their acute symptoms and complaints had subsided.

Differential diagnosis: Dependence on CNS depressants by patients who have developed tolerance while using the agents for sleep needs to be distinguished from the *claim* of dependence on CNS depressants associated with DIMS made by individuals in whom the diagnosis of drug abuse would be more appropriate. Malingering patients also will dissimulate a chronic tension/somatic disturbance condition, which they testify interferes with sleep (giving a disruption similar to the accounts of patients with persistent psychophysiological DIMS, A.1.b). In both situations, a careful psychological and medical history and a detailed inquiry into the pattern of drug utilization are helpful in differentiating drug abuse from the condition described in this section.

In addition to consideration of whether the current drug-tolerant or withdrawal status of the patient is as represented, *the patient must be reinvestigated, following the supervised withdrawal of hypnotic compounds,* in an attempt to learn the etiology of the primary DIMS that provoked drug use. DIMS associated with tolerance to (or withdrawal from) CNS depressants is a condition that may be imposed on any other DIMS. In many cases, it is clear that an earlier DIMS associated with a major psychological component (such as borderline personality, chronic tension–anxiety, or depression) brought the patient to the initiation of hypnotic agents. These psychological patterns are generally consistent with the diagnosis of symptom and personality DIMS (A.2.a), persistent psychophysiological DIMS (A.1.b), and DIMS with affective disorder (A.2.b), respectively.

Polysomnographic examination will assist in uncovering other possible pathophysiological sources of the primary DIMS such as sleep apnea DIMS syndrome (A.4.a), sleep-related (nocturnal) myoclonus (A.5.a), other medical, toxic,

and environmental conditions (A.6), or atypical polysomnographic features (A.8.b).

Polygraphic reexamination after withdrawal is particularly important with the advent, during drug withdrawal, of leg jerks satisfying the criteria for sleep-related (nocturnal) myoclonus. If the muscle phenomena are secondary to drug withdrawal, they will not be evident in later recordings.

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A. 3. b. Sustained Use of CNS Stimulants

Key words and phrases: stimulants, analeptic agents, amphetamines, diet pills, caffeine withdrawal, overdosage, drug abuse.

Essential features: *DIMS associated with sustained use of CNS stimulants is an insomnia caused by wake-inducing agents taken in excessive quantities and too late in the day, coupled with the frequent tendency to raise the dosage of the agents when tolerance develops to their desired daytime effects.*

With this pattern, sleep onset is delayed, total sleep time declines, and sleep stages 3/4 and REM are sharply reduced as a function of the prolonged sleep latency and frequent sleep interruptions. The poor nocturnal sleep may increase the tendency to take the stimulant drug during the day so as to maintain alertness. Ultimately, the patients become susceptible to sudden episodes of sleepiness during the day: the classical "crash" of the stimulant-dependent individual. The vicious cycle of increasing ingestion and increasing tolerance to the stimulant agent invites severe reactions of this kind, particularly acute if the dosage is reduced or eliminated.

Associated features and other information: This syndrome is incurred by diverse types of individuals of all ages (some of whom do not desire the wake-inducing effects of the agents): weight reducers; excessive drinkers of caffeine beverages; patients given stimulant drugs following a mistaken diagnosis of narcolepsy or for viral infections that cause somnolence; depressed patients prescribed analeptics for mood elevation and reduction of daytime "fatigue"; asthmatics who inhale sympathomimetic drugs heavily; anxious individuals who feel more confident after a "pill"; and drug abusers looking for a "high." Most individuals originally become habituated to stimulants because they are prescribed for a medical condition.

Other symptoms that occur during and after the period of drug use also may be secondary to the stimulant ingestion. These include anxiety, irritability, personality changes, difficulty in concentration, disturbed interpersonal relationships, and severe depression with suicidal potential. If the pattern is unchecked, the insomniac individual may begin to take hypnotics in order to sleep, along with the

stimulants by day. Toxic conditions and psychotic reactions to amphetamine-like drugs occasionally develop on high dosages (see A.6).

Differential diagnosis: Patients who were put on stimulant drugs earlier because of suspicion of narcolepsy (B.6)—particularly if the history indicates cataplexy or the other accessory symptoms of narcolepsy—should have appropriate polysomnographic investigation. This should be carried out to confirm the diagnosis of narcolepsy *after* the patient has been free of stimulant medication for at least 2 weeks.

Other patients who had complained of sleepiness before being given stimulants should also be withdrawn and investigated for a predisposing condition such as persistent psychophysiological DOES (B.1.b), DOES with affective disorders (B.2.a), sleep-related (nocturnal) myoclonus (B.5.a), and sleep-induced respiratory impairment (B.4).

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A. 3. c. Sustained Use of or Withdrawal from Other Drugs

Key words and phrases: poor sleep, cancer chemotherapy, thyroxine, dilantin, diazepam, tricyclic antidepressants, MAO inhibitors, ACTH, cortisone, oral contraceptives, alpha-methyl-DOPA, propranolol, tranquilizers, marijuana, cocaine, LSD, opium, heroin, PCP, compensatory effect of agent.

Essential features: *DIMS associated with the sustained use of or withdrawal from drugs other than CNS depressants or stimulants is sometimes reported. Drugs that interfere with sleep exert their effect directly; a second group of agents exerts a mild CNS depressant effect, which may not be noticeable to the patient; however, with withdrawal, compensatory DIMS results.*

The drugs that have sleep-interfering effect include antimetabolites, cancer chemotherapeutic agents, thyroid preparations, anticonvulsant agents such as dilantin, monoamine oxidase (MAO) inhibitors, adrenocorticotropic hormone (ACTH), oral contraceptives, alpha-methyl-DOPA, propranolol, and many others. These drugs generally antagonize the sleep-onset process and may, in addition, cause sleep to be interrupted frequently or foreshortened. Variations in the extent of DIMS parallel the dosage and use of the drug; only such a concordance verifies that the reported DIMS is drug related.

The second group of agents includes diazepam, the major tranquilizers, sedating tricyclics (at times also MAO inhibitor antidepressants), illicit drugs such as marijuana, cocaine, phencyclidene, and opiates, and even agents that contain aspirin.

During the period in which the drugs in the second category are used, sleep is not disturbed. If any action on sleep is noted, it is enhancing; some of the agents are frequently used to help sleep continuity problems (e.g., sedating tricyclics,

phenothiazines, marijuana). DIMS develops only in the withdrawal period with reduction in dosage or discontinuation of the drugs. When REM sleep has been reduced by the direct action of the drug, as with MAO inhibitors or some of the tricyclic antidepressants, intense dreaming erupts that will often disturb the unsuspecting patient as REM sleep rebounds during the drug withdrawal period.

Associated features and other information: The clinical picture during withdrawal is virtually identical to that described for CNS depressants (see A.3.a). If, however, withdrawal from the compound is gradual, the patient usually continues to sleep fairly well.

If after a few weeks postwithdrawal of the mildly sedating group, disturbed nocturnal sleep persists, the original or residual causes of the individual's sleep complaints should be investigated.

During withdrawal it is not uncommon to observe leg jerks similar to sleep-related (nocturnal) myoclonus (see A.5.a). Whether the withdrawal period DIMS is partly associated with this secondary myoclonus (if so, code them under A.6), or whether the myoclonus is only a temporary and asymptomatic phenomenon (if so, code them under D.4.n) is not always easy to establish. Careful delineation of the polysomnographic arousal pattern in relation to the muscle jerks is required for this determination.

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A. 3. d. Chronic Alcoholism[2]

Key words and phrases: addictive use of alcohol, chronic drinking alcoholic, chronic "dried out" alcoholic (not a toxic condition, i.e., delirium tremens alcoholic liver disease, Wernicke's encephalopathy, or Korsakow's psychosis).

Essential features: *DIMS associated with chronic alcoholism is characterized during drinking episodes by progressive disintegration of the sleep architecture: persistent sleep interruptions leading to low total sleep time, fragmented REM sleep periods, and reduced REM sleep. When the chronic alcoholic commences withdrawal, deep NREM sleep (stages 3/4) diminishes and REM sleep rebounds until withdrawal is complete. In severe alcoholics, deep NREM and REM sleep may be unremittingly diminished.*

During the initiation of the drinking period, alcohol intake may potentiate sleep and stages 3/4 temporarily. However, with sustained and heavy consumption, disruption of sleep structure begins to take place as REM sleep periods are interrupted and shortened, and finally a sleep maintenance DIMS develops.

[2] For alcohol use as a sedative in individuals who are not chronic alcoholics, see A.3.a; for toxic withdrawal syndrome—delirium tremens—in chronic alcoholics, see A.6.

Acute withdrawal from alcohol in the chronic, drinking alcoholic is characterized by a dramatic prolongation of sleep latency, a sharp reduction in deep NREM sleep, and an increase in stage REMS. (Nonalcoholics who use alcohol as a sedative do not show, on giving it up, the reduction in deep NREMS, see A.3.a.) REM sleep latency shortens, and the number of stage REM transitions increases. REM sleep may be abnormal [with increased electromyographic (EMG) activity], and compensatorily increased to very high levels for a few days, a pattern that is associated with progression of the clinical picture to the toxic withdrawal (delirium tremens) syndrome (see A.6).

Associated features and other information: Persistently low slow wave sleep and fragmentation of REM sleep may be the pattern for many weeks after the last bout of drinking. This pattern, which resembles that seen in aged subjects or mildly brain-damaged patients, may be fixed in a substantial number of chronic, "dried out" alcoholics, though most alcoholics show a return to almost normal sleep structure in 10–14 days after drinking ends.

Differential diagnosis: The patient who self-prescribes ethanol for an underlying psychophysiological DIMS is described in A.1.b and should be differentiated from the chronic alcoholic patient.

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A. 4. *associated with*
Sleep-induced Respiratory Impairment
a. Sleep Apnea DIMS Syndrome

Key words and phrases: central apnea, respiratory muscle apnea, mixed central and obstructive (upper airway) apnea, insomnia with sleep apnea, sleep awakening complaint, snoring, normal pulmonary function while awake.

Essential features: *Sleep apnea DIMS syndrome applies to the cases of sleep-related cessation of breathing in which insomnia is usually the chief symptom. In this syndrome, central cessation of breathing is a prominent finding on polysomnographic recording. When upper airway obstruction is the major cause of the apneic episodes, the presenting complaint is excessive daytime somnolence (for a full description of sleep apnea DOES syndrome, see B.4).*

In the sleep apnea DIMS syndrome, patients have short sleep latencies, usually falling asleep within minutes. However, they wake up several times during the

course of the night, sometimes gasping for air or with the sense of choking. These episodes may be accompanied by anxiety persisting for several minutes after awakening. The polysomnogram demonstrates a pathological number of apneic events in the sleep of these patients. These events are described in section B.4.

Two types of sleep apneas associated with a presenting complaint of DIMS are identified by the polysomnogram. In the more frequent case, there is a predominance of central apnea: cessation of breathing attempts during both REM and NREM sleep. In other cases, the polysomnogram reveals predominantly mixed central and obstructive apnea. The longest apneic episodes are observed concomitant with bursts of rapid eye movements during sleep. Only some apneic episodes lead to complete behavioral awakening. The critical factor in the awakenings does not seem to be hypoxia or hypercapnia inasmuch as equivalent blood levels of oxygen and carbon dioxide occur at other times without resulting in full arousal. Respiration in patients with this syndrome is completely normal in the awake state.

Polysomnography also demonstrates cardiac arrhythmias in some of these patients. An arrhythmia may be secondary to the onset of apnea but may also be concomitant with its onset. It is in the latter case that complete asystole is most frequently seen.

Associated features and other information: Patients report feelings of daytime tiredness and fatigue but usually do not take naps. Snoring frequently occurs during the night. However, snoring is not quite as characteristic or repetitious as in the predominantly obstructive sleep apnea DOES syndrome associated with daytime somnolence (B.4.a). The Multiple Sleep Latency Test, done during the day, does not show the very short sleep latencies in central and mixed apneas as are demonstrated in patients with predominant obstructive sleep apnea.

The sleep maintenance insomnia that characterizes sleep apnea DIMS syndrome may also be associated with sexual problems. Patients occasionally complain of a loss of libido and difficulty having erection. They are also subject to depressive reactions. The hemodynamic complications of this syndrome are considerable if not adequately treated. This issue is more fully described in B.4.a.

The prevalence of sleep apnea DIMS in the general population is unknown. The syndrome seems to be more frequent in the elderly (over age 60) than in younger age groups and is concentrated in males. Sleep apnea associated with insomnia has been found to have an incidence of 0–10% (mean, 7%) of all insomniac patients in a group of more than 1500 polysomnographically studied sleep clinic patients in the United States and abroad.

Differential diagnosis: With respect to the differential diagnosis of DIMS, sleep apnea must be considered when a patient presents with a short sleep latency and complaint of difficulty maintaining sleep. Patients with depression may also present chiefly sleep maintenance insomnia (see A.2.a). Insomnia secondary to neurological factors—sleep-related (nocturnal) myoclonus (A.5.a) and sleep-related epileptic seizures (D.4.b) associated with apnea—and non-apnea sleep-related cardiorespiratory problems (see A.4.b; D.4.j,k) must be differentiated from the central and mixed apnea syndrome.

Patients with sleep-related abnormal swallowing syndrome (D.4.i) and gastroesophageal reflux (D.4.1) may present with a complaint similar to that reported with sleep apnea DIMS syndrome; abrupt awakenings during the night coupled with feelings of choking or marked shortness of breath, extreme anxiety, and sometimes also a feeling of dying. Their daytime histories may reveal esophagitis symptoms, but not always. The sleep polysomnogram will easily distinguish the two conditions.

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A. 4. b. Alveolar Hypoventilation DIMS Syndrome

Key words and phrases: central alveolar hypoventilation, primary alveolar hypoventilation, hypopnea, "Ondine's curse," cordotomy, neurological lesions, pulmonary function may be abnormal while awake.

Essential features and other information: *Alveolar hypoventilation DIMS syndrome is a consequence of ventilatory impairment worsened by sleep. The conditions—including primary alveolar hypoventilation ("Ondine's curse"), which is usually reported in infants—may be associated with disrupted sleep and complaints of insomnia [for alveolar hypoventilation (DOES) syndrome, see B.4.b].*

Ventilatory studies reveal unresponsiveness to chemical control of ventilation during wakefulness and sleep in primary alveolar hypoventilation, but spirometric studies and other pulmonary tests demonstrate normal lung functioning. In sleep, tidal volume decreases with the appearance of hypercapnia and hypoxemia and further declines with their worsening. Sleep-related central apnea may be interposed intermittently or repetitively with the hypopnea. The cause of this syndrome is believed to be related to CNS pathology, though no definite lesions have been uncovered as yet by neurohistological examination. The condition is a decided threat to survival.

Alveolar hypoventilation in adults may be secondary to conditions such as massive obesity, chronic obstructive pulmonary disease, involvement of chest bellows secondary to scoliosis muscle impairment, and others. Adults who, during wakefulness, have alveolar hypoventilation with the chemical accompaniment of hypoxemia and hypercapnia may develop nocturnal sleep disturbance. Sleep, and particularly REM sleep, appears to aggravate the deterioration of the arterial blood gas measurements. Both hypopnea and apnea (central, mixed, or obstructive) may be observed during the sleep of these patients.

In addition to chest and lung causes of alveolar hypoventilation, patients with a

cordotomy or a neurological lesion involving structures that control ventilation may show the appearance or exacerbation of alveolar hypoventilation during sleep. Accordingly, decreased tidal volume or pure central apnea associated with hypoxemia and hypercapnia may present only as a sleep-related phenomenon. Cases in this latter group have been incorrectly classified as "Ondine's curse" despite the existence of specific and identifiable histological lesions.

Differential diagnosis: Patients with true primary hypoventilation syndrome ("Ondine's curse"), or with alveolar hypoventilation resulting particularly from neurosurgical or neurological lesions, must be distinguished from the "central sleep apnea" patients described in the sleep apnea DIMS syndrome (A.4.a). The central sleep apnea patients have neither abnormal ventilatory responses in the awake state nor any discernible brain or cord lesions.

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A. 5. *associated with*
Sleep-related (Nocturnal) Myoclonus and "Restless Legs"
a. Sleep-related (Nocturnal) Myoclonus DIMS Syndrome

Key words and phrases: nocturnal myoclonus, leg jerks, nonepileptic, insomnia.

Essential features: *Sleep-related (nocturnal) myoclonus DIMS syndrome is a condition in which insomnia is associated with the occurrence during sleep of periodic episodes of repetitive and highly stereotyped leg muscle jerks. In some individuals, excessive daytime sleepiness rather than insomnia is the chief complaint [see sleep-related (nocturnal) myoclonus DOES syndrome, B.5.a].*

The myoclonic jerks are generally bilateral but may involve either leg alone without apparent pattern. The myoclonus is for the most part independent of generalized body movement during sleep and is not observed in waking. The contractions always consist of extension of the big toe in combination with partial flexion of the ankle, knee, and sometimes hip. They are consistently *followed* by a partial arousal or awakening. Between the episodes of myoclonus, the polysomnogram is normal.

The diagnosis of sleep-related (nocturnal) myoclonus depends on a set of specific criteria pertaining to the muscle contractions, which distinguish them from types of leg muscle activity associated with other conditions. The anterior tibialis EMG shows repetitive myoclonic contractions, each lasting 0.5–10 seconds; the interval between jerks is typically 20–40 seconds (jerks that are separated by an interval of less than 5 or more than 120 seconds cannot be included in a period of myoclonus); the repetitive contractions are grouped in at least three separate and

distinct episodes of a few minutes to an hour or more, each containing a minimum of 30 jerks; the myoclonic contractions must be initiated *in* sleep—jerks occurring during drowsiness, before the onset of NREMS stage 1, and *following* arousals are not counted. Variability, not only in the amount but in the very presence of the muscle jerks, has been reported in certain individuals over a series of recording sessions.

Patients often have no knowledge of the myoclonic twitches that lead to this DIMS. Rather, they present a history of frequent nocturnal awakenings and unrefreshing sleep, though some patients with this condition also are unaware of the interruptions of sleep and emphasize other complaints (see B.5.a).

Associated features and additional information: Sleep-related (nocturnal) myoclonus may be suggested by a number of other difficulties that have a higher incidence in these patients than in the general population. These include leg cramps, family history of sleep maintenance disorders, restless sleep and disruption of bedclothes, and falling out of bed. A history from a bed partner is pivotal to a complete clinical description of the disorder, because the muscle contractions may be beyond the patient's awareness. Such an observer will alert the examiner to the diagnosis by mentioning the intermittent and rhythmic leg kicking.

Sleep-related (nocturnal) myoclonus is seen predominantly in middle-aged and older individuals of both sexes. It is extremely rare in children. Patients with this condition occasionally have the much rarer "restless legs" syndrome (A.5.b) as well, whereas virtually all patients with "restless legs" also have sleep-related (nocturnal) myoclonus. Stress and emotional upheaval may cause the myoclonus to appear or worsen. Estimates of the incidence of this disorder among severe insomniacs range from 1 to 15%. A few studies suggest familial patterns for this disorder, though documentation is not yet sufficient.

Differential diagnosis: It is not certain that sleep-related (nocturnal) myoclonus is a unitary disorder inasmuch as it seems to be associated with, or evoked by, a variety of conditions. Episodes of sleep-related myoclonus may develop in chronic uremia and other metabolic disorders (see A.6); some patients with narcolepsy (B.6) also have sufficient episodes, numbers, and frequency of myoclonic twitching to justify the diagnosis of sleep-related (nocturnal) myoclonus, and some patients treated with tricyclic antidepressant compounds manifest this disorder, as do patients during withdrawal from a variety of drugs such as anticonvulsants, benzodiazepines, barbiturates, and other hypnotic agents. Sleep-related myoclonus associated specifically with ingestion or withdrawal from drugs should be distinguished from the drug-free DIMS disorder, sleep-related (nocturnal) myoclonus, classified here (A.5.a). In the former case, the condition should be categorized either under DIMS associated with other medical, toxic, and environmental conditions (A.6) or under asymptomatic polysomnographic finding (D.4.n), depending on appraisal of the nature of its role in connection with a concurrent DIMS.

Sleep-related (nocturnal) myoclonus must also be differentiated from certain forms of sleep-related epileptic seizures (D.4.b) and myoclonic epilepsy, from "hypnic jerks" (a generalized, and usually single, body twitch manifested occa-

sionally in the transition to sleep), and from a number of forms of waking myoclonus—such as in the Lance-Adams syndrome (intention myoclonus), Alzheimer's and Creutzfeldt-Jakob's diseases, and other neuropathological conditions.

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A. 5. b. "Restless Legs" DIMS Syndrome

Key words and phrases: disturbed sleep, creeping sensations when legs stationary, need to keep moving, sleep-related (nocturnal) myoclonus, and restless legs.

Essential features: *DIMS is associated with the "restless legs" syndrome because an individual feels extremely disagreeable deep sensations of creeping inside the calves whenever sitting or lying down. These dysesthesias are rarely painful, but agonizingly relentless, and cause an almost irrestible urge to move the legs, thus interfering with sleep.*

Associated features and other information: Vigorous exercise of the lower extremities reportedly relieves the symptom, but the relief gained from such exercise varies. In severe cases the dyesthesias reappear almost as soon as the exercise is terminated.

The etiology of the "restless legs" syndrome is currently unknown. Inadequate circulation is often suggested. Some cases have been linked to motor neuron disease.

Almost all patients with "restless legs" syndrome also have sleep-related (nocturnal) myoclonus (A.5.b), which independently causes arousals from sleep due to repetitive leg muscle contractions *without* a sensory component; only a small proportion of the cases of sleep-related (nocturnal) myoclonus—a more frequent condition than "restless legs"—also have "restless legs." The mechanism that explains the association of the two conditions is not understood.

The "restless legs" syndrome becomes more severe with age. It is exacerbated by sleep deprivation and occurs with particular gravity during pregnancy. Curiously, the syndrome often disappears with fever. The unremitting leg sensations may give rise to severe emotional disturbances in some patients, including severe depression and suicide.

Approximately one-third of patients with "restless legs" syndrome show a familial incidence. The syndrome is probably transmitted as an autosomal dominant trait with reduced penetration.

Differential diagnosis: When considering the diagnosis of "restless legs" syndrome, several other possibilities should be ruled out: painful leg cramps from various causes are not uncommon (they are sometimes related to iron and calcium deficiencies); the "restless legs" phenomenon often appears in chronic uremia; "growing pains" in children occasionally may be quite severe; and the agitation and restlessness of some anxious, emotionally upset patients may mimic the continuous moving of the patient with "restless legs."

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A. 6. *associated with*
Other Medical, Toxic, and Environmental Conditions

Key words and phrases: medical, neurological, diseases, toxic, environmental, pain, discomfort, nonspecific symptoms, parasomnias causing DIMS.

Essential features: *This category covers those medical (including neurological), toxic, and environmental conditions that are invariably associated with DIMS. Additionally included are conditions having a significant association with DIMS, though DIMS is neither invariable nor necessarily the major or most urgent symptom or sign.*

The onset, time course, and termination of the insomnias classified here are tied to the stage of the related medical (neurological), toxic, or environmental condition. Amelioration of the basic condition brings about a reduction of the sleep problem, either immediately or gradually.

Certain symptoms are also included in this DIMS category. A number of these are common to many diseases. Accordingly, when the focus is on symptoms, they—not the diseases—are responsible for the sleeplessness. As would be expected, sleep usually improves or becomes normal during temporary symptomatic relief.

Relationship to other DIMS: Though DIMS is frequently an intrinsic property of an underlying medical condition or its physical symptoms, other causes of insomnia may obtain under circumstances of medical illness. If, for example, insomnia remains when somatic symptoms are relieved, the DIMS is likely to have become sustained by psychophysiological factors (see A.1.a,b).

An underlying medical condition or symptom may be uniquely induced by sleep or aggravated by sleep, a certain stage of sleep, or the usual recumbent position of sleeping individuals. Some of these conditions are categorized in section D, the parasomnias: dysfunctions associated with sleep, sleep stages, or partial arousals. Any parasomnia (section D) that is responsible for the *clearly allied insomnia* should *also* be coded as a cause of DIMS under this category (A.6), unless—as in the case of sleep-induced respiratory impairment (A.4.), sleep-related (nocturnal)

myoclonus (A.5.a), and atypical polysomnographic features (A.8.b)—provision has been made for explicit coding elsewhere in section A.

Another group of conditions that categorically qualify for this category (A.6) but have been given special coding elsewhere in section A are the DIMS syndromes induced by pharmacological agents. Examples include chronic alcoholism (A.3.d) and habitual use or withdrawal from CNS depressants, stimulants, and other drugs (A.3.a,b,c). However, when the response to or withdrawal from a drug has a toxic character, as in delirium tremens, the reaction is included, and should be coded, in this section (A.6).

Medical (neurological) conditions: DIMS are significantly associated with certain medical conditions. In most of these conditions, the fundamental etiology of the DIMS is unknown, unspecified, or poorly understood. In some, DIMS is caused by symptoms such as pain, fever, and pruritis. When there is a psychophysiological DIMS response (A.1.a) as well, the sleep disturbance is not correlated with solely the severity of the primary medical or neurological condition.

Central nervous system conditions

Central nervous system lesions are commonly associated with DIMS. The direct cause may be due to symptoms such as pain, paraesthesia, and abnormal movements; a fundamental change in state of consciousness; or disruptions of the sleep–wake cycle. Some disorders are familial. Lesions of various etiologies in the brainstem and hypothalamus are especially liable to cause DIMS, as are the many causes of cerebral atrophy. Total insomnia, or "persomnia," is sometimes seen in association with CNS lesions.

In organic mental states other than schizophrenia, a consistent reduction in total sleep is seen. Deep NREM (stages 3/4) sleep and REM sleep are reduced. Awakenings from sleep are often associated with episodes of nocturnal confusion.

After an acute period, patients with presenile and senile psychoses persist in a chronic sleep disturbance. Fragmentations of sleep are associated with interrupted REMS periods, alpha intrusions, dissociations of sleep stage patterns, and insertions of stage REM sleep during NREMS stages.

Central nervous system disorders especially associated with DIMS include the following:

Neoplasms of the CNS: pineal tumors and other tumors that affect the hypothalamus, brainstem, and third ventricle; tumors that cause abnormal movements, epilepsy, or increased intracranial pressure.

Vascular disorders: cluster migraine, migranous neuralgia, nocturnal pseudo hemiplegia, vascular lesions of the brainstem, chronic cerebrovascular disease.

CNS infections: acute and chronic diseases such as cerebral syphilis; Sydenham's chorea; trypanosomiasis (Gambian or Rhodesian sleeping sickness); slow virus infections (such as kuru); brain abscess; tetanus (*Clostridium tetani* infection), giving rise to tonic spasms that persist during sleep; encephalitis lethargica (associated in the acute phase with reversal of the sleep–wake rhythm, and in both the acute and chronic phases associated with abnormal movements during

sleep); subacute inclusion encephalitis (also associated with abnormal myoclonic movements during sleep) and other infective encephalopathies.

Degenerative conditions: some of poorly understood etiology such as Morvan's syndrome, *Chorée fibrillaire de Morvan* (not to be confused with Morvan's disease); jumping syndromes such as myriachit ("jumping Frenchman of Maine"); multiple sclerosis; Parkinson's disease; Shy-Drager's syndrome (also a cause of sleep apnea); and chronic brain syndromes such as senile dementia, senile nocturnal mania and confusion, and Alzheimer's disease.

In degenerative diseases like amyotrophic lateral sclerosis, Unverricht's familial myoclonic epilepsy, and the paramyoclonus of Friedreich, abnormal movements during sleep may be the more direct cause of DIMS.

The confusional pattern at night in patients with organic mental disorders should be differentiated from the very brief episodes of nocturnal confusion often experienced by elderly patients subjected suddenly to a new environment (see A.1.a). However, it is not known to what extent degenerative changes in the CNS are responsible for symptoms of insomnia in otherwise apparently normal elderly people.

Trauma to the CNS (including also neurosurgical intervention), particularly to the brainstem.

Toxic encephalopathies: listed in the section Toxic factors (below).

CNS disorders of multiple or unknown etiology: Examples of such disorders associated with DIMS include myoclonic epilepsy, other sleep-related epilepsy (D.4.b), palatal myoclonus, hemifacial spasms, excessive startle, and miscellaneous states of mental retardation.

Hypnagogic startle and physiologic myoclonus during sleep are normal findings of no known significance, although they may become more prominent in states of fatigue.

Muscular and peripheral nerve problems: myotonic dystrophy, peripheral neuritis of many etiologies, night cramps (systremma), fibrositis syndrome (which may cause *and* be exacerbated by DIMS), growing pains or charley horses, carpal tunnel syndrome, tired arm syndrome, and "honeymooners arm."

Other medical conditions

Various types of medical conditions are associated with DIMS: *endocrine and metabolic diseases,* Addison's disease, Cushing's syndrome, diabetes mellitus (Somogyi effect, nocturnal diarrhea, peripheral neuritis), hyperthyroidism, hypothyroidism, nutritional disorders, pregnancy and postpartum disturbances; *renal failure; certain bacterial, fungal, viral, and parasitic infections* such as tuberculosis (with night sweats), syphilis, and filariasis; *arthritis and rheumatoid disease; gastrointestinal disease,* hepatic failure (often with nocturnal delirium); *alcohol-associated pathologies,* alcoholic liver disease, delirium tremens (during which a sharp REM sleep rebound is observed), Wernicke's encephalopathy, Korsakow's psychosis, visceral (especially vesical) crises of tabes dorsalis; *Lafora's disease; gastric and duodenal ulceration; sleep-related dyspepsia and regurgitation; allergies from foods, chemicals, or miscellaneous environmental fac-*

tors, especially those causing nocturnal nasal congestion, pruritis, or asthma; *pulmonary disease of various etiologies,* usually due to nocturnal dyspnea; *heart disease with left heart failure* such as in systemic hypertension, aortic valvular disease (especially aortic incompetence in which patients are disturbed by a pounding pulse); *nocturnal angina and palpitations; total blindness,* probably due to circadian rhythm disruption and persistant psychophysiological (A.1.b) or psychiatric (A.2.a,b) DIMS; *polycythemia rubra vera; anemia.*

Symptoms

The following symptoms, common to many diseases, may be associated with DIMS: *pain,* emanating from heartburn, duodenal ulcer, cholecystitis, arthritis, rheumatism, fibrositis, migraine, angina pectoris, passage of kidney stone, "teething," and metastatic disease of bone; *paraesthesiae* such as that from costoclavicular compression, diabetic peripheral neuropathy, and malignancy; *pruritis,* reported in skin disorders, monilial infections in diabetics, intestinal worms, hyperparathyroidism, and irritation from wet bedclothes; *pyrexia; visceral tension,* as in intestinal or bladder distension; *strangury,* a common nocturnal symptom with multiple etiologies; *sleep-related proctalgia; shortness of breath,* as in paroxysmal nocturnal dyspnea, asthma; *persistent cough;* and *enforced positions,* as with orthopedic prostheses or casts.

DIMS associated with illnesses that give rise to *abnormal movements* during sleep are worthy of special consideration (in addition to sleep-related nocturnal myoclonus and "restless legs," A.5). These sleep-disrupting movement conditions comprise several infectious and degenerative disorders of the CNS (mentioned above) that result in abnormal movement; excessive startle, in neurological diseases of various etiologies; myoclonic epilepsy; metabolic and toxic disorders, exemplified by uremia and heavy-metal poisoning. [Note that when temporary conditions that present like sleep-related (nocturnal) myoclonus develop *secondary to the actions of or withdrawal from drugs* (see A.5.a and A.3.a,c), they should be classified here, A.6, if it is polygraphically determined that the muscle jerks make an *independent* contribution to the DIMS condition; if not, they should be coded under asymptomatic polysomnographic finding, D.4.n.]

Parasomnias: Though parasomnias may first be induced or exacerbated by sleep, they may in turn cause DIMS. In this event, the condition should be coded in section D as a parasomnia, and *also* in this section (A.6) as a *cause of DIMS*. Examples of such parasomnias include bruxism (D.4.c); gastroesophageal reflux, causing regurgitation, heartburn, and dyspepsia (D.4.1); abnormal swallowing syndrome (D.4.i); painful erections (D.4.g); nocturnal angina (D.4.k); sleep-related asthma (D.4.j); and occasionally, cluster headaches (D.4.b) and repetitive dream anxiety attacks (D.4.a).

Environmental factors: Physically measurable environmental factors can be associated with DIMS. The absolute level of the noxious stimulation is often less crucial than the sensitivity of the patient to it. Sensitivity to environmental disturbances in nocturnal sleepers increases toward morning. It also appears to be enhanced with increasing age.

Three conditions must obtain to make a diagnosis of DIMS associated with

environmental factors: (1) the DIMS is temporarily associated with the introduction of a physically measurable, disturbing stimulus; (2) the *physical* properties of the noxious stimuli are the critical disturbing elements, not their psychological meaning; and (3) the removal of the noxious stimulus causes an immediate or at least a gradual return to adequate sleep.

In many of the environmental conditions, it is likely that symptoms such as pain, movement, or psychological response are the more direct causes of the sleep disorder. When DIMS is related to a barely perceptible but psychologically meaningful environmental stimulus, the problem should not be classified here (see A.1.a).

Sleep-disturbing environmental circumstances encompass heat, cold, noise, light, movements of a bed partner, and the necessity of remaining alert in a situation of danger or when having to provide attention to an infant or invalid. Allergic reactions to environmental factors can be associated with DIMS and are classified in this general section (see Other Medical Conditions, above).

DIMS associated with hospitalization may be related to medical or surgical management because of such factors as imposed, abnormal sleep–wake schedules or discomfort from drainage tubes and hemodialysis. (Hospitalization also may result in DIMS caused primarily by unfamiliar surroundings or fearful expectations. When the psychological reaction is paramount, the DIMS should be classified as A.1.a.) Experiences of individuals with institutionalization are especially significant because of frequent careless attitudes toward use of sedative–hypnotic drugs (even in the absence of actual DIMS).

Toxic factors: DIMS are among the toxic effects of arsenic, mercury, copper (Wilson's disease), and other heavy metals; carbon monoxide; radiation; cytotoxic chemotherapy; acute (alcoholic hallucinations, pathological intoxication) and chronic (Wernicke-Korsakow syndrome) alcohol administration; most stimulants (amphetamine psychosis) and tobacco. Toxic and aberrant (involving an acute brain syndrome) reactions to narcotics and other drugs of abuse are associated with DIMS and should be coded here.

In the toxic psychoses from alcohol, drugs, and all other causes, sleep is often fragmented and total sleep time is sharply reduced. Stages 3/4 and REM sleep are greatly diminished in amount. Characteristic waveforms such as spindles and K complexes become indistinct. Return to normal sleep architecture may occur with appropriate treatment.

Pink disease (acrodynia), a now rare sequel of mercury poisoning in infants, is of interest because of the reported sleep–wake reversal and the possible relationship to Morvan's syndrome.

Incidence: Although the incidence of DIMS associated with medical, toxic, and environmental conditions is unknown, the most common causes, *in adults,* may be chronic pain (especially that due to rheumatism and arthritis); nocturnal dyspnea; nocturnal discomfort from pruritis; peripheral neuritis; enforced positions; strangury; dyspepsia; cerebral degeneration; abnormal movements; secondary disturbances of the circadian sleep–wake cycle; and the environmental factors associated with hospitalization. *In children,* the prominent causes of DIMS are pain and irritation from wet bedclothes; teething; parasitic worms; gastrointestinal disorders; tonsil and adenoid enlargement; pain from gas-

trointestinal upset or joint disease, and, more rarely, chorea, encephalitis, pink disease, and rickets.

Differential diagnosis: The most common differential diagnoses are situational and persistent psychophysiological DIMS (A.1.a,b), personality and symptom psychiatric disorders (A.2.a), and persistent sleep–wake schedule disturbances (C.2).

When a medical, toxic, or environmental factor is independent and additional to other coded determinants of a DIMS, it should be coded separately.

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A. 7. Childhood-Onset DIMS

Key words and phrases: life-long poor sleeper, childhood onset, prepubertal, consistent poor sleeper independent of emotional variations, symptomatic by day, not short sleeper.

Essential features: *Childhood-onset DIMS is a sleep-onset and sleep-maintenance insomnia, resulting in daytime symptoms of inadequate sleep, that is characterized by a distinctive history of (unexplained) development before puberty, and persistence into adulthood.*

In some individuals, documentation exists of this DIMS being observed in early infancy. The clinical and polysomnographic presentation of childhood onset DIMS is very similar in the adult to persistent psychophysiological DIMS and symptom and personality disorder DIMS except for its early commencement and the absence of assignable, recent conditioning factors and psychopathological patterns (see A.1.b and A.2.a for comparisons).

Associated features and other information: Nevertheless, the distinction in terms of psychological features is not always sharp. More than a few

patients who report early onset of insomnia may have also developed nonspecific psychological disturbances, and some have a history of family turmoil early in their lives. It is not a simple matter, in the face of a combined clinical picture of some psychological problems and early sleep disturbance, to determine whether the former is independent of the chronic insomnia, secondary to it (as often may be expected), or whether they both result from unknown or unspecified early experiences. It is also possible that the early insomnia might have been short-lived except that negative learning and tension-anxiety factors had also been mobilized in relation to sleep, albeit now forgotten.

Childhood-onset DIMS also may constitute a CNS shift or dysfunction of the sleep–arousal equilibrium. This possibility is supported by the poorer responsiveness of early-onset DIMS patient to treatment compared to patients with other types of DIMS. To the degree that the term "primary insomnia" has been used to suggest a fundamental CNS imbalance of sleep–wake mechanisms, it may apply to childhood-onset DIMS, particularly in those cases in which psychological factors seem notable in their absence.

Differential diagnosis: The severity and nature of the psychological disturbance, irrespective of the age at onset, should determine whether a patient is classified as a psychological DIMS (A.1.b or A.2.a) or as childhood onset.

A helpful clinical fact concerning childhood-onset DIMS in relation to persistent psychophysiological (A.1.b) and symptom and personality disorder DIMS (A.2.a) is that, in the former, poor sleep patterns tend to continue almost unvaried through both poor and good periods of emotional adaptation.

These patients are separated from short sleepers (9.a) by the presence of daytime symptoms of fatigue, irritability, tenseness, mild depression, difficulties in awake attention, and, at times, daytime sleepiness. The latter group, though frequently able to trace a short sleep tendency back to childhood, both function and feel well during the day with little sleep. They do not have a DIMS.

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A. 8. *associated with*

Other DIMS Conditions

a. Repeated REM Sleep Interruptions

Key words and phrases: rhythmic interruptions of sleep, repeated dream arousals, unknown etiology, not dream anxiety attacks, not a REM sleep parasomnia.

Essential features: *DIMS associated with repeated REM sleep interruptions is a sleep maintenance insomnia with a characteristic pattern of awakenings starting in the first REM sleep period, about 90 min after sleep onset, and recurring in almost every subsequent REM sleep period.*

REM sleep interruption insomnia does not necessarily decrease the ratio of

REM sleep to total sleep, because on falling back to sleep, patients with this DIMS may return to REM sleep almost immediately. Many awakenings, however, are not short-lived. Total sleep time may be curtailed to 4–6 hours in this condition.

Unless the intervals between the interruptions of sleep are absolutely unvarying (e.g., every 90 minutes), which REMS–NREMS cycles rarely are, or the patient remembers a vivid dream with every awakening, polysomnography is required to make the diagnosis of REM sleep interruption DIMS. This diagnosis applies only if the awakenings are longer than 2–3 minutes, affect more than 75% of all REM periods, and occur only rarely (less than 20%) in NREM sleep.

Additional features and other information: REM sleep interruption DIMS is not a common cause of insomnia, probably accounting for much less than 10% of reported DIMS. Most reported cases are adults over age 35, with men outnumbering women. In the typical patient, clinical psychopathology—particularly depression—has been or is a problem, and the patient's insomnia may be a feature of the current psychological difficulties. Frequently, the patient reports having had a period of extremely disturbing dreams or nightmares. The REM sleep interruptions, which later progress to this uniquely fixed type of sleep continuity insomnia, seem initially to represent a learned avoidance of the dysphoric dreams. (REM sleep periods in this condition, because of their frequent interruptions, are shorter, though more frequent.) Whether this condition of repetitive arousals from REM sleep develops only in patients with a very low REM sleep awakening threshold is not known.

Differential diagnosis: This condition must be distinguished from other conditions in which the awakenings are elicited by somatic phenomena evoked in REM sleep. Included among these possibilities are sleep-related cluster headaches (D.4.h), angina pectoris and paroxysmal nocturnal dyspnea (D.4.k), REM sleep-related sleep apnea (A.4.a) or painful erections (D.4.g), and atypical polysomnographic features (A.8.b). If the awakenings are *always* accompanied by a vivid nightmare, the diagnosis of dream anxiety attacks (A.4.a) would be a proper additional designation.

DIMS associated with repeated REM sleep interruptions is clearly an alternative diagnosis, in the face of this singular pattern of nocturnal awakenings, to DIMS with symptom and personality disorder (A.2.a), DIMS with affective disorders (A.2.b), or persistent psychophysiological DIMS (A.1.b).

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A. 8. b. Atypical Polysomnographic Features

Key words and phrases: miscellaneous atypical polysomnographic recordings, alpha-delta sleep, "mini"-arousals, nonrestorative sleep.

Essential features: *DIMS with atypical polysomnographic features is a condition in which sleep is experienced as frequently interrupted and "nonre-*

storative," and the sleep stage structure is marked by abnormal physiological features.

In the patient's judgment, the quality of sleep is poor (e.g., "light," "restless"). Even after 7–8 hours of sleep, one feels unrefreshed. The most common deviation from normal sleep tracings is the presence of high voltage alpha waves superimposed on the NREM sleep EEG slow waves (e.g., "alpha sleep" or alpha-delta sleep). REM sleep is spared the alpha "riddling" that characterizes deep NREM stages. Additionally, the recording may contain numerous, short alpha intrusions and EMG bursts (short arousals). There is a suggestion of insufficient spindle activity or an abundance of REM sleep phasic activity in certain cases but these observations have not been documented.

Associated features and other information: Patients displaying atypical polysomnographic features are unhappy about their sleep. They feel tired, "washed out," and insufficiently rested during the day. The complaint of stiff and achy muscles is common. However, daytime symptoms do not take the form of the extreme sleepiness seen in DOES syndromes (see section B).

Final diagnosis of DIMS with atypical polysomnographic features can be made *only* by recorded demonstration of abnormal physiological conjunctions (noted above) in the presence of reasonably normal sleep duration. The patient's subjective complaint of "poor sleep" is not sufficient. Patients with this condition may have resorted to liberal use of sleep medications, which must be withdrawn before diagnosis is attempted.

Differential diagnosis: The finding of a secondary type of atypical polysomnogram is not unusual during use of stimulant or sleep medications. For example, increased alpha activity and spindles often are observed during drug intake and withdrawal, particularly with barbiturates and amphetamines. Only if the atypical polysomnographic features persist after drug wash-out should the primary DIMS be classified under this category.

Numerous other DIMS syndromes, particularly DIMS in depression (see A.2.a) and persistent psychophysiological DIMS (A.1.b)—with long-term loss of sleep or with specific sleep stage deprivation—may show an atypical sleep structure such as short REM sleep latency or spindles in REM sleep, respectively. These syndromes should not be diagnosed as DIMS with atypical polysomnographic features (A.8.b) unless the polysomnographic abnormalities continue after sleep loss has been corrected. It should be expected that in a few cases a primary sleep structure problem with initial low grade sleep symptoms, may additionally develop persistent psychophysiological DIMS (A.1.b). Double classification is recommended if this combination of two syndromes is documented. The sleep pattern and, consequently, the sleep structure of patients with occult systemic disease associated with daytime fatigue may appear aberrant (see A.2.a, secondary depression).

The category of DIMS with atypical polysomnographic features was not meant to cover parasomnias (D), sleep apnea (A.4.a), or sleep-related (nocturnal) myoclonus (A.5.a). It appears also to be a different condition from repeated REM sleep interruptions (A.8.a), in which frequent arousals and alpha intrusions are found in REM sleep.

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A. 9. No DIMS Abnormality

a. Short Sleeper

Key words and phrases: short sleeper, "healthy" insomniac or hyposomniac, asymptomatic, one end of normal sleep duration continuum.

Essential features: *Short sleeper is the designation for an individual who consistently sleeps (and feels the "need" to sleep) substantially less in 24 hours than the conventional amount of sleep for one's age group. The sleep, though short, is unbroken and normal. This pattern is associated with an absence of complaints about quality of sleep, daytime sleepiness, or difficulties with awake mood, motivation, and performance. No true DIMS exists despite the occasional desire and attempts (unsuccessful) by the individual to sleep longer. Short sleep appears to be at one end of the normal, individual sleep requirement continuum.*

For purposes of comparison, a regular pattern of daily sleep totaling less than 75% of the conventional quantity for age fits the criterion for short sleeper. Some individuals sleep for periods of only 45 minutes to 3 hours each day without compromise of waking faculties. The polysomnogram shows a short sleep latency and very few arousals after sleep onset. Absolute amounts of sleep stages 3/4 are normal, but stages REM and NREM 2 are lower than in conventionally sleeping adults. The patient has no problem with time distortion or ability to be accurate about the quality of sleep.

Associated features and other information: Certain psychological features are associated with male short sleepers. The subjects are basically normal psychologically but have a tendency to hypomanic behavior. They are described as smooth, efficient persons who are distinct "nonworriers." Generally the individuals' concerns in seeking assistance for sleep center about the awkwardness they feel in family and social relationships because of the short sleep behavior. They worry that perhaps they are psychologically or medically abnormal and that, in addition to their own suspicions, others will regard them as deviant. Generally these considerations pertain only to short sleepers who retire for less than 4 hours.

Usually the short sleep pattern begins in early adolescence or young adulthood and endures throughout life, without the advent of known impairments or complications. The diagnosis of short sleeper most likely refers to a very small segment of the population, probably accounting for fewer than 5% of reported insomnia cases. The tendency to short sleep seems to run in families and favors males.

Demographic data from several population studies appear to link short sleep to reduced life expectancy. This relationship probably has its source mainly in short total sleep time patterns resulting from medical and sleep pathologies, not in short sleep as represented by the short sleeper. In other words, the survey studies from

which such correlations are drawn were not able to explore the probability that root pathological causes are responsible for *both* the higher mortality ratios and the circumscribed sleep. The question of a causal connection between unconventional sleep durations that are not associated with pathological processes and shortened life expectancy must remain moot in advance of suitable data.

Differential diagnosis: It is important to differentiate a short sleeper from an insomnia patient. The diagnosis may be made chiefly from the history. The insomnia patient complains of difficulty either in falling asleep, remaining asleep, or both. Key to the patient's concerns are major and often desperate complaints concerning daytime functioning, exhaustion, and unsatisfactory mood. These patently attenuate with better sleep.

Essential to valid identification of the short sleeper is the consistency of short sleep, with no weekend or holiday reversions to conventional or long sleep patterns. The short sleeper, by definition, reports a regular pattern of reduced sleep, but unlike a "poor" sleeper, he is satisfied with his sleep and does not complain of lack of sleep, the quality of sleep, or impairments in awake functioning. The short sleep is restorative. As indicated above, the decision to consult help is often tied to the individual's belief that sleeping very little is abnormal and may lead to medical disabilities. If the individual has any sleep complaint at all, it is that longer sleep is rarely possible when attempted. The very short sleeper may also find it difficult to deal with boredom. These are often the symptoms of "insomnia" that are related by older individuals who feel they *should* sleep longer. Careful assessment in the history that sleep is short, but without evidence of actual sleep "disturbances" or difficulties during waking, is against DIMS and favors short sleep.

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A. 9. b. Subjective DIMS Complaint without Objective Findings

Key words and phrases: subjective complaint, pseudo insomnia, sleep hypochondriasis, distorted sleeping time sense, unknown etiology.

Essential features: *Subjective DIMS complaint without objective findings is the designation for a convincing and honest complaint of "insomnia"—made by an individual lacking apparent psychopathology—that is at variance with laboratory evidence of normal sleep length, architecture, and physiology. Though polysomnography may reveal sleep latencies of less than 15–20 minutes and sleep durations in excess of 6½ hours, the same subjective*

description of disturbed sleep at home is applied to the sleep in the laboratory. The patient is sure that reversal of the "insomnia" will improve awake functioning.

This diagnosis therefore requires a persistent DIMS complaint, coupled with polysomnographic recordings repeatedly within normal limits, in an individual with a personality pattern not commensurate with conventional hypochondriasis, malingering, or other psychological disturbance.

Associated features and other information: A subjective conviction of insomnia in the absence of objective confirmation is seen in all age groups and somewhat more frequently in women. This paradoxical picture may be uncovered in almost one-quarter of all persons presenting with insomnia complaints. Such a perplexing condition, in terms of cause or mechanism, probably encompasses different types of problems and individuals.

Some reasons for the disparity between subjective and objective DIMS parameters have been advanced: excessive mentation during sleep may contribute to the "sense" of being awake; physiologic abnormalities may exist in the sleep tracing that are too subtle to be detected by recording methods currently in use (e.g., "micro-arousals"); data exist that some of these patients are grossly inaccurate in their estimations of time spent asleep; others possibly have a long sleep "need," and their DIMS symptoms may be understandable in the context of a requirement for much longer sleep than 6–8 hours daily; alternatively, a subgroup of these patients may be obsessive about sleep processes, just as other persons are obsessionally hyperalert about certain somatic functions and systems. The symptoms may represent the sleep analogue of hypochondriasis or somatic delusion. Accordingly, they may indicate an important psychological call for help and the need for psychotherapeutic comprehension. The scope of evaluations of "normal" psychological status in patients with subjective DIMS has not as yet been sufficient to remove suspicion that emotional and cognitive pathologies would be more fully revealed given more probing psychological exploration.

An explanation seems to be apparent in at least some of the elderly population who have subjective DIMS complaints without objective findings. These patients are sure they have insomnia because they believe they should be sleeping as much as in former years. Their appraisal of inadequate current sleep is not in line with expected sleep quantities for their age group, and this viewpoint requires adjustment.

Differential diagnosis: Individuals with subjective DIMS complaints without objective findings must be differentiated from patients with DIMS associated with atypical polysomnographic features (A.8.b) and persistent psychophysiological DIMS (A.1.b). Frequently, patients with the latter two DIMS also show objectively good sleep when being studied polysomnographically, but unlike the subjective DIMS patient, they are subjectively aware of sleeping better—when they do—in the laboratory; that is, their subjective sleep assessments usually match the quality of the sleep recording. In subjective DIMS without objective findings, though laboratory sleep is polysomnographically adequate, the patient *insists* that he is not sleeping well in the laboratory, just as he is certain his sleep is poor at home. Recordings at home have demonstrated adequate sleep there as well by polysomnographic standards.

It is clearly necessary to separate the subjective DIMS patients from malingerers who claim they sleep poorly in order to obtain drugs and for other reasons, perhaps attention. Most subjective DIMS complainers (without objective findings) who present themselves to their doctors with assertions of severe insomnia, are prescribed sleep medications because their subjective insomnia is accepted at face value.

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B. DOES: Disorders of Excessive Somnolence

The disorders of excessive somnolence (DOES) are a varied group of functional and organic conditions in which the chief symptoms may include inappropriate and undesirable sleepiness during waking hours, decreased cognitive and motor performance, excessive tendency to sleep, unavoidable napping, increase in total 24 hour sleep, and difficulty in achievement of full arousal on awakening. The diagnosis of DOES should generally be reserved for patients with complaints of sleepiness who have a manifest or demonstrable tendency to fall asleep quickly in the waking state when sedentary or who have sleep "attacks." This type of symptomatology should not be confused with a complaint—often *called* sleepiness—that actually describes physical tiredness and loss of mental alertness without an increase in sleep behavior. The latter are awake state symptoms frequently accompanying DIMS.

Among the DOES conditions, the intermittent DOES (periodic) syndromes are the least well understood. From the reports of Kleine-Levin syndrome (B.9.a.i) in the literature, it is not clear that the patients represent one diagnostic entity. That there is a strictly periodic, menstrual-associated DOES syndrome (B.9.a.ii) is well established. The published reports describing sleep drunkenness (B.9.c) leave considerable doubt as to the singularity of the condition. Insufficient sleep (B.9.b) and subjective DOES complaint without objective findings (B.10.b) are new diagnoses that are not based on a great number of cases. The classification committee decided that diagnosticians should have access to such entries because patients illustrating these situations do appear. However, there is no certainty that the need for these diagnostic designations will stand the test of time. Another justification for B.10.b is that it is the DOES analogue of subjective DIMS complaint without objective findings (A.9.b). It is already apparent that undocumented sleepiness is a much less frequent complaint.

It should be noted that psychophysiological DOES (B.1) and psychiatric disorders DOES (B.2) are not classified ahead of other diagnoses in the DOES group to indicate a high frequency of occurrence, but rather to maintain parallelism with the same DIMS diagnoses (A.1 and A.2). The latter do appear at the beginning of the DIMS listing because of their expected high incidence.

As mentioned also in the prefatory note to Section A, two conditions are entered in both the DIMS and DOES groupings because of different symptom presentations: sleep apnea syndrome (A.4.a and B.4.a) and sleep-related (nocturnal) myoclonus and "restless legs" (A.5 and B.5).

B. 1. Psychophysiological

a. Transient and Situational

Key words and phrases: transient reaction, acute stress, episodic, intermittent, situational, transient anxiety, brief reactive depression, conservation-withdrawal, less than 3 weeks.

Essential features: *Transient and situational DOES consists of a disruption of the normal sleep–wake pattern marked by excessive difficulty remaining awake and a tendency to remain in bed unusually long periods or to return to bed frequently during the day to nap. This change is experienced suddenly in response to an identifiable recent life change, conflict, or loss.*

Though some individuals may increase total sleep somewhat at the onset of such a reaction, actual extension of sleep over days has not been documented. The polysomnogram is normal in all respects. Sleep architecture does not change—i.e., REMS–NREMS stage durations, proportions, and periodicities, as well as sleep spindle and K complex counts, show no systematic shift from expectable base lines. Nevertheless, the individual may complain of sleepiness, certainly of lassitude, and often seeks out the eyes-closed, recumbent position. To be classified as transient and situational DIMS, the disposition to sleep excessively may not last longer than 3 weeks following termination of the instigating circumstance.

Associated features and other information: Without exception, the individual with transient DOES can link the symptoms directly to a precipitating event, and only infrequently develops more serious psychiatric symptoms. Illness in one's family, death of a close friend or relative, marital separation or divorce, a job demotion or change—all may trigger a shift to reduced alertness and the tendency to withdraw into sleep. The course of this DOES is usually short, lasting from 1 or 2 days to 3 weeks. Full restoration of the previous sleep–wake pattern is observed with resumption of premorbid emotional functioning. Though short-lived, the reaction may happen intermittently.

Withdrawals into sleep may be infrequently observed in response to disappointment. It is possible to conceptualize the episodes as transitory depressed states, though not all patients who show this reaction appear depressed.

Internal challenges such as anger or fear can bring on CNS depression rather than arousal in some individuals; that is, the internal balance between alertness and somnolence may shift suddenly toward the latter. This may be observed in adults and children who respond to a challenge with a yawn. Overburdened college students frequently deny or escape from school requirements in sleep. Why certain people seem prone to increased sleepiness in the face of threat, rejection, or change, whereas most individuals have insomnia under such circumstances, is beyond the range of available data. Both psychological and biological differences between individuals must be considered as possibilities.

Some psychological observers believe that excessive sleepers have "passive" makeups, tend to worry, and withdraw from their own aggressive feelings. It must also be noted that a sleep reaction can be adaptive rather than maladaptive in certain situations. Sleep has been proposed by some to function in part in the service of energy conservation.

Differential diagnosis: The diagnostic differentiation of this disorder from a major or minor affective disorder, or an anxiety disorder, with DOES may be difficult to establish when the patient is first seen (see B.2.a,b). Patients in the process of developing the more major reactions initially present a similar clinical picture but usually can be identified by predisposing factors for psychiatric illness.

Psychophysiological reactions to anxiety-inducing treatments for medical disease and hospitalization are associated with transient DIMS (A.1.a). However, transient DOES also may be found, albeit more rarely, as a psychological reaction to being treated for or recovering from nonpsychiatric illnesses. Somatic sources of somnolence must be ruled out in such instances (see B.8).

Drug abusers (see B.3.a) commonly present a history similar to transient DOES in an effort to obtain stimulant agents.

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B. 1. b. Persistent

Key words and phrases: persistent, conditioned reinforcement of sleepiness, chronic somatized tension–fatigue, chronic stress, greater than 3 weeks.

Essential features: *Persistent psychophysiological DOES applies to a chronic disposition to weariness, excessive sleeping, bedrest, and daytime napping when confronting stress–tension and when coping capacities are overwhelmed.*

This condition is rare and has uncertain clinical demarcations. It may be observed in persistently stressed persons lacking vigorous powers of adaptation or who have lost a clear sense of purpose. The condition may represent the extension of a transient or situational DOES (see B.1.a): a protracted somnolent response to perceived threat or internal aggression. There is no objective evidence that the daily accumulations of sleep are actually excessive.

Additional information and other features: Occult anxiety is often at the base of a chronic disturbance in initiating or maintaining sleep (see persistent psychophysiological DIMS, A.1.b), but unremitting tension–anxiety may lead to listlessness, indifference, and weariness. Some individuals who repeatedly experience a sense of fatigue when encountering challenges are gradually conditioned to develop an established, self-nourishing, psychological defense of tiredness. This may be expressed as a physical slowing down in the face of seemingly unresolvable reality problems, environmental circumstances that leave little room for hope, or stress when it exceeds the point of endurance. Such fatigue-prone patients learn to "take to bed."

Many of the patients to whom the traditional term of "neurasthenia" has been applied fit the pattern described here (though others complain of sleeplessness and exhaustion). A likely complication of the long periods of bedrest and numerous daytime naps is the development of a disorder of the sleep-wake schedule—irregular sleep-wake pattern, C.2.e—in which sleepiness and insomnia both appear because of the disintegration of a regular biological phase rhythm.

This disorder may be a variant of chronic despair, or mild depression, that is masked in a somatically expressed form. It may also represent a kind of "nonre-

storative sleep," leading to continuous efforts to obtain more sleep. A tendency exists for the patients to be concerned about somatic illnesses and symptoms which are associated with few actual findings. The taking of sedatives for "nerves" adds to the DOES symptomatology.

Differential diagnosis: An effort should be made to distinguish this condition from chronic, mild, bipolar affective disorder (depressed) in which hypersomnia is common. Mild toxic and allergic reactions, as well as reactions to drugs, must also be separated (see B.8).

Before concluding that a patient has persistent psychophysiological DOES, careful determination needs to be made that sleep is not covertly disturbed at night by another cause (see section A, DIMS). For example, sleep-related (nocturnal) myoclonus (B.5.a) may present this way.

Drug abusers, with an interest in obtaining prescriptions for stimulants, may also give a history of persistent DOES symptoms.

These patients are not clearly differentiable from cases of subjective DOES complaint without objective findings (B.10.b), except that they have more manifestly negative, despairing outlooks, or are under considerable stress–tension.

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B. 2. *associated with*

Psychiatric Disorders

a. Affective Disorders

Key words and phrases: bipolar depression, manic–depressive, major depression, dysthymic disorder.

Essential features: *DOES associated with affective disorders is found in both major affective and other depressive (cyclothymic, dysthymic) syndromes. Excessive daytime sleepiness may be reported in the initial stages of many mild depressive disorders and characteristically in the depressed phase of the bipolar affective syndrome. Sleepiness also may be observed to fluctuate with intermittent affective lowering as exemplified in the shifting moods and DOES in cyclothymic personality.*

The disturbance is subjectively described as increased sleepiness during the day with a proclivity for napping and prolonged sleep at night. This type of disturbance is not found in major or less severe depressions of nonbipolar type. In the bipolar depressions, the severity of the sleep disturbance is correlated with severity of the affective episode. Despite the fact that bipolar depressed patients are in EEG-confirmed sleep during 95% of bedtime (and do not complain of waking), they awaken feeling unrefreshed.

In major bipolar depression, polysomnography reveals a short REM sleep latency as well as reduced NREM sleep stages 3/4. This pattern is most commonly found in middle-aged depressed individuals, but it is also a characteristic feature in

young adults with bipolar depressions, and even in childhood and adolescent depression. This pattern does not apply to patients with secondary depression (see A.2.a).

Associated features and differential diagnosis: Because of the high incidence of somatic symptomatology—including disturbances in appetite (often an increase associated with weight gain), reduced concentration, and increased fatigue—affective disorders with hypersomnia must be differentiated carefully from hypersomnic conditions having metabolic and drug etiologies (see B.8). Also, toxic causes of DOES and organic mental disorders should be separated (see B.8). The documented severity of the depression and the presence of vegetative symptoms of depression help to differentiate this condition from persistent psychophysiological DOES (B.1.b).

Other causes of DOES with a history of sleepiness and depression should be considered. These, such as narcolepsy (B.6), sleep-induced respiratory impairment DOES syndrome (B.4.a,b), sleep-related (nocturnal) myoclonus (B.5), and the intermittent DOES (periodic) syndromes—sometimes menstrually associated (B.9.a.i,ii)—must also be ruled out by detailed history and polysomnography.

The use of drugs and alcohol, which is common in this clinical population, may affect both the clinical presentation and the type and degree of sleep disturbance observed in the patient.

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B. 2. b. Other Functional Disorders

Key words and phrases: "neurotic" personality, personality disorder, dissociative disorder, borderline, hypochondriacal, conversion, schizophrenia.

Essential features and other information: *DOES associated with other disorders may be found in categorical, nonaffective, symptom and personality disorders such as the dissociative somatoform, fugue, amnesia, and borderline states, and the schizophrenic disorders.*

Patients at all ages past adolescence may manifest sleepiness and nap behavior in combination with psychiatric conditions (sleepy children with psychological difficulties generally are depressed, B.2.a). Careful psychiatric examination is required to determine the accurate psychiatric classification. These symptoms are not evidently associated with actual net 24 hour extensions of sleep.

Polysomnographic monitoring and a detailed sleep history are required to rule out the presence of an inapparent DOES of different etiology, particularly drug-induced, coexisting with the psychiatric symptoms.

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B. 3. *associated with*

Use of Drugs and Alcohol

a. Tolerance to or Withdrawal from CNS Stimulants

Key words and phrases: CNS stimulants, tolerance, withdrawal, amphetamines, "uppers," caffeine, morning withdrawal.

Essential features: *Tolerance to or withdrawal from CNS stimulants is diagnosed when an individual has become physiologically dependent or psychologically habituated to stimulant drugs such as methylphenidate, amphetamines, and caffeine, and now presents with daytime sleepiness, frequent napping, difficulty with arousal from sleep, morning drowsiness, and 10 hours or longer nocturnal sleep periods (for DIMS with these agents, see A.3.b).*

Patients commence use of the agents or have them prescribed in order to counteract daytime DOES symptoms, fatigue, and depression (see sustained use of CNS stimulants, under DIMS, A.3.b). As indicated in that discussion, the patient, expectant of elevation of mood and relief of tiredness or sleepiness, may not be prepared for the countervailing "low" and somnolence experienced when the drug wears off. Nevertheless, with the insidious growth of tolerance, the effect and duration of action of a given stimulant dosage shrinks. "Miniwithdrawals," bringing sleepiness and "crashes," may take place repeatedly when doses of stimulant wear off. The confirmed addict is knowledgeable of tolerance and has become painfully aware of the rapid onset of somnolence that follows reduction or elimination of drugs. However, the amateur stimulant taker becoming tolerant to his drug—the mirror image of the tolerant and now insomniac chronic user of sleeping pills (see A.3.a)—is concerned as he paradoxically becomes sleepy and depressed despite taking arousing agents. Even with planned drug withdrawal of CNS stimulants, the patient may not anticipate the degree of somnolence encountered.

Daytime complications, particularly from amphetamine-type drugs, include irritability, automatic behaviors and amnesiac episodes, blackouts, rapid descents into depressed moods, paranoid thinking, disturbances in autonomic functioning, and weight loss. Raising the dose of stimulant temporarily relieves these symptoms but in turn sets the stage for renewed and worsened manifestations of withdrawal. The patient may feel hopelessly dependent on the stimulant drugs (and also, perhaps, on sleeping pills at night to overcome arousal effects of late-ingested analeptics) and increasingly incapable of functioning competitively at work.

Excessive use of caffeine beverages (more than 10 cups daily) must be included in this disorder because of progressive dependence on caffeine as tolerance develops. Probably a widespread and easily overlooked expression of morning drowsiness (due to withdrawal of caffeine) is the daily requirement felt by the average coffee consumer to have one to three cups immediately on arising in order to "wake up."

Individuals with other causes of DOES, particularly narcolepsy (B.6), are placed on CNS stimulants for therapeutic purposes. As tolerance develops and dosages are raised, DOES symptoms reappear and worsen. Some of the worsening is due to secondary DOES symptoms accruing from drug tolerance.

Many of the patients who overuse CNS stimulants have predisposing personalities (e.g., schizoid, sociopathic) or psychoneurotic conditions that render them vulnerable to drug effects.

Differential diagnosis: The sleepy, either emotionally troubled or depressed patient (described here), spending long periods in bed, may be confounded with the patient who has a psychiatric symptom, personality, or depressive disorder (see A.2.a,b) for which he is taking stimulant agents to achieve mood elevation. A history of why the drug was originally instituted and an evaluation of the patient's symptoms after complete withdrawal both assist diagnostic separation of the basic disorder.

Differentiation from other possible underlying DOES conditions is undertaken by withdrawal of the patient from all drugs, followed by assessment of symptoms and polysomnographic study. Patients who are subsequently discovered to have depression, or other causes of DOES underlying stimulant ingestion, should be classified in terms of the underlying disorder in addition to classification here.

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B. 3. b. Sustained Use of CNS Depressants

Key words and phrases: CNS depressants, sedatives, hypnotics, tranquilizers, "downers," alcohol, direct effect of agent.

Essential features: *Sustained use of CNS depressants associated with DOES is diagnosed when daytime excessive sleepiness and napping may be attributed to the use of CNS depressants or to other drugs that have a sedating effect (for discussion of these agents relative to the complaint of insomnia, see DIMS, A.3.a,c).*

In addition to the addicted individual's daily use of opiates, barbiturates, and other sleep-inducing agents for somnolent effects, patients—particularly the elderly—sometimes develop DOES symptoms while employing drugs for therapeutic purposes in moderate and high dosages, e.g., anti-insomnia (sleeping pill) and certain antiseizure and antihypertensive agents; major and minor tranquilizers; tricyclic antidepressants; contraceptive pills; antihistamines; beta-adrenergic blockers; muscle relaxants; and alcohol.

Symptoms may include grogginess by day, depression, irritability, restlessness, shakiness, agitation, automatic behaviors, amnesiac episodes, and paranoid thinking. If the medications are taken in the evening, sleep time also may be excessive at night. The polysomnogram reveals a reduction in sleep stage REM and an increase in deep NREM (stages 3/4) sleep with acute or intermittent drug intake and a reduction in both under circumstances of continuous drug use.

Associated features and other information: Some patients intentionally, or without conscious awareness, use drugs to erect anxiety-limiting, social and interpersonal barriers in response to chronic, stressful situations. They may not recognize the soporific qualities of the agents, and how dependent on and adapted to them they become. Eventually, social and marital problems, as well as occupational disabilities, may arise. Such patients do not differ from the individuals classified under both transient and persistent psychophysiological DOES (B.2.a,b) (who spontaneously and involuntarily use sleep as a defensive withdrawal from stress and threat) except in terms of the use of drugs to provide the same result. A patient who shows withdrawal and social isolation as a psychological defense may be discovered only if studied carefully when he presents the use of sedative agents associated with a history of somnolence.

Patients are often initiated to drug-induced somnolence following treatments or hospitalization for medical conditions (see B.8). Use of very large bedtime doses of CNS depressants may lead to alveolar hypoventilation (DOES) syndrome (B.4.b).

Differential diagnosis: Little difficulty generally exists in differentiating this condition from other DOES based on the patient's account of his employment of drugs and medications. Reassessment of the patient after withdrawal from medication will serve to rule out a masked source of DOES.

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B. 4. *associated with*
Sleep-induced Respiratory Impairment
a. Sleep Apnea DOES Syndrome

Key words and phrases: sleep apnea, obstructive apnea, obstructive upper airway apnea, mixed central and obstructive apnea, snoring, micrognathia, tonsillar and adenoid hypertrophy, partial obstruction, normal pulmonary function while awake.

Essential features: *Sleep apnea (DOES) syndrome is a potentially lethal condition characterized by multiple obstructive or mixed apneas during sleep associated with repetitive episodes of inordinately loud snoring and excessive daytime sleepiness [for a description of sleep apnea (DIMS) syndrome, see A.4.a].*

The characteristic snoring pattern noted with this syndrome is one in which inspiratory snores gradually increase when obstruction of the upper airway de-

velops; a loud, choking inspiratory gasp then occurs as the patient's respiratory efforts succeed in overcoming the occlusion. The aroused patient usually is aware of neither the breathing difficulty nor of the numerous accompanying body movements that at times violently disturb his sleep, and reports from a sleep observer are necessary for an adequate description of the patient's sleep breathing pattern. This syndrome is often called obstructive or upper airway sleep apnea.

Excessive daytime sleepiness is typically the presenting complaint, though there is considerable variation in the intensity of somnolence. The patient may minimize the degree of drowsiness or deny any impairment of alertness. The sleep attacks tend to be prolonged (more than 1 hour) and unrefreshing. Many patients report blackouts, disorientation, and periods of automatic behavior associated with amnesia. Once asleep, the patient is usually very difficult to arouse and is often unresponsive even to painful stimulation. Such patients typically exhibit postdormital disorientation, "fogginess," dulling of sensorium, and incoordinations—comprising a disorder of initiating wakefulness, secondary "sleep drunkenness." The most prominent presenting symptom, particularly in children, may be the reappearance of enuresis after complete toilet training; in these cases, learning difficulties, decreased school performance, and hyperactivity interrupted by hypersomnolence are often seen, especially in older children.

Cardiac arrhythmias are frequently observed during sleep and range from premature ventricular contractions and ventricular tachycardia to atrioventricular block and sinus arrest. The sleep apnea DOES syndrome carries an increased risk of sudden death during sleep, usually due to arrhythmia. Sleep apnea has been implicated in some cases of sudden infant death syndrome, though documentation is lacking.

Apneic episodes during sleep are defined as cessations of air flow at nose and mouth lasting 10 seconds or longer and can be readily documented by polysomnographic recordings. They are associated with brief EEG arousals, usually without full awakening, prior to resumption of breathing. Repeated K complexes may occur toward the end of individual NREM sleep apneic periods. Variation in night-to-night frequency of apneic pauses exists in many patients, with increased frequency appearing to follow upper respiratory infections or use of sedating drugs or alcohol. NREM stages 3/4 sleep is either absent or much reduced even in children, except the very young (who have relatively few sleep apneic episodes). The preponderance of apneic episodes within stages REM or NREM 1 and 2 sleep is subject to considerable individual variation. The occurrence of REM sleep periods at sleep onset is found in some patients, undoubtedly due to chronic REM sleep deprivation caused by the frequent arousals.

Three types of sleep apnea can be distinguished by polysomnography: (1) *central apnea,* characterized by cessation of air flow resulting from termination of respiratory effort; (2) *obstructive, or upper airway, apnea,* characterized by cessation of air flow despite persistent respiratory efforts; (3) *mixed central and obstructive apnea,* defined by the occurrence of a central phase (no air flow and no respiratory effort) followed by an obstructive phase in the latter part of the episode. The frequency of apneic episodes per night may number in the hundreds, with all three types of apnea usually present, but in the vast majority of patients the obstructive type predominates, and always with a DOES syndrome. The

severity of consequent hypercapnia and hypoxemia is influenced by the duration, as well as by the type, of apneic episode. It is most marked in the obstructive and mixed types, which lead to DOES.

Associated features and other information: Many patients report diffuse headaches lasting several hours after waking, thus adding to their general malaise. Additional symptoms include development of obesity and drenching night sweats with heat intolerance. Some individuals awaken as a consequence of their apneic pauses and may complain of nocturnal chest discomfort, a choking or suffocating sensation, or vague anxiety. Such patients often demonstrate less marked impairment of alertness, and even total absence of daytime drowsiness (see sleep apnea DIMS syndrome, A.4.a).

Although a modest degree of obesity is often affiliated with sleep apnea DOES syndrome, many patients with this disorder are not significantly overweight, and morbid obesity is present only in a minority. About 5% fit the classical description of the obese Pickwickian patient. Many patients with severe sleep apnea DOES syndrome demonstrate a short, thick neck, with or without obesity. A relatively small proportion of sleep apnea patients exhibit actual anatomic abnormalities of the upper airway. Many individuals have an unusually brisk pharyngeal, or "gag," reflex, though complete absence of the latter has been noted in a few cases. Waking respiratory functions are usually within normal limits, but a rapid deterioration of values is noted when the patient becomes drowsy. Hypertension is present in approximately 40% of cases of sleep apnea DOES syndrome at the time that the disorder is diagnosed. Clinical evidence of either or both right- and left-sided heart failure may be evident in severe cases.

The course of this disorder is generally progressive and chronic, eventually leading in severe cases to profound impairment and life-threatening complications. Appropriate treatment can significantly control the apneic episodes and may even reverse cardiovascular complications. Hemodynamic and cardiac abnormalities induced by the repetitive sleep apneic episodes only gradually affect the waking period. The time course of the illness depends on a number of factors such as the severity and type of apneic episodes, the patient's age, gender, and the presence of obesity or other anatomic aggravating factors. Although excessive daytime sleepiness is most often the presenting complaint, the sequence in which symptoms appear may vary. The appearance of secondary cardiac failure often heralds the onset of fulminant worsening of this condition, with rapid deterioration ensuing in many such cases.

Although sleep apnea occurs at all ages, sparing neither infants nor the elderly, more than half of the patients are 40 years of age or older when first diagnosed. In most adult patients, however, abnormal respiration during sleep appears to antedate the profound daytime sleepiness by many years.

Daytime grogginess is frequently incapacitating and commonly results in job loss, self-injury, marital and family problems, and poor school performance. Patients are labeled as lazy or as having a primary psychiatric disorder. Secondary depression, anxiety, irritability, and even profound despair are not uncommon. Patients occasionally note loss of libido and erectile potency. Sometimes a complaint of impotency without daytime drowsiness is the presenting complaint.

Two endocrine disorders—hypothyroidism and acromegaly—have been linked

with obstructive sleep apnea as a consequence of secondary mechanical airway problems. Obstructive sleep apnea secondary to naso-pharyngeal polyps or lymphomatous involvement of Waldeyer's ring also has been described. In most cases, no anatomic abnormality exists, and the etiology of the condition probably resides in the CNS. However, in children, a frequent cause of daytime somnolence and sometimes of intellectual dullness is sleep apnea (DOES) syndrome resulting from reversible nasal or pharyngeal obstructions such as hypertrophied tonsils and adenoids.

The prevalence of the sleep apnea syndrome is unknown, but it appears to be surprisingly common. Twenty to sixty percent of patients with excessive drowsiness evaluated at different sleep disorders centers have sleep apnea.

In cases of sleep apnea syndrome not associated with classic narcolepsy, men outnumber women by a ratio of greater than 30:1. A strong hereditary influence is suggested at least in some cases of sleep apnea syndrome.

Variants of sleep apnea (DOES) syndrome: (1) *Incomplete obstruction of the airway*. The patient may present partial or near complete obstruction of the airway, with some persistence of air exchange at the level of either the mouth and nostril or both. These events have been called "hypopnea" (actually obstructive hypopnea). During partial obstruction, oxygen desaturation and hypercapnia are usually not seen, but marked cyclic arrhythmias are observed in association with repetitive incomplete obstructive events. Measurement of endoesophageal pressure demonstrates increases. Femoral arterial pressure and systemic pressure increase progressively if partial obstructions are closely repetitive. Fiberoptic scope visualizations coupled with EMG studies of the oropharyngeal muscles have clearly visualized these partial obstructions. The mechanisms responsible appear to be similar to those leading to complete obstruction. (2) *Heavy snoring*. Heavy snoring causes partial obstruction of the airway during sleep. This subobstruction is similar to, but usually milder than, the partial obstructions described in the previous paragraph. (3) *Decrease in diaphragmatic contractions*. Unfortunately, the term "hypopnea" is also used in a second sense, in this case referring to a progressive decrease in air flow (as in incomplete obstruction above) but *secondary to decreased diaphragmatic motor strength*, and considered equivalent to a central apnea (see A.4.a). During such events, measurements of endoesophageal pressure demonstrate a decrease compared to previous normal values. Oxygen desaturation and hypercapnia occur, but changes in pulmonary and systemic arterial pressures are usually of lesser intensity than during "obstructive hypopnea" of similar duration.

Differential diagnosis: Sleep apnea DOES syndrome can be differentiated from narcolepsy (B.6) by persistent and pervasive sleepiness unrelieved by short refreshing naps, prolonged naps that leave the patient groggy, frequent and characteristic snoring, flailing and motor restlessness during sleep, postdormital headaches, absence of cataplexy, and more variable age of onset. Sleep apnea DOES syndrome can be differentiated without difficulty from intermittent DOES (periodic) syndrome (B.9.a) by the former's lack of periodicity, progressive chronic course, and developing complications. Alveolar hypoventilation (DOES) syndrome (B.4.b) can be differentiated from sleep apnea DOES syndrome by the respiratory pattern.

Other chronic DOES conditions that may need to be ruled out by appropriate laboratory and neurologic tests include DOES secondary to drug dependence (see B.3.a,b), intracranial lesions (particularly diencephalic tumor), and other neurological disorders such as nocturnal seizures, acromegaly and myotonic dystrophy, as well as hypothyroidism and Prader-Willi syndrome (B.8).

In a minority of cases during periods of sleep apnea, awakenings that are accompanied by sensations of choking, chest discomfort, or suffocation may be confused with nocturnal angina, early congestive heart failure (D.4.k), hysterical episodes (globus hystericus) (A.2.a), or insomnia (see A.4). Sleep-related gastroesophageal reflux (D.4.l) and sleep-related abnormal swallowing syndrome (D.4.i) can also produce similar symptoms.

Depressive episodes associated with DOES (B.2.a) can be differentiated by psychiatric interview and psychometric testing. Polysomnographic recordings with appropriate respiratory monitoring are mandatory for characterization and documentation of sleep apnea severity, and should be performed particularly in cases of DOES in which adequate observations of nocturnal respirations are lacking.

Cheyne-Stokes respiration is often mistaken for obstructive sleep apnea. It can be aggravated or induced by sleep and, in turn, may be associated with mild to marked DOES. Cheyne-Stokes respiration should be classified under other medical, toxic, and environmental conditions (B.8).

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B. 4. b. Alveolar Hypoventilation DOES Syndrome

Key words and phrases: hypoventilation, noncentral and central factors, myotonic dystrophy, narcolepsy, poliomyelitis, CNS neoplasm, cervical cord lesions, obesity, normal pulmonary function while awake.

Essential features and other information: *Alveolar hypoventilation (DOES) syndrome consists of several conditions marked by impaired ventilation in which the respiratory abnormality appears or greatly worsens only during sleep and in which significant apneic pauses are not present. The ventilatory dysfunction is characterized by inadequate tidal volume or respiratory rate during sleep [for alveolar hypoventilation (DIMS) syndrome, see A.4.b].*

Alveolar hypoventilation may be seen as sleep progresses regardless of sleep stage or can be specifically sleep-stage linked. The pathogenesis of this condition varies. Sleep-induced hypoventilation can arise from factors external to or within the CNS.

A major role of nonneurological factors in alveolar hypoventilation (DOES) syndrome such as obesity and thoracoskeletal deformities may be documented in some cases.

Individual cases have a demonstrated impaired ventilatory drive without apneas as a feature of myotonic dystrophy, narcolepsy, poliomyelitis, other encephalitides, CNS neoplasia, ventrolateral cervical spinal cord lesions, and other primary CNS disorders.

Differential diagnosis: A careful drug history and appropriate toxicological studies may prove necessary to exclude alveolar hypoventilation (DOES) syndrome secondary to respiratory depressants. Repetitive partial occlusion of the upper airway may result in a syndrome of sleep-induced obstructive hypoventilation without true apneas and without clearly identified CNS or other etiologic factors and may be associated with severe degrees of hypoxia despite the persistence of minor degrees of air exchange. A more complete discussion of this syndrome is included in sleep apnea DOES syndrome (B.4.a) under variants of the syndrome.

For DOES conditions that require diagnostic differentiation from alveolar hypoventilation (DOES) syndrome, see differential diagnosis in B.4.a.

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B. 5. *associated with*

Sleep-related (Nocturnal) Myoclonus and "Restless Legs"

a. Sleep-related (Nocturnal) Myoclonus DOES Syndrome

Key words and phrases: nocturnal myoclonus, leg jerks, nonepileptic.

Essential features and other information: *Sleep-related (nocturnal) myoclonus DOES syndrome consists of highly stereotyped abrupt contractions of certain leg muscles during sleep (the same as in the detailed description in DIMS, A.5), except that the associated sleep disturbance gives rise to a complaint of excessive daytime sleepiness and, typically, little awareness of, or complaint offered, concerning the nocturnal arousals and leg jerks.*

Etiological mechanisms are unclear for either the associated complaint or the specific causes of the abnormal movement.

Differential diagnosis: It bears emphasizing that sleep-related (nocturnal) myoclonus is often occult and frequently presents only as a nondescript hypersomnolence before polysomnographic examination. With polysomnog-

raphy, its appearance—individually or in combination with and in other conditions (see A.5)—may be determined.

When sleep-related (nocturnal) myoclonus presents as excessive daytime somnolence, it should alert the diagnostician to the possibility of narcolepsy (B.6), in which patients occasionally have myoclonic activity of the legs. Also, in sleep apnea DOES syndrome (B.4.a), generalized body movements are often observed in association with the beginning of effective breathing. Additionally, the hyperactivity in bed of the obstructive sleep apnea patient may be confused with the leg twitches in sleep-related (nocturnal) myoclonus (see B.4.a). Inasmuch as sleep-related (nocturnal) myoclonus does not usually present with obligatory naps with or without dreaming, hypnagogic imagery, cataplexy, or gasping apneic respiration, it usually can be differentiated clinically from these two conditions.

Sleep-related (nocturnal) myoclonus DOES syndrome frequently mimics persistent psychophysiological (B.1.b) and drug-related (B.3) DOES. This is particularly so considering that few patients are without some psychological features of note or some history of drug use, respectively. Sleep-related (nocturnal) myoclonus DOES syndrome can also be periodic and remitting in subjective and polysomnographic presentation. Accordingly, the intermittent DOES syndromes (B.9.a) must be considered when such a clinical picture is encountered.

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B. 5. b. "Restless Legs" DOES Syndrome

This condition may present with complaints of DOES as well as DIMS (for a full description of the condition, see A.5.b).

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B. 6. Narcolepsy

Key words and phrases: narcolepsy–cataplexy syndrome; narcolepsy syndrome; symptom tetrad: sleepiness and irresistible sleep, cataplexy, sleep paralysis, and hypnagogic imagery; abnormal and dissociated facets of REM sleep; sleep-onset REM periods.

Essential features: *Narcolepsy is a syndrome consisting of excessive daytime sleepiness and abnormal manifestations of REM sleep. The latter include frequent sleep-onset REMS periods, which may be subjectively appreciated as hypnagogic hallucinations, and the dissociated REM sleep inhibitory processes, cataplexy and sleep paralysis. The appearance of REM sleep within 10 minutes of sleep onset is considered evidence for narcolepsy.*

Excessive daytime sleepiness is usually the first of the tetrad of symptoms of narcolepsy to appear. A history of cataplexy is a characteristic and unique feature of narcolepsy. Sleepiness may persist throughout the day or may be relieved by an obligatory nap that provides some refreshment. Sleep attacks usually occur at times when sleepiness is common such as after meals or in the late afternoon. These attacks may be irresistible and can occur while eating, riding a bicycle, actively conversing, and even during active sexual relations.

Associated features and other information: Cataplectic attacks may consist of either brief, almost imperceptible weakness of isolated muscle groups giving rise to jaw drop, head drop, facial sagging, weakening of the knees or loss of grip, or sudden paralysis of all skeletal muscles with a complete postural collapse. Consciousness is usually clear during brief episodes. However, cataplexy and sleep attacks can occur in combination, or cataplexy can grade smoothly into a full-blown REM sleep episode, and the patient may experience intense sleepiness and possibly hypnagogic hallucinations (i.e., REM sleep dream imagery) along with or succeeding the paralysis. Cataplectic episodes are almost always triggered by eruptive expressions of emotion such as laughter, exhilaration and anger. The frequency of occurrence varies widely from less than one a week in some patients to countless attacks in a single day in others.

Sleep paralysis and hypnagogic hallucinations, which occur separately or in combination in about half of the patient population, are often referred to as auxiliary symptoms. Sleep paralysis overcomes the patient either at the entry into or emergence from sleep. The patient is unable to move, but usually regains the use of his muscles after one or more minutes. Hypnagogic hallucinations are vivid, perceptual, dreamlike experiences occurring at sleep onset or on awakening. The accompanying affect is often fear or dread.

About half of narcoleptic patients report the frequent occurrence of memory disturbances or "blackouts" (lapses of memory) and automatic behaviors. Other symptoms include ptosis, blurred vision, and diplopia. Finally, many patients report that nocturnal sleep is fragmented by frequent awakenings.

Narcolepsy usually commences in the second decade. It is not unusual for a patient to consult several physicians before the diagnosis of narcolepsy is made or even considered.

The typical course of narcolepsy is that sleep attacks begin several years before cataplexy. Symptoms tend to remain constant once they appear. Only *11–14%* of

patients have all four of the classic tetrad. Impairment is mild to severe depending on the degree of daytime sleepiness and the frequency of sleep and cataplexy attacks. Narcolepsy is estimated to occur at the rate of 4 cases per 10,000 with no gender dominance. Relatives of narcolepsy index cases have a 60-fold greater risk of having the disease than individuals in the general population. Speculation about the etiology of narcolepsy in former years included the allegation that narcolepsy is a psychogenic disturbance. It is now clearly recognized as a pathophysiological CNS condition and documented to be inherited in dogs, which show the full narcolepsy–cataplexy syndrome.

Narcolepsy gives rise to many complications, among which are accidents, medication side effects, and habituation to stimulant drugs. Reactive depression is a frequent problem in narcoleptic patients. However, the paranoid thinking and personality changes commonly reported in narcolepsy are better linked to the CNS stimulants used to treat the condition.

Narcolepsy can be disabling and even in less severe cases eventuates in serious interpersonal, family, and social consequences. Patients note difficulty with marriage and occupation because of the sleepiness, sleep attacks, and cataplexy. They and others around them are often not sure about the degree of their voluntary control over their symptoms, particularly the sleepiness. Their capabilities in interactions with spouses, children, and employers are gravely compromised. Before the disease is recognized, the patients are often thought of as unmotivated, uninterested, hostile, or depressed by the people around them.

Differential diagnosis: The most characteristic polysomnographic feature of narcolepsy is the occurrence of sleep-onset REM periods. These may be demonstrated in multiple daytime naps or in a nocturnal sleep recording. Occasional sleep-onset NREM periods mean that multiple recordings may be required for a positive demonstration. Sleep-onset REMS periods may occur in some other conditions such as drug withdrawal (A.3.a, B.3.a), previous REM sleep deprivation (as in obstructive sleep apnea, A.4.a), alcoholism (A.3.a,d; A.6), psychotic depression with DIMS (A.2.b), and some sleep–wake schedule variations (see C). However, when these conditions are ruled out, the occurrence of sleep-onset REM periods is highly diagnostic of narcolepsy. Some objective measure of excessive daytime sleepiness may be helpful. The Multiple Sleep Latency Test and pupillography are both helpful in this regard.

Idiopathic CNS hypersomnolence (B.7) is without sleep-onset REMS periods or auxiliary symptoms, but often mimics the sleepiness symptoms of narcolepsy.

Narcolepsy must be differentiated from all other DOES conditions, particularly sleep apnea (B.4.a), which also presents with a history of sleep attacks, often described as obligatory but not quite as irresistible as in narcolepsy. Sleep apnea may be identified by a history of snoring and hypertension and by polysomnographic demonstration of cardiac arrhythmias and long pauses in respiration during sleep. It should be noted that narcolepsy–cataplexy patients may have an unusual prevalence of apneas during sleep (if only a few, code them under D.4.n; if many, consider a double condition and code A.4.a or B.4.a in addition to narcolepsy).

It may at times be necessary to distinguish hypothyroidism (B.8), epilepsy (D.4.b), hypoglycemia (B.8), fugue states (B.2.b), alcoholism (A.3.d; A.6), myasthenia gravis (B.8), multiple sclerosis (B.8), intracranial space-occupying le-

sions (B.8), hysterical trance states (B.2.b), depressions and neuroses (B.2.a,b), and schizophrenia (A.2.c; B.2.b) from narcolepsy either by history and appropriate laboratory investigations or both. Coma is easily differentiated from narcolepsy because narcoleptic naps and cataplectic attacks, which often merge into sleep attacks, are both easily reversible by stimulation of the patient, who then regains consciousness.

The distinction between narcolepsy and other causes of excessive daytime sleepiness can sometimes be difficult because of the variable order in which the several symptoms may appear. Cataplexy, sleep paralysis, and hypnagogic hallucinations may be manifested before or after the sleep attacks and excessive daytime sleepiness, which always eventually make their appearance. Familial sleep paralysis (see D.4.e) can be differentiated from narcolepsy because the sleep paralysis is isolated and sleep attacks never develop. Once a patient develops cataplexy as well as sleep attacks and excessive daytime sleepiness, the combination is sufficiently dissimilar from all other medical conditions that the diagnosis of narcolepsy is easily made.

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B. 7. Idiopathic CNS Hypersomnolence

Key words and phrases: NREM narcolepsy, harmonious hypersomnia, idiopathic DOES, familial type, isolated type, no true "sleep attacks," no cataplexy, sleep paralysis or hypnagogic imagery, subwakefulness syndrome.

Essential features: *Idiopathic CNS hypersomnolence is characterized by recurrent daytime sleepiness, but "sleep attacks" do not occur because the sleepiness is not as irresistable as in narcolepsy. Naps are lengthy, not refreshing, and preceded by long periods of drowsiness. If actual sleep is resisted, automatic behaviors occur due to "microsleeps." There is a familial and an isolated type of this condition.*

Patients complain of virtually constant sleepiness in idiopathic CNS hypersomnolence. Sleep latencies are usually very short in the daytime (Multiple Sleep Latency Test) as well as at bedtime. The majority of patients sense that they sleep very deeply through the night. They do not have the frequent disruptions of sleep that mark the nocturnal sleep structure in narcolepsy. Total nocturnal sleep time is often of long duration. The capacity to arouse may be normal, but many patients report great difficulty waking up and experience "sleep drunkenness" (see B.9.c for discussion of "sleep drunkenness" as a disorder).

Polysomnographic monitoring fails to uncover either sleep-onset REM periods or excessive apneas in sleep. In some cases, a high REM sleep percent is observed that is not characteristic of idiopathic CNS hypersomnolence and may represent a specific subsyndrome.

Associated features and other information: *The familial appearance of this disorder* is observed most frequently. Associated symptoms are common in the familial and less so in the *isolated type:* headaches are frequently noted and may be migrainous in quality, and fainting attacks (syncope) occur often in females. These may relate to the peripheral vascular complaints of the Raynaud's disease type quite often observed in this disorder. Accordingly, a parasympathetic dysfunction is suspected but not documented. Dopamine metabolism is reportedly abnormal in these patients. This syndrome is estimated to account for 12–15% of patients who bring a complaint of excessive daytime sleepiness to a sleep clinic.

The idiopathic CNS hypersomnolence syndrome has been described repeatedly in the scientific literature, but perhaps because of its various designations and disagreements about it constituting a unitary condition, it is relatively neglected by physicians and public health officials. The syndrome has been recognized for 100 years and has been named *independent, idiopathic,* or *NREM narcolepsy;* or *idiopathic, functional, mixed,* or *harmonious hypersomnia.* Often more disabling than narcolepsy, idiopathic CNS hypersomnolence does not respond to CNS stimulants such as the amphetamines and methylphenidate, as does the somnolence associated with narcolepsy and other types of DOES. In addition, such medications in patients with this disorder seem to exacerbate the associated symptoms of headache, migraine, nausea, and syncope. However, methysergide, which is of no assistance in narcolepsy, alleviates the primary symptoms of idiopathic CNS hypersomnia in a number of cases, implicating 5-hydroxytryptamine (serotonin) neurochemical systems in the etiology.

Differential diagnosis: Narcolepsy without auxiliary symptoms is the principal alternative diagnosis (B.6). Its polysomnographic feature, sleep-onset REMS periods, distinguishes the two entities, as does the broken nocturnal sleep of narcolepsy. To eliminate the complaint of somnolence associated with depression (see B.2.a), in which somatic symptoms are also reported, systematic sleep latency measurements demonstrate the much more rapid tendency to fall asleep in this condition.

Two *secondary* syndromes of daytime somnolence (which are classified in B.8) must be eliminated before diagnosing the isolated form of idiopathic CNS hypersomnia. In *communicating hydrocephalus,* daytime somnolence may be a revealing symptom of the early but progressive hydrocephalus in children and adults. Clinical features of hydrocephalus may be completely absent. Electroencephalogram, skull x-ray film, and computed tomography scan are necessary in order to eliminate this diagnosis; in *posttraumatic hypersomnolence,* 6–18 months after head trauma, patients may gradually develop severe daytime hypersomnolence, which may encompass all features of the isolated form of CNS hypersomnolence. Cerebrospinal fluid data in the posttraumatic condition, obtained with the probenecid test, may not be helpful in that they can demonstrate an abnormal homovanillic acid level. The response to stimulants is also frequently as negative,

and social disability as severe, as in idiopathic hypersomnolence. The *only* difference may be in long-term prognosis; posttraumatic cases infrequently improve over time, whereas no improvement is expected in idiopathic CNS hypersomnolence.

In a probably related condition, the *subwakefulness syndrome* (see Glossary), patients complain of fatigue and drowsiness, which lead them to sleep a great deal. However, one does not fall asleep against one's will in this condition, whereas one finally succumbs to sleep in idiopathic hypersomnolence. Polysomnography reportedly demonstrates sleep stage NREM 1 and, rarely, stage 2, but no stages 3/4 during daytime naps in the former.

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B. 8. *associated with*
Other Medical, Toxic, and Environmental Conditions

Key words and phrases: medical, neurological, diseases, toxic, environmental, nonspecific symptoms, parasomnias causing DOES.

Essential features: *This category covers the medical (including neurological), toxic, and environmental conditions that are invariably associated with excessive daytime somnolence. Additionally included are conditions having a significant association with DOES, though DOES is neither invariable nor necessarily the major or most relevant symptom or sign.*

The onset, time course, and termination of the somnolence classified here are tied to the stage of the related medical, neurological, toxic, or environmental condition. Alleviation of the basic condition brings about a reduction in the excessive somnolence, either immediately or gradually.

Relationship to other DIMS: Many medical, toxic, and environmental conditions are associated with DOES either as direct causes or because of the daytime consequences of sleep deprivation (see A.6). Certain other conditions qualify for the B.8 category but are coded elsewhere as unique entities [e.g., sleep-induced respiratory impairment (B.4); also B.5–7]. DOES associated with tolerance to or withdrawal from CNS stimulants (B.3.a) and sustained use of CNS depressants (B.3.b) are also coded elsewhere except for the toxic or paradoxical effects of the agents.

Any parasomnia (section D) that is responsible for clearly applied hypersomnolence should also be coded under this category (B.8) unless—as in the case of sleep-induced respiratory impairment (B.4) and sleep-related (nocturnal) myoclonus DOES syndrome (B.5.a)—provision has been made for explicit coding elsewhere in section B.

Conditions: A more detailed review of the medical (neurological), toxic, and environmental conditions affecting sleep and wakefulness is found in A.6. DOES is especially prominent in the following conditions: *endocrine and metabolic disorders* such as hypothyroidism, apathetic hyperthyroidism (usually observed in the elderly), diabetes, and hypoglycemia; *nutritional deficiency states; uremia; gross obesity; hypercapnia* (usually from chronic pulmonary disease); *liver failure; CNS disorders* such as brain tumors (especially tumors, as in the pineal, that impinge on the third ventricle and tumors of the posterior hypothalamus); *subarachnoid and subdural hemorrhage* and *raised intracranial pressure,* from any cause; *toxic encephalopathies; postencephalitic states; neurosyphilis; numerous infections,* fungal, bacterial, and viral (including encephalitis lethargica and trypanosomiasis); *degenerative states* such as multiple sclerosis and chronic brain syndromes; *CNS trauma;* miscellaneous *pyrexial states;* and *anemia.*

Daytime somnolence is a common, sometimes the only, symptom of progressive hydrocephalus in both children and adults. Gradual and eventually severe daytime somnolence can develop 6–18 months following head trauma (see differential diagnosis, B.7).

Cheyne-Stokes respirations may be worsened in sleep and lead to DOES. This condition should be classified here (not in sleep apnea DOES syndrome, B.4.a). This abnormal ventilatory pattern may arise from prolongation of circulation time, as in congestive heart failure, or from increased or decreased ventilatory response to carbon dioxide. It may also be seen with superimposed upper airway obstruction in the crescendo phase.

Sleep-induced hyperpnea from conditions such as metabolic acidosis, hepatic encephalopathy, restrictive lung disease, and chronic partial obstruction of the upper airway should also be classified in this category when it causes DOES.

Neurosurgical intervention in the treatment of Parkinsonism, intractable pain, psychiatric disorders, and other conditions can produce both hypersomnolence and abnormal sleep. The anatomical locations of the resulting CNS lesions are more important than their etiology in determining the effects on sleep and hypersomnolence.

Environmental conditions—for example, boredom, social isolation, physical confinement, and abnormal forced sleep-wake schedules—are probable common causes of DOES. These situations may be associated with stress and conditioning, in which case the disorder should be coded under persistent psychophysiological DOES (B.1.b).

Food and environmental allergies may be a significant cause of DOES, though the evidence is not conclusive.

The *normal aged* show a somewhat greater prevalence for daytime somnolence (and also for reduced nocturnal sleep) than other age groups. This may be a sign of cerebral deterioration. However, in the elderly—as in other conditions such as encephalitis and renal failure—DOES may actually be an expression of an associated abnormal sleep–wake rhythm (differentiate from C.2.b–e).

Differential diagnosis: *Fatigue, malaise, exhaustion, lethargy,* and *tiredness* are often mistakenly used synonymously for sleepiness, but they are, in fact,

different entities. Where possible a distinction should be attempted and is frequently critical to diagnosis. The former are common symptoms of many chronic systemic diseases and deficiency states, which in certain instances may be distinguished by weight loss, a sign much less typically associated with true DOES.

Signs of *disordered consciousness* such as obtundation, stupor, confusion, delirium, coma, intoxication, narcosis, and hyperventilation, as well as psychiatric states involving catatonia, fugue, hysteria, withdrawal, depression, and paranoid unresponsiveness, may resemble excessive somnolence but should be distinguished by neurological and mental state examinations.

Sleepiness caused by medical, toxic, and environmental factors must be distinguished from other causes of DOES, particularly psychologically related causes (B.1,2), and from persistent disturbances of the sleep–wake schedule (C.2).

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B. 9. *associated with*

Other DOES Conditions

a. Intermittent DOES (Periodic) Syndromes

i. Kleine-Levin Syndrome

Key words and phrases: Kleine-Levin, periodic hypersomnolence, periodic hypersomnia.

Essential features: *Kleine-Levin syndrome is a relatively rare con-*

dition consisting of recurrent periods of exceedingly prolonged sleep (from which the patient may be aroused) with intervening periods of normal sleep and alert waking. During the hypersomnic episodes, wakeful periods are usually marked by withdrawal from social contacts and a return to bed at the first opportunity. However, the patient may also display apathy, irritability, confusion, voracious eating, and loss of sexual inhibitions.

Kleine-Levin syndrome is generally ascribed to an intermittent, organic dysfunction in limbic or hypothalamic structures without known cause.

Associated features and other information: In this puzzling disorder, the disturbance in appetite occasionally may be expressed as anorexia rather than excessive food consumption. The disturbance in social interaction is associated with a range of bizarre behaviors, not only sexual exhibitionism and inappropriate advances, but also delusions and hallucinations, frank disorientation, memory impairment, incoherent speech, truculence, and excitation or depression. It must be emphasized that any of these behaviors, including particularly the megaphagia, may be absent in a given case. Unexplained fevers occur in a minority of patients. Metabolic disturbances include abnormalities in urinary ketosteroid excretion, and discrepant results in metapirone and ACTH tests of pituitary–adrenal function.

In most cases, neither metabolic nor psychiatric disturbances are evident during the intervening periods of normal alertness, though some individuals show persistence of lesser degrees of personality disorder during remissions. Mild to moderate EEG abnormalities have been described during the wakeful portions of the hypersomnolent periods—most often consisting of intermittent slowing (theta or delta, medium- or high-voltage activity in various locations)—which reportedly clear following termination of an "attack." Direct entry into REM sleep at sleep onset is occasionally observed.

In the typical patient, several attacks of hypersomnia—each lasting for several weeks—are experienced yearly. With few exceptions, the first attack occurs between ages 10 and 21. Rare instances of onset in the fifth decade of life have been reported. The disorder is typically self-limited, a spontaneous and enduring remission occurring in most cases before age 40. Usually, no residual abnormalities or complications are noted.

Physical or emotional stress and febrile illness may precipitate the periods of excessive sleep. A relationship to familial epilepsy has been suggested but not proven.

Kleine-Levin syndrome is uncommon, but its exact prevalence has not been determined. Only 100 cases suggesting this condition have been reported, and in many of these the diagnosis can only be accepted as provisional.

The diagnosis of this syndrome is usually reserved for males, but it may be diagnosed in females in whom the classic findings (particularly hyperphagia and hypersomnia) are not menstrual related (see B.9.a.ii).

Differential diagnosis: The Kleine-Levin syndrome should be reserved for cases in which periodic or intermittent episodes of somnolence are associated with most of the classical findings: inappropriate behaviors (especially sexual), relentless consumption of food, clouded sensorium, and nonspecific

metabolic and EEG abnormalities. Intervening remissions must be relatively free of disturbances of sleep and waking.

This disorder must be differentiated from neoplastic and inflammatory conditions of the diencephalon or limbic system (B.8). Although dilatation of the left temporal horn is found in a few cases, there is a relative absence of neuroradiological findings, attacks are characteristically self-limited in nature, and the cerebrospinal fluid is typically benign. Elevated levels of 5-hydroxyindoleacetic acid in spinal fluid have been noted in several cases. Temporal lobe epilepsy (D.4.b) may be assessed by EEG.

Primary diagnoses of manic–depressive illness, schizophrenia, dissociative disorder, drug intoxication, and metabolic encephalopathy should be considered and excluded by appropriate psychological and laboratory screening (see B.2.a; A.2.c; B.2.b; B.3.a,b; and B.8, respectively).

Disorders of the 24 hour sleep–wake schedule (see section C) can be differentiated from Kleine-Levin syndrome by the dramatic and frequently bizarre behaviors associated with the latter. Progressive hypersomnolence syndromes secondary to neoplastic, inflammatory, vascular, or toxic conditions of the brain should be classified under DOES, other medical, toxic, and environmental conditions (B.8).

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B. 9. a. ii. Menstrual-associated Syndrome

Key words and phrases: menstrual associated, periodic hypersomnia with menses.

Essential features and other information: *Menstrual-associated DOES syndrome is defined as marked hypersomnolence in a regular, temporal relationship with menstrual periods.*

Very few cases of this disorder have been polygraphically monitored, but evidence is available that shows sleep architecture to be relatively normal, though sleep stages REM and NREM 3/4 may be proportionately low. Some data exist that in nonhypersomnolent women, sleep stages 3/4 are consistently maximal in the premenstrual period when hormone levels are at their peak. Some behaviors during the hypersomnolent period may be bizarre and therefore reminiscent of Kleine-Levin syndrome (B.9.a.i). A search for consistent endocrine changes at the time of attacks has not as yet been rewarded, but suspicion continues that a hypothalamic etiology will eventually be uncovered.

Differential diagnosis: The invariant relationship between menses and the onset of excessive sleep renders menstrual-associated DOES syndrome distinct from other periodic DOES syndromes (see B.9.a.i., Kleine-Levin), including disorders of the 24 hour sleep–wake schedule (see section C). Care must be taken to establish that interattack intervals are characterized by relatively normal patterns of sleep and arousal so as to exclude this syndrome from the

episodic aggravation of *continuous* DOES syndromes that is at times referable to menstrual periods.

Periodic drowsiness secondary to the use of sedating analgesics (B.3.b) at times of menstrual discomfort should be differentiated by history.

Thorough neurological evaluation appears warranted in order to insure that observed symptoms do not represent episodic, abnormal discharges from mass lesions in temporal or limbic structures (B.8) occurring at times of menses.

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B. 9. b. Insufficient Sleep

Key words and phrases: insufficient sleep, voluntary curtailment, lack of awareness of cause of symptoms.

Essential features: *Insufficient sleep is defined as an earnest complaint of DOES and associated waking symptoms presented by an individual who gives evidence of persistently failing to obtain sufficient daily sleep needed to support alert wakefulness. The individual is voluntarily, but often unwittingly, chronically sleep deprived.*

Examination reveals no inability to fall asleep or remain asleep suitably. Polysomnography reveals normal sleep structure with the exception of an increase in deep NREM (stages 3/4) sleep and a high ratio and intensity of REM sleep when longer than recently obtained sleep durations are allowed in the laboratory.

Associated features and other information: The sleepy individual, depending on the chronicity of sleep loss, may develop secondary symptoms—e.g., irritability, difficulty in concentration, reduced vigilance, distractability, reduced motivation, depression, fatigue, restlessness, incoordination, malaise, loss of appetite, gastrointestinal disturbance, painful muscles, diplopia, and dry mouth. The secondary symptoms may become a focus of concern for the patient, serving further to obscure the primary cause of the difficulties, and leading to apprehension or depression concerning health status.

One example of this uncommon clinical presentation is the ambitious, hard-driving, factory worker who denies his true sleep needs in the service of working two jobs, sleeping 4½ hours nightly; another is the hospital guard who stays up late nightly with friends—they follow a culturally determined sleep pattern of 3 a.m.–11 a.m.—but has to be at work at 7 a.m. and develops fatigue, weight loss, and depression.

Sleep loss in the absence of factors that tip the CNS sleep–wake equilibrium toward arousal (such as anxiety, depression, or stimulant agents) inevitably leads to sleepiness. When individuals who are psychologically and somatically normal

obtain less sleep than they biologically require, they are "sleepy" the next day. This phenomenon is reinforced in the experience of most people and taken for granted.

Many circumstances militate against adequate sleep; for example, battlefield combat, preparation for school examinations and assignments, writing deadlines, and political campaigning. However, individuals engaged in such activities who are sleep deprived should not be classified here unless they are unaware that more sleep will be recuperative and seek help for their symptoms. Low intelligence, cultural factors, or psychological "denial" may dispose to the individual searching out causes other than the obvious one. If unchecked, this condition may lead to psychological difficulties, depression, and poor work performance.

Differential diagnosis: A clear and detailed history of current sleep pattern in relation to amounts of sleep routinely obtained in the past, currently desired, possible to achieve, and actually obtained is revealing. Evidence that the disparity between the need for sleep and the amount actually achieved is substantial and is *unappreciated by the patient in its significance* is diagnostic. However, a therapeutic trial of longer sleep must be shown to reverse the symptoms.

This easily overlooked but real syndrome must be differentiated from both persistent psychophysiological DOES (B.1.b) and DIMS (A.1.b), because somnolence in this condition may be coupled with tension and other psychophysiological symptoms. Other DOES conditions that cause unalloyed sleepiness are less likely to be confused. Irregular sleep–wake pattern (C.2.e) can cause similar symptoms, but the patient usually complains of "insomnia."

B. 9. c. Sleep Drunkenness

Key words and phrases: sleep drunkenness, may be a symptom of other DOES.

Essential features: *Sleep drunkenness is an abnormal form of awakening in which the lack of a clear sensorium in the transition from sleep to full wakefulness is prolonged and exaggerated. A confusion state develops that often leads to individual or social inconvenience and sometimes to forensically significant or frankly criminal acts. Essential to the diagnosis is the absence of sleep deprivation.*

Associated features and other information: Clarity of perception and rational judgment in the sleep-drunken state return later than the capacity to carry out more or less complex motor acts prompted by stimuli. Consequently, inappropriate behavior occurs. The affected individual acts irrationally or impulsively, possibly because the subject's understanding of external (and internal) stimuli is faulty or incomplete, or because the judgment that directs his actions is governed by motivations arising out of deep-seated wishes or fears. Such motivations, if undesirable, are suppressed, inhibited, or controlled by conscious faculties during full wakefulness.

Sleep drunkenness has been diagnosed only in adults to date, and the number of reported occurrences is few. There is a preponderance of males in the reports, but these data are far from convincing. The syndrome has an affinity for certain families.

Forced arousal at any point in sleep, but particularly from deep NREM sleep, favors sleep drunkenness. Sleep deprivation, physical exertion leading to tiredness, and hypnotic medication enhance the potential of an episode.

Differential diagnosis: There is some doubt that sleep drunkenness is a true pathological entity as opposed to being a major symptom of some other sleep disorder [e.g., sleep apnea DOES syndrome (B.4.a) or idiopathic CNS hypersomnolence (B.7)]. Relatively few cases have been described in recent years, and the classic description of the syndrome was made before a number of other sleep disorders were known. This question ultimately will be resolved by empirical case series data.

Differentiation from somnambulism (D.1) rests on the fact that somnambulism occurs during sleep, not after awakening, and motor responsiveness is slow and fumbling in somnambulism, whereas in sleep drunkenness it is quick and impulsive.

Hysterical dissociative states (see B.2.b) usually have readily discernible motivations. They tend to be repetitive and to fit smoothly into the context of events. Clinical and EEG evidence of epilepsy aids in the diagnosis of epileptic confusional state (see D.4.b) rather than sleep drunkenness. Toxic confusional states (see B.8) are usually more prolonged than sleep drunkenness; moreover, the consumption of the intoxicants may be demonstrable by history taking or by laboratory testing.

Finally, when stimulants (e.g., coffee) have been heavily and habitually ingested in the morning, sudden withdrawal may lead to a sleep drunkenness type of behavior (see B.3.a).

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B. 10. No DOES Abnormality

a. Long Sleeper

Key words and phrases: long sleeper, "healthy" hypersomniac, asymptomatic, one end of normal sleep duration continuum.

Essential features: *Long sleeper is the designation for an individual who consistently sleeps (and feels the "need" to sleep) substantially more in 24 hours than the conventional amount of sleep for one's age group. The sleep, though long, is normal in architecture and physiology. Sleep efficiency and sleep–wake schedule are normal. This pattern is without complaints about quality of sleep, daytime sleepiness, or difficulties with awake mood, motivation, and performance. Despite the occasional desire and attempts by the individual to sleep less, no true DOES exists. Long sleep appears to be at one end of the normal, individual sleep requirement continuum.*

A young adult long sleeper appears to require and regularly sleeps more than 9 hours out of 24. Some may sleep as long as 12–14 hours. Many long sleepers—

because of occupational needs—function with reasonable success on only 9 hours per day. Less than that total leads to daytime symptoms emblematic of insufficient sleep (e.g., sleepiness, reduction in alert cognitive efficiency). Like short sleepers (A.9.a), long sleepers have conventional absolute amounts of sleep stages 3/4, but stages REM and NREM 2 are somewhat higher than normal. The patient has no problem with time distortion or ability to be accurate about the quality of his sleep.

Associated features and other information: Though generally within the normal range of psychological functioning, long sleepers who have been studied (typically male subjects) appear to have characteristic personality features. They are higher on most scales (significantly on the social introversion scale) of the Minnesota Multiphasic Personality Inventory than short sleepers (A.9.a), with normal expected values falling generally between these two groups. When interviewed, long sleepers appear either mildly depressed or anxious. They are generally described as "worriers." Similar to short sleep individuals, long sleepers (who sleep more than 10–11 hours) seek assistance because of awkwardness in family and social relationships, and because they believe that they are psychologically or medically abnormal and that others will regard them as deviant.

Usually the long sleep pattern begins in childhood, is established by early adolescence, and endures for one's lifetime without evidence of early conditioning, later impairments, or complications. Long sleepers comprise only a small proportion of the population, probably no more than 2% of males between ages 20 and 35. The tendency to this pattern favors certain families and (unlike short sleep) females over males.

Demographic data from several population studies appear to link long sleep as well as short sleep to reduced life expectancy. This relationship probably has its source mainly in long total sleep time patterns associated with medical and sleep pathologies, not in long sleep as represented by "long sleeper." In other words, the survey studies from which such correlations are drawn were not able to explore the likelihood that root pathological causes are responsible for *both* the higher mortality ratios and the protracted sleep. The question of a causal connection between unconventional sleep durations that are not associated with pathological processes and shortened life expectancy must remain moot in advance of suitable data.

Differential diagnosis: Differentiating simple long sleepers from pathological DOES conditions that also may commence in adolescence [e.g., patients with narcolepsy (B.6), postviral sleeping sickness (see B.8), and other early causes of DOES] may be difficult. Correct diagnosis of long sleeper is made chiefly by exclusion of specific diagnostic features associated with the other conditions and by requirement of an absence of complaints concerning the quality of the individual's awake state functioning. Many pathological DOES have an acute onset, rarely show the stable sleep duration of the long sleeper, and have a demonstrable polysomnographic abnormality.

Essential to valid diagnosis of long sleeper is the consistency of the pattern, attested to by a carefully kept "sleep log." The pattern should be naturally greater than 9 hours on a virtually daily basis over a 2 week span, without reversion to

shorter sleep durations on work or school days. The decision to consult help should have nothing to do with symptoms of nonrestorative or disturbed sleep or complaints about napping or sleepiness during awake hours. The only complaint about the wake period from a long sleeper generally focuses on its curtailment.

It is possible, but not reportedly common, that a customary long sleeper may become temporarily or persistently insomniac. However, under such circumstances, a knotty situation may confront the diagnostician who is called on to evaluate a complaint of insomnia and daytime symptoms due to insufficient sleep in an individual who is sleeping perhaps 7–8 hours nightly. Information gained from a life history of sleep pattern and requirement, from evaluation of the patient's current psychological status, and from assessment of sleep fragmentation, despite a 7–8 hour sleep aggregate, may validly entitle a long sleeper to a diagnosis of DIMS associated with either situational (A.1.a), persistent psychophysiological (A.1.b), psychological symptom (A.2.a), or affective (A.2.b) factors.

A patient of this sort is not confused with a case of DIMS complaint without objective findings (A.9.b), because the former gives an accurate subjective assessment of recorded length and quality of sleep compared with a polysomnographic recording.

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B. 10. b. Subjective DOES Complaint without Objective Findings

Key words and phrases: subjective complaint of excessive sleepiness, not excessive sleep, sleep hypochondriasis, distorted use of term "sleepy," unknown etiology.

Essential features: *Subjective DOES complaint without objective finding is the designation for an honest complaint of excessive somnolence (sleepiness not sleep) that cannot be objectively verified by clinical examination, family observation, or objective tests of the tendency to fall asleep or to have "microsleeps."*

Typically, patients present a 1–5 year history of rather vague somnolence and compromised effectiveness in work and social activities. They complain of "sleepiness," not excessive, uncorroborated sleep. (Accordingly, this condition is not the exact physiological opposite of subjective DIMS complaint, A.9.b.) Physical and psychological evaluations are also normal, though some patients have mild obsessional or depressive features associated with the primary complaint. Symptoms often are noted at a point when responsibilities first necessitate regular hours of wakefulness and sleep. Reports by family members or friends are uneven in support of the self-appraisal of excessive somnolence. Nocturnal polysomnography is without EEG, respiratory, cardiac, or movement abnormalities. The

Multiple Sleep Latency Test reveals latencies that average well above the abnormally low range and no REM sleep onsets.

Associated features and other information: Patients in this category do not disclose an obvious motivation for seeking a confirmed diagnosis of their professed sleepiness. They present as passive, perplexed individuals. As yet, no obvious age or sex pattern has emerged. When confronted with the diagnostician's failure to confirm excessive somnolence, the patient passively accepts the findings without anger; notwithstanding, they ask for treatment. Developmental and family history are generally negative. The patients are much rarer than subjective DIMS (A.9.b).

Several possibilities should be mentioned in terms of this puzzling condition. It is difficult to measure sleepiness except as the tendency to fall asleep or to engage in "microsleeps." A subjective sense of drowsiness may be actual but is currently beyond confirmation. Some of these patients may, in addition, not be able to fall asleep easily under study conditions. Also, some individuals who complain of "sleepiness" may not be able to differentiate this feeling from tiredness, fatigue, weariness, as well as others. Yet another possibility is that some of these patients may have an unusual sensitivity to mildly irregular sleep and waking habits and may actually have a persistent but occult sleep–wake cycle disorder (C.2) with inefficient wake phases. Finally, the patient may be overconcerned with sleep (see A.9.b) and focuses in an anxious, distorted way on his *expected* state of alertness, concluding that he is sleepy. Such a sleep hypochondriasis would represent a need for psychological assistance.

Differential diagnosis: Subjective DOES complaint without objective findings is difficult to distinguish in the already uncertain differentiation between cases of persistent psychophysiological DOES (B.1.b) and DOES with depression (B.2.a). However, in the former condition, despair and negativity, or chronic stress–tension is prominent, whereas in B.2.a, clinical depression is evident.

It is essential to rule out undisclosed systemic illness (B.8), which may present with complaints of unremitting somnolence and lassitude. Incipient narcolepsy (B.6) or idiopathic CNS hypersomnolence (B.7) may present this way, though sleep latency tests have proven quite affirmative in even early cases of the latter two conditions.

If successful, careful regularization of the sleep–wake schedule and other habits should, over time, help separate subtle cases of irregular sleep–wake pattern (C.2.e).

As with subjective DIMS complaint without objective findings (A.9.b), malingerers seeking drugs may have an investment in providing the history described.

C. Disorders of the Sleep–Wake Schedule

The disorders of the sleep–wake schedule consist of an arbitrary collection of clinical syndromes, some exogenously instigated and others—like delayed sleep phase (C.2.b), advanced sleep phase (C.2.c), and non–24-hour sleep–wake schedule (C.2.d)—appearing to have an endogenous component. The conditions deserve to be treated as a separate group because of a shared, cardinal feature—every disorder represents one form or another of *initial misalignment* between sleep and wake as behaviors and the individual or societal circadian phase matrices in which they are usually contained.

According to the patient, the problem is that he cannot sleep when he wants, needs, or expects to. Correspondingly, the awake periods of the patient occur inappropriately (e.g., in the middle of the night) or are uncomfortable because of drowsiness. Despite the surface nature of the chief complaints, however, investigation of the symptoms in disorders of the sleep–wake schedule reveals no evidence of an abnormality in the *installation* or *maintenance* of the sleep and awake states, only an abnormality in the rhythm of their occurrence with respect to either the phase of the individual's inner physiological milieu or the phase of one's expected state of functioning in society. (The former factor is paramount in conditions C.1.a, C.1.b, C.2.a, and C.2.e; and the latter factor in C.2.b, c, and d.)

The reader's understanding of these conditions and the development of symptoms perhaps will be assisted by discussion here of an aspect of our biological functioning—the circadian periodicity of many biological processes—only assumed in the discussions of disorder.

The periodic patterning of physiological functions—an inheritance of the largely light–dark-dependent, basic rest–activity cycle that is characteristic of all living matter—is tied in humans to the repeated and regular clock-hour scheduling of the major sleep and awake periods in the 24 hour day. The other physiological functions seem to be recruited to the sleep–wake periodicity. These processes are not all coincident with the onset and offset of sleep, but show circadian rhythms of high and low activity systematically arranged about the sleep–wake transition points. (For example, in consistent night sleepers, temperature begins to fall in the late afternoon, continues to fall as sleep supervenes, reaches its low at the end of the second third of the sleep period, is on the rise before the morning awakening, and peaks in the early afternoon.)

Sleep–wake patterning—serving as the pacemaker for the other physiological variations—is, in turn, itself physiologically ingrained (i.e., reinforced) in its temporal positioning by the associated functions. In effect, the activities of sleeping and waking may be viewed as the behavioral top of an iceberg of insensible physiological processes, such as temperature variation and hormone elaboration. In the event that sleep and awake periods are removed to new, temporal locations in the 24 hour cycle, their subterranean physiological affiliates tend to remain fixed. In other words, one may elect—as in rapid time zone change (C.1.a) or "work shift" change (C.1.b)—to reposition sleep–wake conduct, but one does not succeed in moving *pari passu* the whole spectrum of related processes. They tenaciously hold to their former temporal positions. Though the *impetus* for

resituation of the chronobiological contexts for the new circadian sleep–wake schedule begins *immediately,* the actual realignment takes *several weeks or months* depending on the physiological parameter. Not only temperature, endocrine, and metabolic processes show this inertia; so do central nervous system processes related to alertness and sleepiness, namely, the preparatory neurophysiology for the sleep and wake states. Accordingly, after an acute sleep–wake cycle shift, waking is superimposed on brain functions "biased" towards sleep; conversely, the position vacated by waking behavior and now replaced by sleep (or an attempt at sleep) maintains its physiological orientation to the awake state. That is why sleep is not generally successful at a new time for a considerable period following a sleep–wake schedule change.

The reader may gain a visual impression of this complex set of interrelationships from an illustration. The factors to be illustrated are (1) the clock times of the sleep–wake behavior and (2) the clock time periodicity of internal circadian rhythms that are affiliated with the sleep–wake cycle. These factors will be exemplified if the reader will imagine two discs, one just in front of the other, that both rotate about the same axis. Behind them, and also on the same axis, sits a larger diameter, fixed 24 hour clock face without hands. The front disc, which is made of transparent plastic, represents the sleep–wake schedule (number 1, above). The sleep position in the schedule (8 hours, or a one-third wedge) is opaqued on the disc. The rear disc of the pair represents the internal circadian pattern (number 2, above). This disc is surfaced with a paint, chemically treated to remain white only so long as light shines upon it. It slowly reverts back to black if no light hits it.

With this device, we may portray the situation of an individual whose sleep period is regularly 2300 to 0700 hours. The opaque third of the front disc, corresponding to 2300–0700 hours on the clock face, has for a long time been situated over one area of the rear disc. Accordingly, the rear disc has long ago turned black in that third of its area and is otherwise white. The black portion of the rear disc illustrates the atunement of all other physiological processes to the circadian sleep period.

Now, a "work shift" change occurs. This may be indicated in the apparatus by rotation of the front disc so that the opaque third (the sleep period) is now positioned over the 0700–1500 hours area. What will happen over the course of the several weeks following the shift?

The formerly black 2300–0700 portion of the rear disc is now covered by the waking transparent portion of the front disc and progressively lightens through shades of gray to white, whereas its 0700–1500 area (the new sleep period), now cut off from light, remains light for a while and only incrementally darkens with each day of the new schedule. Such a period of gradual achromatic change characterizes the symptomatic transition period of the "work shift" syndrome. In summary, the new 8 hour position of sleep behavior is still tied to the physiology of a former waking period (it darkens very slowly); at the same time, the new 8 hour position of awake behavior is still tied to the physiology of a former sleep period (it lightens very slowly). The readjustment takes several weeks, during which, brain processes are neither fully synchronized for sleep or waking in the latter's required periods.

In rapid time zone change ("jet lag") syndrome (C.1.a), it is the rear disc that would be rotated in order to exemplify the temporal repositioning of the internal circadian rhythm, which takes place in the new time zone. The maintained clock time of sleep–wake behavior, indicated in this case by the stationary front disc, is characteristic of travelers in a new time zone—they orient to going to sleep at night and waking up in the morning, just as they do in their home time zone. Again, there is a period of slow fade-in/fade-out on the rear disc, illustrating the way "jet lag" also causes a misalignment of sleep–wake behavior and the internal circadian pattern.

In frequently changing sleep–wake schedule (C.2.a), the condition is illustrated by rotating one or the other disc (depending on whether the situation causing the syndrome is a chronic shifting of time zones or of work schedule) to a new position every day or every few days. As a result of the repeated shifts, the rear disc of internal physiology will show changing shades of gray in several, perhaps overlapping, areas—exemplifying a severely disrupted biocircadian pattern.

Some Additional Points: Though not covered as syndromes in this section, sleep–wake schedule disturbances are also induced by long-lasting DIMS or DOES conditions. This occurs because the periodicity of sleep–wake behavior is ultimately affected by persistent abnormalities in production and maintenance of the sleeping and waking states. Accordingly, disturbances in the 24 hour sleep–wake pattern are routinely seen in the affective disorders. Hypomanic (A.2.b) and acute schizophrenic (A.2.c) patients act like individuals with delayed sleep phase (C.2.b); they frequently cannot fall asleep until 3:00 or 4:00 a.m. and may then sleep until the late morning, if permitted. Bipolar depressed (B.2.a) patients also typically extend their periods in bed late into the morning. It is speculated by some observers that sleep–wake cycle shifts may play a fundamental role in the etiology of these conditions. Finally, narcoleptic (B.6) patients suffer not only from daytime sleep attacks and sleepiness, but from fragmented sleep at night. As a result, they also commonly incur substantial disruption of their internal circadian rhythmicity.

C. 1. Transient

a. Rapid Time Zone Change ("Jet Lag") Syndrome

Key words and phrases: acute "jet lag," acute phase shift of sleep, only east–west not north–south travel.

Essential features: *Rapid time zone change ("jet lag") syndrome is a transient sleep–wake schedule disorder resulting from a single rapid change of multiple time zones. It is immediately produced when one attempts to continue sleeping and waking on one's usual clock hour schedule in the new time zone. The syndrome consists of sleepiness, fatigue, and their mental consequences during the wake period and insomnia during the sleep period.*

Associated features and other information: Though symptoms may last for 7–10 days after rapid transit through numerous time zones, sleep–wake disturbances generally abate after about 2 days in the arrival location subject to

major individual differences. Symptoms resulting from time zone displacement are often associated with—but are not dependent on—sleep deprivation. Involvement in social contacts and reinforcement of accepted time cues for awake activities assist in the adaptation process. Disturbances may last longer after *eastward* flights.

Temporary difficulties with appetite may be noted. Adaptation of the timing of physiological functions other than sleep and waking may take 8 or more days. For example, awakenings to urinate may be more frequent. Readaptation of *all* functions following return to the time zone of origin is usually more rapid. Elderly travelers may be physiologically less tolerant of time zone displacements than younger people, and there also seems to be substantial variation in the symptom picture depending on age. It would also be expected that individuals subject to insomnia, particularly persistent psychophysiological DIMS (A.1.b), are more susceptible to "jet lag" disturbances.

It should be emphasized that this sleep–wake schedule syndrome is not an inherent or obligatory attribute of travel *per se*. It results from the dyssynchrony that occurs between a person's internal sleep–wake phases and the once-parallel clock hour sleep–wake times *as an attempt is made to maintain the same clock hour schedule in the destination time zone*. Two corollaries follow.

First, the syndrome is altogether avoidable in the new environment, if an effort is made to continue one's internal sleep–wake cycle unchanged by rotating it to the times in the new zone corresponding to its internal (i.e., former clock time) periodicity. In effect, the individual in the new environment would not be accepting the influence of usual day/night cues on waking and sleeping behavior. However, this may be difficult to arrange.

Second, the "jet lag" syndrome does not pertain to north–south travel, even when lengthy, providing the time zone does not change by more than 1–2 hours. This is so owing to the persistence of synchrony of internal sleep–wake phases with clock time. There may, of course, be symptoms after north–south journeys secondary to sleep deprivation if the traveling interrupts or interferes with customary sleep duration (e.g., a night flight).

Despite the increasing prevalence of "jet lag" syndrome as air travel for work and recreation increases, little information is available as to how this disorder can be avoided or treated. Acute effects on performance are generally of greater concern and consequence to the business traveler than to the vacationing tourist.

Differential diagnosis: Given an adequate history, little difficulty exists in identifying the rapid time zone change syndrome. Few cases come to the attention of physicians despite the often severe short-term symptoms.

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C. 1. b. "Work Shift" Change in Conventional Sleep–Wake Schedule

Key words and phrases: night work, acute phase shift of sleep.

Essential features: *"Work shift" change in conventional sleep pattern causes sleep–wake symptoms that begin immediately when one's work period—generally night work—is scheduled during the habitual sleep phase. Sleepiness and performance decrements occur during the new work–wake period, and sleep, which now is scheduled during the former work–wake period—generally in the daytime—is disrupted and shortened.*

This sleep–wake shift is equivalent to a rapid, multiple time zone change (C.1.a), effected *in situ*, and results in a similar disruption of internal sleep–wake phases. However, symptoms develop in C.1.a because of the tendency to *fit* one's sleep–wake pattern to the prevailing schedule, whereas in the "work shift" condition, they develop because of the need to *disengage* one's sleep–wake pattern from the conventional societal schedule.

The symptoms may improve during the second or third week of work on an altered sleep schedule but usually persist to some degree until a socially conventional schedule is resumed. Few individuals manage to adapt their internal pattern completely to their work–awake schedule, even after many years, because of the tendency to resume the prevailing society sleep–wake pattern on weekends and vacations. Under such circumstances, the conventional pattern remains fundamentally entrained and is resumed easily with rapid subsidence of symptoms.

Additional voluntary (e.g., weekend) or work-related changes in sleep scheduling tend to prolong the period of symptoms after a shift, though a brief return to the conventional schedule may be temporarily associated with a lessening of symptoms.

Associated features and other information: Insomnia during the sleep period occurs with or without increase in sleep latency. Gastrointestinal disturbances, chills, lassitude, and irritability are frequently associated symptoms. The desire to nap and difficulties with mental faculties also may occur during work hours. Occasional tours of night work may cause disturbances in family and social life that may be distressing to some individuals.

Differential diagnosis: A distinction must be made between the condition of single or intermittently altered schedules, described in this category, and the condition resulting (see C.2.a) when the work schedule is recurrently shifted—e.g., every fortnight ("rotating shift work"). In the former, symptoms may be severe at first but eventually diminish either with uncompromising adherence to the once shifted schedule or with resumption of a conventional sleep–wake schedule.

Unconventional or altered sleep schedules resulting from "work shift" changes lead to impaired ability to sustain sufficiently long sleep intervals. This may be partly because the shift worker usually reverts to sleeping at night on weekends and vacations, thus precluding a stable sleep–wake pattern (see C.2.a). Separa-

tion of a "work shift" alteration of sleep–wake schedule from other conditions associated with similar sleep patterns may sometimes be difficult. This is especially true when such conditions are suspected as chronic tension–anxiety (see persistent psychophysiological DIMS, A.2.a,b), drug and alcohol dependency (see A.3), and other sleep maintenance DIMS, e.g., REM sleep interruption DIMS (A.8.a).

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C. 2. Persistent

a. Frequently Changing Sleep–Wake Schedule

Key words and phrases: shift work, rotating shift work, recurrent "jet lag," air crew fatigue, disorganized sleep hygiene, chronic phase shifting.

Essential features: *Frequently changing sleep–wake schedule causes a mixed picture of chronic DIMS and DOES distinguished by disrupted and usually shortened periods of sleep (sometimes occurring several times per day), sleepiness and performance decrements during any scheduled wake period, and difficulty reestablishing a consolidated sleep period even when circumstances temporarily permit sleep schedule regularity.*

This syndrome, which has become increasingly prevalent in modern society, can be a consequence of several situations: (1) frequent, multiple time zone shifts experienced by regular air travelers (e.g., flight crews, peripatetic businessmen, and international negotiators); (2) repeatedly changing work and sleep schedules imposed on rotating shift industrial personnel; and (3) frequent, often self-imposed, and chaotic sleep schedule shifts by individuals who disregard, sometimes unwittingly, the importance of sleep hygiene (e.g., college students, disco aficionados, soldiers in battle). Regardless of the cause of the symptoms, their severity depends on the number of hours shifted each time, frequency of the shifts, and the duration of the intervening periods of sleep–wake cycle stability. These factors determine the extent of schedule disruption, because too frequent shifts (less than 3–4 intervening weeks) impose new changes on previous shifts to which physiological adaptation is still in progress.

Associated features and other information: Among the somatic complications of this syndrome, a tendency for peptic ulcer disease to begin within a few years of starting shift work is fairly well established. Other somatic complications are suspected but remain unproven. Severe mood changes and unremitting—or waxing and waning—cognitive difficulties may occur. Increasing use of and dependence on hypnotic, analeptic, and tranquilizing drugs, as well as

alcohol, are dangers in individuals whose occupations require frequent sleep–wake schedule shifts.

Shift work may cause disruptions of personal, family, and social life that can be intolerable to the worker. However, some shift workers actually prefer night work or rotating shift schedules and deny significant impairment. Older workers are more likely to develop symptoms than younger ones, and they adjust more slowly to a sudden phase shift.

Special note should be made of workers whose shifts do not rotate, but who work at unconventional hours (usually nighttime) *regularly*. Such individuals often revert to the conventional pattern of daytime wakefulness and night sleep on weekends and vacations and, accordingly, experience some of the consequences of chronic phase shifting. Even persons who scrupulously maintain their work–sleep schedule on days off have some difficulties. The reason for this phenomenon is not clear.

Differential diagnosis: Because of the sometimes serious emotional complications related to chronic sleep–wake schedule disruption, syndromes must be differentiated that combine psychological changes with sleep phase shifts, insomnia, and excessive somnolence (see DIMS associated with psychiatric disorders, A.2.a,b).

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C. 2. b. Delayed Sleep Phase Syndrome

Key words and phrases: phase lag, phase delay, stable asynchrony relative to typical environmental pattern, appears like sleep-onset DIMS with secondary DOES symptoms.

Essential features: *Delayed sleep phase syndrome is marked by sleep onsets and wake times that are intractably later than desired, actual sleep times at virtually the same daily clock hour, no reported difficulty in maintaining sleep once begun, and inability to advance the sleep phase by enforcing conventional sleep and wake times. The syndrome often presents with the major complaint of difficulty in falling asleep at a desired conventional time and may appear like a sleep-onset DIMS. Daytime DOES symptoms commence secondarily with sleep loss.*

The cause of delayed sleep phase syndrome is unknown. Rather than a DIMS or DOES, the disorder seems to be a perpetuated involuntary asynchrony between

the patient's internal sleep–wake phase schedule and clock time (and other environmental entraining cues for sleep); the phase transition times of individuals with this disorder occur later than is typical (i.e., phase lag).

Sleep-onset and awakening clock times are stable, but later than desired or required for social and occupational purposes. The latency to sleep onset is not prolonged and there is generally no difficulty in arousal after an adequate daily allowance of sleep. However, if the individual must rise at 7:00 a.m. in order to get to work or school, and consistently is unable to fall asleep until 3:00–6:00 a.m., sleep time becomes insufficient (except in the infrequent case of short sleeper, A.9.a), and a symptomatic sleep debt gradually accumulates. When such individuals are not required to awaken "early," as on vacations, daily sleep length is normal because sleep is extended, and symptoms of DOES disappear. Bedtimes and wake times during such a period remain on a regular schedule, and the patient sleeps late (e.g., 3:00–11:00 a.m.). A careful sleep–wake diary is accordingly needed for diagnosis.

When forced by work or school requirements to arise "early," patients with delayed sleep phase syndrome regularly make an attempt to retire earlier but continue to experience difficulty in initiating sleep (which they report as "insomnia"). Because of the need to rise "early," the sleep is short but unbroken and restful—though difficulty arising and subsequent sleepiness is reported under these circumstances. Typically, the individual achieves peak alertness and efficiency only late in the waking day. The DOES symptoms, including inadequate mental functioning in vocational activities, often constitute a significant impairment in daily living.

Associated features and other information: The individual is perplexed that he cannot find a way to fall asleep more quickly. All efforts to advance the sleep phase to a more suitable time by means of early bedtimes, enforced early risings, and the use of hypnotics yields little permanent success. Hypnotics aggravate the daytime symptoms, and dependence on them further complicates the clinical situation.

This syndrome sometimes begins during early childhood. In young adults it is common for onset to follow an episode of shift work, nocturnal disruptions due to illness, or late-night studying. Frequently, however, adult patients give an early history of symptoms of this disorder. Similar symptoms may be noted in family members. Recent efforts to differentiate carefully the clinical picture of this syndrome from that of DIMS with secondary DOES, for purposes of more suitable treatment, have been promising. Delayed sleep phase syndrome probably represents a small proportion of patients presenting to sleep clinics with insomnia, but its actual incidence among patients diagnosed as "sleep-onset DIMS" must be regarded as untested because of widespread failure to identify this condition. In other words, its role in conditions combining long sleep onset and late morning sleep requires further elucidation.

Differential diagnosis: The pattern of delayed sleep onset seems also to occur in previously unaffected individuals with the start of psychiatric disturbances. In bipolar affective disorder, manic (or hypomanic) type, sleep onset is commonly delayed a few hours, but sleep is short without enforcement (see

A.2.b). Sleep-onset and arise times may both be delayed in schizophrenic decompensations (see A.2.c). Whether the latter condition is triggering the appearance of delayed sleep phase or of symptoms that this disorder temporarily mimics is not clear.

Patients with phase delay syndrome should be differentiated from individuals who habitually go to sleep and wake late for social and other reasons but then complain of sleep-onset insomnia and difficult morning awakening on the sporadic days they must go to bed and get up early. Such individuals suffer instead from a transient sleep–wake cycle disturbance compounded by sleep loss, which usually accompanies an acute phase shift (see sections C.1.a and particularly C.1.b). Reestablishment of a regular earlier bedtime and wake time achieves appreciable success in the transient case, whereas it is ineffective in phase delay syndrome.

Another important differentiation is from non–24-hour sleep–wake schedule (C.2.d). In the latter, incremental and sequential delays of the sleep phase occur. Other disorders that may be confused with delayed sleep phase syndrome include A.1.b, persistent psychophysiological DIMS of primarily the sleep-onset type, particularly when patients with delayed sleep phase also have sleep deprivation as a complication because of enforced early arising.

Childhood-onset DIMS is differentiated by sleep maintenance difficulties and short sleep (see A.7). Patients presenting with DIMS associated with heavy sleep medication intake must be studied carefully in terms of history, and after drugs are withdrawn, in order to assess the presence of a delayed sleep phase syndrome with superimposed drug effects (see A.3).

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C. 2. c. Advanced Sleep Phase Syndrome

Key words and phrases: phase lead, phase advance, stable asynchrony relative to typical environmental pattern, evening inability to stay awake and early morning wakefulness.

Essential features: *Advanced sleep phase is characterized by sleep-onset and wake times that are intractably earlier than desired, actual sleep times at virtually the same daily clock hour, no reported difficulty in maintaining sleep once begun, and inability to delay the sleep phase by enforcing conventional sleep and wake times. Unlike delayed sleep phase (C.2.b), this condition does not interfere with the work or school day. The major presenting complaint is the inability to stay awake in the evening and sleep in the morning until desired conventional times.*

Though the patient may suffer negative personal, family, and social consequences from having to go to sleep consistently in the early to midevening, this condition does not eventuate in sleep loss. There are no symptoms of DOES, and hence no impairments of waking function compromise daytime mental activity unless the individual must stay up late for social or vocational reasons and nevertheless wakes up early. This leads to chronic sleep deprivation and daytime napping.

Associated features and other information: Sleep-onset times of 8:00–9:00 p.m. are typically paired with wake times of 3:00–5:00 a.m. Whereas most individuals productively use their awake time until it is necessary to get ready for work or school, some are disturbed by the long, solitary, morning awake periods. Attempts to achieve conventional sleep and wake times by trying to stave off sleep onset and extend morning sleep are usually not successful. Like delayed sleep phase, this condition represents a perpetuated, involuntary asynchrony between the patient's internal sleep–wake schedule, and clock time and other societal entraining cues for sleep.

Advanced sleep phase is less common than delayed sleep phase syndrome, and it is uncertain whether it actually constitutes a clinical pathological syndrome. It is seen in some young adults who seem unable to change their sleep–wake schedule though they might wish to because of personal and social needs. It is sometimes seen in elderly patients and is frequently associated with, or confounded by, symptoms of depression.

Differential diagnosis: The early awakening pattern in the elderly is reminiscent of DIMS with depression (A.2.b). The evening sleepiness makes it necessary to distinguish advanced sleep phase from persistent psychophysiological DOES (see B.2.b).

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C. 2. d. Non-24-Hour Sleep–Wake Syndrome

Key words and phrases: free-running pattern, incremental asynchrony relative to typical environmental pattern, periodic insomnia, periodic excessive sleepiness.

Essential features: *Non–24-hour sleep–wake syndrome is distinguished by an incremental pattern of delays in sleep-onset and wake times to steadily later times on successive days; that is, the individual's internal sleep–wake schedule becomes progressively more asynchronous with conventional clock times for phase transition by a fixed amount daily, usually less than 2 hours. This is a reflection of a greater than 24 hour sleep–wake period length in the individual in society, typically about 25 hours. When the individual's internal biological rhythm is out of phase with the societal sleep–wake conventions, the chief complaint is difficulty getting to sleep at night coupled with inability*

to remain awake during the day; when "in phase," there may be no complaint. Typically, there is periodic alternation between the two in the patients (see below).

This sleep–wake pattern may be intermittent or continuous in its progression. If the patient has readjusted expected bedtimes and arise times to correspond more closely to the times of falling asleep and waking up, the diagnostic features of the disorder are clearer (in that the average daily sleep time and degree of alertness are normal). However, most patients make intermittent attempts to wake up at conventional morning times and thereby limit total sleep. This leads to progressive sleep loss and secondary DOES symptoms, which interfere with work or school functions. A careful sleep–wake diary is needed for diagnosis.

Associated features and other information: Impairment of performance may be marked in affected individuals who attempt to participate in scheduled social activities on a daily basis. Partial or total occupational disability usually describes the status of the patient. Peak alertness and efficiency are reached only late in the waking day.

Blindness or schizoid personality may predispose to this condition. Strenuous efforts to maintain a 24 hour sleep–wake schedule lead to cyclic periods of sleep deprivation. These efforts can lead to use of and dependency on hypnotic and analeptic drugs, which are of little value in alleviating symptoms but may complicate the clinical presentation of the syndrome. A significant finding is that, with attempts to hold to a normal 24 hour sleep–wake schedule, the pattern of delayed or disrupted sleep and daytime sleepiness changes progressively with each day (because of the serial shifting of the patient's underlying sleep–wake cycle). Consequently, every few weeks the patient reports a period during which he is asymptomatic.

Differential diagnosis: Non–24-hour sleep–wake syndrome should be differentiated from delayed phase syndrome (C.2.b). In the latter, incremental delays of the phase of sleep do not occur. Other disorders that may be confused include irregular sleep–wake pattern (C.2.e) and persistent psychophysiological DIMS (A.1.b). [See also differential diagnosis of delayed sleep phase syndrome (C.2.b) for additional disorders to consider.]

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C. 2. e. Irregular Sleep–Wake Pattern

Key words and phrases: no circadian rhythm, loss of *zeitgebers* (time or scheduling indicators), grossly disturbed internal sleep–wake phase rhythm.

Essential features: *Irregular sleep–wake pattern is defined as disorganized and variable sleep and waking behavior, which disrupts the regular sleep–wake pattern. This condition is associated with frequent daytime naps at irregular times and excessive bedrest. Sleep at night is not of adequate length, and the condition may present as a DIMS, though total 24 hour sleep is normal for age.*

The symptoms include inability to fall asleep at the desired, conventional bedtime or to remain asleep for a sleep period of adequate length. The patient thus complains of "insomnia." Some degree of subjective cognitive impairment and sleepiness characterize the awake intervals between sleep.

The key feature of this condition is loss of a clear sleep–wake rhythm. Sleep becomes broken up into several or more short blocks in each 24 hours. Given the type of individual or situation, the requirement for a clear rest–activity pattern is reduced and the individual begins to nap excessively. Some individuals no longer adhere even to a defined pattern of mealtimes or scheduled activities. Along with sleeping, these may become *ad lib*. The major 24 hour sleep–wake phases lose clear definition.

Associated features and other information: Not only may sleep and wake phases be disrupted, but endocrine, temperature, and other circadian function curves lose their expectable fluctuations and flatten. Lassitude, weakness, and a number of somatic symptoms usually follow. The patient generally has no understanding that the napping and insomnia are mutually reinforcing and that the latter is not so much the cause of the problem as the result of the disordered, near-random 24 hour scheduling.

Nocturnal insomnia may be a result of irregular sleep–wake pattern but also may cause it if the individual is in a position to stay in bed excessively and nap frequently. The course of this condition is not self-correcting and tends to be chronic. It may be punctuated by futile diagnostic and treatment efforts, and also by the use of hypnotic and analeptic drugs. Significant drug dependence may occur.

Differential diagnosis: Irregular sleep–wake pattern—in terms of its sleep–wake phase and other rhythm disruptions—must be differentiated particularly from frequently changing sleep–wake schedule (C.2.a). Though both conditions may be phenomenally almost equivalent, the latter is incurred by individuals —because of work requirements or knowingly (and sometimes uncaringly)— who disrupt their sleep–wake phase schedule voluntarily.

This condition needs to be separated from several sources of DIMS affiliated with psychological and drug intake features (see A.1–3). Two distinguishing features of DIMS syndromes are (in some) the inability to nap during the daytime and (in all) the lack of a normal 24 hour accumulation of sleep. The sleep and nap pattern in some narcolepsy patients (B.6) may be superficially similar to the irregular sleep–wake pattern described here.

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D. Dysfunctions Associated with Sleep, Sleep Stages, or Partial Arousals (Parasomnias)

This section, the parasomnias, consists of a group of clinical conditions that are not disorders of the processes responsible for sleep and awake states per se. Rather they are undesirable physical phenomena, either exclusively appearing in sleep (e.g., somnambulism) or exacerbated by sleep (e.g., asthma). These events are manifestations of central nervous system activation usually transmitted into skeletal muscle or autonomic nervous system channels. Hence the abnormalities described in this section are reflected in a wide spectrum of systems. Some are disposed to appear in conjunction with a certain sleep stage (such as sleep-related painful erections, D.4.g); others (such as sleep-related bruxism, D.4.c) occur in several stages, and still others (such as familial sleep paralysis, D.4.e) show a predilection for the transition from sleep to arousal. It has been speculated that certain conditions—sleepwalking (D.1), sleep terror (D.2) and sleep-related enuresis (D.3)—may, in fact, be disorders of the state of partial arousal.

The classification committee recommends reservation of the term "nightmare" for dream anxiety attacks (D.4.a). Nightmare has a long tradition of application—particularly in Europe—to both sleep terror attacks and dream anxiety attacks. However, it is felt that because of the sharply different physiological and behavioral features of these two conditions, separate terminologies would be appropriate. Such differentiation would reduce confusion in diagnosis and treatment, as well as in the education of patients concerning the two conditions.

D. 1. Sleepwalking (Somnambulism)

Key words and phrases: deep NREM sleep, stages 3/4, absence of dreaming, does not always progress to walking, semipurposeful automatisms, amnesia, nonepileptic.

Essential features: *Sleepwalking consists of a sequence of complex behaviors that are initiated in the first third of the night during deep NREM (stages 3/4) sleep, and frequently, though not always, progress—without full consciousness or later memory of the episode—to leaving bed and walking about.*

This pattern starts with sitting up and execution of perseverative motor acts, followed by semipurposeful automatisms that, in addition to walking, may include dressing, opening doors, eating and executing bathroom functions. Attempts to communicate with or gently arouse the sleepwalking person are met with avoidance.

The walking behavior may terminate spontaneously in an awakening followed by several minutes of mild disorientation. On the other hand, the individual may return to his own bed from a sleepwalking sojourn without ever reaching consciousness or may lie down in another place to continue sleep, mystified the next morning to find himself there.

The sleepwalking episode may terminate before proceeding to walking: after sitting up and carrying out repetitive motor activity such as picking at the blanket, the individual commonly lies down again and resumes normal sleep.

A sleepwalking episode may last from a few minutes to more than a half-hour. As a rule, amnesia prevails the next morning for all behaviors and environmental encounters during the attack. Reports of dreaming from sleepwalking contain only fragmentary imagery and little plot or development.

Polysomnographic recording frequently shows extremely high amplitude EEG slow waves in the stage 4 sleep just preceding the muscular activation that ushers in the attack. EEG flattening, i.e., arousal, at times occurs before evidence of motoric activation. In the usual instance, as walking ensues, the high amplitude slow wave pattern gives way to an admixture of lighter NREM stages and slow, nonreactive EEG alpha activity. It is relevant that in both sleepwalk-prone and normal children sleepwalking can be provoked by standing the child on his feet during stage 4 sleep. This condition, like other stage 3/4 sleep attacks, accordingly may be viewed as an abrupt liberation of motor activity under circumstances of incomplete arousal.

Associated features and additional information: Though higher cortical functions are inefficient and coordination poor, visual inspection operates during sleepwalking. Objects in the individual's path are avoided or negotiated, but despite open eyes the patient's expression is blank or dazed. It is a myth that sleepwalkers always make their rounds in pristine safety. They can stumble or lose balance, and injury is not uncommon over hazardous routes such as through windows or down fire escapes. Frenzied behavior or aggression to persons or objects is infrequent. Sleep talking may occur during sleepwalking, but articulation is poor; dialogue is rare.

Patients with sleepwalking have a higher-than-normal incidence of other episodic disorders associated with deep NREM sleep such as sleep-related enuresis (D.3), sleep terror (D.2), and sleep drunkenness (B.9.c). Convulsive disorders and history of previous CNS infection or trauma also are more common than in the normal population.

Onset of sleepwalking may occur at any time after walking has been learned. Most commonly it is first reported between ages 6 and 12. Sleepwalking adults uniformly give a history of at least some episodes in childhood, then freedom from the disturbance until its recurrence in the third or fourth decade.

Sleepwalking usually lasts several years in children and adolescents, irrespective of its frequency. Most juveniles with the disorder are free of it before their twenties. The disturbance tends to be more chronic in adults.

Sleepwalking usually has a benign course, except for accidental injury. No resultant impairments of waking mentation or behavior have been noted. Sleepwalking is self-limited and does not directly predispose to other illness, though repetitive episodes may lead to secondary problems in family and interpersonal relationships. Sleepwalking in children is not considered to be caused by psychological factors and does not signify psychopathology. On the other hand, persistence of sleepwalking into adulthood, or its return, is highly associated with diverse forms of personality disturbance and psychopathology.

Factors associated with deepening of nighttime sleep, such as fatigue or prior sleep loss, increase the probability of sleepwalking in children. Sedative and hypnotic drugs predispose to attacks in patients with repetitive sleepwalking. In some individuals, stress and emotional tension may increase the frequency of attacks.

Sleepwalking is fairly common in childhood and adolescence. As many as 15% of all children have one or more sleepwalking experiences. The prevalence of frequent sleepwalking behavior is in the range of 1–6% in children but is relatively rare in adults. Attacks may be more common in males.

The tendency to sleepwalk has a genetic loading. It is observed in identical twins. Relatives of sleepwalkers show a higher incidence of the condition than in the normal population. In families of patients with sleepwalking, the individuals tend to be deep sleepers.

Differential diagnosis: The differential diagnosis of sleepwalking requires separation from sleep-related epileptic seizures (D.5.b.), psychomotor epilepsy, fugue states, and sleep drunkenness (B.9.c.).

Psychomotor epileptic seizures may occur at night and produce episodes of confusional automatic behaviors similar to sleepwalking except that the patients almost never return to their own beds. Also, during epileptic attacks patients are totally unreactive to environmental stimuli, and automatisms like swallowing and rubbing of hands are more common. Patients with seizure-induced behavior often manifest the same pattern in the awake state as well, and the activity is usually associated with recordable seizure discharge. It must be emphasized that documented epilepsy does not preclude the same patient also having nonepileptic sleepwalking. The two conditions are not uncommonly associated.

Fugue states are distinguishable from sleepwalking on several counts: they are rare in children, typically begin in wakefulness, last for hours or days, are not characterized by disturbances of consciousness, and are usually associated with other evidences of severe psychopathology.

Some forms of DOES (section B) may be characterized by difficulty in awakening, as well as by excessive sleep. Sleep drunkenness (B.9.c.) may resemble sleepwalking except for the former's usual morning appearance and high frequency of aggressive behavior. However, the combination of nighttime awakenings associated with confusion, complex automatisms, and extensive walking is rare except in sleepwalking.

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D. 2. Sleep Terror (Pavor Nocturnus, Incubus)

Key words and phrases: deep NREM sleep, stages 3/4, autonomic arousal, terrifying scream, night terror, not REM sleep, not dream anxiety attack.

Essential features: *A sleep terror is an arousal in the first third of the night from deep NREM (stage 3/4) sleep, almost invariably inaugurated by a*

piercing scream or cry and accompanied by behavioral manifestations of intense anxiety bordering on panic.

In the typical instance, the individual sits up in bed and displays agitated and perseverative motor movements (automatisms), a frightened expression, mydriasis, profuse perspiring, piloerection, rapid breathing, and quick pulse. Following the initial arousal, full waking consciousness is not achieved for 5–10 minutes. A child in this state is inconsolable until the agitation and confusion subside. The child may then recount having had a sense of terror and isolated visual imagery prior to arousal but rarely a vivid dream. Morning amnesia for the entire episode is the rule. Adults frequently have pre-arousal impressions of dread, paralysis, helplessness, palpitations, and respiratory oppression or choking—components of a classical incubus attack, the adult homolog of pavor nocturnus in children.

The severity of a sleep terror is proportional to the duration of the preceding stage 3/4 sleep period. Prior to difficult episodes, the EEG delta waves may be higher in amplitude than usual for deep NREM sleep and breathing and heartbeat slower. The screaming eruption of the attack is accompanied by a two- to fourfold rise in heart rate, and the EEG quickly assumes an alpha pattern. Accordingly, sleep terror, as well as other stage 3/4 sleep attacks, may be viewed as abrupt liberations of autonomic and motor activity under circumstances of incomplete arousal.

Associated features and other information: A sleep terror may progress to sleepwalking, particularly if a vigorous attempt is made to abort the episode by standing the child up. The incidence of sleepwalking and somnambulism is greater in children with sleep terrors than without, and the disorders are substitutive.

In children, sleep terrors characteristically commence between ages 4 and 12, then ebb and disappear in early adolescence. Attacks are extremely variable in frequency both within and among individuals, usually occurring at intervals of days or weeks but sometimes on consecutive nights in severe cases. If sleep terrors do not begin in childhood, they tend to emerge when adults are in their twenties or thirties. Onset after 40 is rare. The condition in adults is often chronic but does not predispose to other illness.

Consistent personality features have not been found in children with sleep terrors. Generally within normal limits of concurrent psychological functioning, children who have sleep terrors also have no greater disposition to mental illness later in life. In contrast, adults with this disorder frequently display emotional problems such as severe chronic anxiety. At both age levels, sleep terror attacks may lead to secondary disturbances in normal family life. Due to embarrassment, children who have frequent episodes often shy away from such normal activities as summer camping and sleepovers at friends' homes.

Daytime stress and fatigue seem to enhance the tendency for a particular attack. Single bedtime doses of tricyclic antidepressants and neuroleptics also may increase the frequency of episodes. The protracted deep NREM sleep stages that are normal for children in the first third of the night may render them particularly susceptible to sleep terror.

The reported incidence of sleep terror is in the range of 1–4% in children. However, it is likely that a much greater proportion of children experience attacks, if only infrequently. Sleep terror is more common in males than females and shows a preference for certain families.

Differential diagnosis: Dream anxiety attacks (D.4.a)—which arise in REM and, infrequently, in NREM stage 2 sleep—may be distinguished from sleep terrors by the timing of their appearance in the middle and latter thirds of the night. Though at times distressing, anxiety dreams are generally associated with less fear, confusion, and sympathetic arousal than sleep terrors—and the anguished, terrified scream that marks a sleep terror arousal is rare. The hallmark of a REM sleep nightmare is the distinct recall of a detailed dream sequence in which a growing threat seems to lead to the ultimate awakening. Many parents misinterpret the fearfulness and fragmentary imagery reports of sleep terrors as indicative of dream anxiety attack, usually a REM sleep phenomenon (the term, nightmare, is reserved for dream anxiety attack, see D.4.a).

Terrifying hypnagogic hallucinations must also be distinguished from sleep terrors. The former may occur at sleep onset in depressed (A.2.b) and narcoleptic (B.6) patients, and in individuals with a chronic, shifting sleep–wake schedule (C.2.a). Vivid hypnagogic images occur at the transitions from waking to sleep. They can be quite dysphoric in some individuals. Sleep-related epileptic seizures (D.4.b), associated with fear and postictal confusion, may produce episodes of similar appearance. However, seizures are rare and usually combined with abnormal EEG discharge and sometimes a convulsion. Patients with this disorder generally display various types of epileptic seizures in waking as well as in sleep.

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D. 3. Sleep-related Enuresis

Key words and phrases: nocturnal bed-wetting; primary, secondary, idiopathic, symptomatic enuresis.

Essential features: *Sleep-related enuresis is a condition characterized by involuntary micturition beginning usually during deep NREM sleep in an individual who has or should have gained voluntary waking control of the bladder. Bed-wetting after 3 years of age is considered to be an enuretic disorder. It is a syndrome mainly, but not exclusively, seen in children.*

Sleep-related enuresis is usually observed in the first third of the night and is uncommon during REM sleep. Most episodes of bed-wetting commence in deep NREMS stages 3/4 or during the passage from deep NREM sleep to lighter stages in the course of arousal. Frequently, sphincter release occurs only after arousal has begun, as is indicated by a typical pattern of body movement prior to micturition

followed by quiescence with the start of urine flow. The individual is difficult to arouse, then confused and disorientated after the enuretic episode. This sequence, as in sleepwalking (D.1) and sleep terror (D.2), contributes to the view that sleep-related enuresis is a disorder of partial arousal. Dreaming is vaguely and infrequently reported in conjunction with bed-wetting, particularly when it occurs in the first hours of the night.

Associated features and other information: In some enuretics, toilet training is not accomplished as expected early in childhood. The customary bed-wetting during the sleep of infancy and early childhood simply persists to an age when it can no longer be regarded as within normal limits ("primary enuresis"). More commonly, a child who has undergone successful toilet training again becomes, either spontaneously or in relation to some stress, enuretic in sleep ("secondary enuresis"). Both primary and secondary enuresis are types of *idiopathic enuresis*. They are not related to *symptomatic enuresis*, which refers to an organic etiology. The former are self-limited conditions. Idiopathic enuresis usually disappears by late childhood or adolescence and is probably a phenomenon of delayed maturation. The course of *symptomatic* enuresis due to urogenital or other disease is generally less benign and depends on successful treatment of the underlying pathology. Causes of symptomatic enuresis include congenitally small bladder; other malformations of the urogenital system; urethral and bladder infections; ureteric reflex; renal, metabolic, and endocrine diseases associated with increased urinary output; and obstetrical damage (to the mother).

Impairment of daytime physical functions resulting from idiopathic sleep enuresis is generally slight. However, the symptom may cause considerable embarrassment and inconvenience to both children and adults, and tends to restrict the child's range of activities.

Traditionally, the bed-wetter who is without a known organic cause for the enuresis is labeled—as a result of the symptom—as having emotional problems, and many come to suffer considerable guilt as well as shame. In fact, enuretic children and adolescents, though probably quite sensitive to stress, have no typical personality pattern or psychopathological features.

Factors that accentuate deep NREM sleep, in which idiopathic sleep enuresis generally occurs, predispose to attacks. Individuals who have sleep enuresis are characteristically very "deep" sleepers and hard to wake. Daytime stress seems to be related to the incidence of enuretic episodes on the following night. Children are predisposed to enuresis when they are subject to changes in their sleep–wake schedule (see C.1.a, b). Nocturnal enuresis, beginning without previous history, may be symptomatic of sleep apnea syndrome (B.4.a). Individuals who have idiopathic enuresis have a higher than normal incidence of sleepwalking and sleep terrors, conditions that may be considered substitutive.

Sleep-related enuresis is a common condition. It is estimated that at age five, 15% of boys and 10% of girls are enuretic. The incidence is higher in institutionalized children. The great majority of children with idiopathic sleep-related enuresis become dry at night by puberty. It is still more common in young adults than in middle-aged and elderly people. Among enuretic adults, 35% are schizophrenic. Idiopathic sleep-related enuresis has a strong familial pattern.

Symptomatic enuresis due to congenital malformation may also show a familial pattern.

Differential diagnosis: Sleep-related enuresis must be distinguished from epileptic seizures (D.4.b) with urinary incontinence. Patients who have enuresis in sleep *due to* seizure activity normally have a history of nocturnal motor convulsions associated with the enuresis and show seizure discharge at the time of the enuretic episode. They may have simultaneous fecal incontinence and often suffer from daytime epileptic seizures as well. However, in the majority of epileptic children who are enuretic, the episodes are not related to epileptic discharge. They more likely represent a coexistent, nonepileptic phenomenon. This can be substantiated by careful nocturnal EEG recording in combination with a technique that denotes the precise time of enuretic episodes.

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D. 4. Other Dysfunctions

a. Dream Anxiety Attacks (Nightmares)

Key words and phrases: nightmare, frightening dream, vivid dream recall, generally REM sleep phenomenon, not NREMS stages 3/4, not night terror.

Essential features: *A dream anxiety attack is an awakening from REM sleep (sometimes light NREM sleep) with unmistakable, detailed recall of an extended and disturbing dream, accompanied by anxiety and some autonomic arousal.*

On arousing from sleep, one is aware of just having had a dream experience involving an immediate and credible threat to survival, security, or self-esteem, which provoked—also in the dream—either a shock, fear, or retaliatory response. Accompanying such a response, there is usually an abrupt motor reaction in the dream (that is generally also expressed in actual physical movement), which seems to precipitate the reentry into the waking state. Dream anxiety attacks are not associated with the level of panic observed commonly in a night terror (D.2). The autonomic hyperactivity is also not as great, though the pulse rate is frequently rapid.

A distinct feature of a dream anxiety attack is that however encompassing the dream experience is, the awakened individual is momentarily oriented to the environment with a clear sensorium.

Associated features and other information: Nightmares usually occur in the mid-to-late portion of the nocturnal sleep period and may be experienced in naps that are more than an hour long.

Nightmares are experienced at all ages and by virtually all people. They tend to

be more prevalent at times of insecurity, emotional turmoil, depression, guilt, aggressive feelings, anxiety, and after painful, real events. Whereas some individuals seem to be peculiarly susceptible to nightmares, others rarely experience them. People with anxiety and other "neurotic" disorders seem predisposed to them. "Bad (dysphoric) dreams," more than dream anxiety attacks, are a feature of depression, though agitated depressives report the latter as well.

Certain pharmacological agents such as reserpine may contribute to nightmares (perhaps via promotion of depression). Reduction or withdrawal of REM-sleep-suppressing agents leads to a rapid and large recovery of REM sleep (REM sleep ratios of 30–50%) often associated with nightmarish dreams. This is a key feature of too rapid withdrawal from sleeping pills and amphetamines and is also part of the picture of acute alcohol withdrawal (see A.3.a, d; B.3.a; A.6). Rebound of REM sleep after sleep loss *unrelated* to drugs is correlated less with dream anxiety attacks.

Recurrent dream anxiety attacks may be emotionally distressing and interruptive of regular sleep. Sleep phobias (see A.2.a) and repeated REM sleep interruption DIMS (A.8.a) may be complications of frequent nightmare attacks.

Differential diagnosis: Hypnopompic imagery in nonnarcoleptic patients may be triggered by a sudden awakening accompanied by sensory stimulation. The images may be vivid but usually are brief. Recurrent, nightmarish dreams may be allied with sleep-related epileptic seizures (D.4.b). As indicated above, a sudden outbreak of dream anxiety attacks may indicate a heavy drinking or drug pattern with recent partial or relative withdrawal (see A.3.a, d; B.3.a; A.6).

The critical differentiation is from night terror (D.2). In this disorder, the individual—commonly a child—typically arouses in terror with a scream. Because it is a NREMS 3/4 phenomenon, night terror occurs in the first third of the night. Autonomic arousal is great, and the individual may be both inconsolable and out of contact with the environment. Dream recall is low grade. Night terrors may also be associated with enuresis and sleepwalking, which rarely accompany a dream attack.

If dream anxiety attacks provoke a DIMS condition, code this parasomnia also under A.2.a or A.8.a, whichever DIMS diagnosis is more appropriate to the clinical presentation of the sleep interruptions.

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D. 4. b. Sleep-related Epileptic Seizures

Key words and phrases: convulsions, tonic–clonic, psychomotor, "sleep epilepsy," recurrent dreams, not sleepwalking, not sleep-related (nocturnal) myoclonus, may or may not be associated with sleep-related enuresis.

Essential features: *Sleep-related epileptic seizures are characterized by attacks of epileptic mechanism that recur in sleep. The seizures either take the form of generalized epileptic convulsions (tonic–clonic, tonic, or myoclonic in type) or are partial seizures of complex symptomatology (often "psychomotor" type with confusion and automatisms).*

In some epileptic patients, seizures occur more or less exclusively during sleep, a condition described as "sleep epilepsy." Patients with sleep-related epileptic seizures may also regularly show seizures associated with awakening; these are usually generalized myoclonic or tonic–clonic seizures and take place in the first hour after awakening. During sleep the attacks have a disposition for the first 2 hours after sleep onset or occur in a second peak around 4–6 a.m. Especially with the convulsive type of epileptic seizures, nocturnal incontinence of bladder or bowel may occur.

Associated features and other information: Some patients with sleep-related epileptic seizures are prone to epileptic seizures in the waking state. This combination is referred to as "diffuse epilepsy." About 25% of the "sleep epilepsies" are symptomatic of demonstrable organic brain disease, lower than the approximately 50% for the "diffuse epilepsies," but higher than the 10% that characterizes the solely "waking epilepsies." In patients whose sleep-related seizures are affiliated with organic brain disease, the latter may also show its effect in low IQ or a neurological deficit. Psychological problems, in reaction to the disease or related to brain dysfunction, commonly coexist.

Sleep-related epileptic seizures may occur at any age but are most frequent in children. The most common type is the tonic generalized seizure seen in "childhood epileptic encephalopathy," or Lennox-Gastaut syndrome, in which 30–100 seizures may occur each night. Parents sometimes mistakenly consider these as simple body movements.

The course of sleep-related epileptic seizures is quite varied, depending mainly on whether the epilepsy derives from permanent or transient pathology, metabolic disease, and genetic or other factors, and depending also on the degree of pharmacological control and age of the patient. Usually no daytime impairments result from the sleep-related epileptic seizures per se, exclusive of impairments referable to underlying pathology.

Little evidence has been uncovered that a change in awake activities predisposes to seizures during sleep on the following night. Some clinical forms of epilepsy (such as Lennox-Gastaut syndrome), however, are more frequently associated with a sleep distribution of seizures. Sleep-related seizures may give rise to sleep behaviors that mimic other nonepileptic parasomnias such as sleepwalking, enuresis, leg jerks, breathing difficulties, etc. Temporal lobe seizures may cause recurrent and stereotyped dream content.

Sleep-related epileptic seizures are common among epileptic patients. Whereas the prevalence of epilepsy is 0.5%, "sleep epilepsy" (in which seizures are more or less exclusively confined to sleep) is observed in 20–25% of individuals with epilepsy; "diffuse epilepsy" (in which some seizures occur in sleep and others in waking) is present in 30–40% of epileptics. No male or female preponderance is described. The genetic factor in patients with "sleep epilepsy" is lower than for

epilepsy in general, about 10% showing a positive family history. A familial form of "sleep epilepsy" is known but rare.

Differential diagnosis: Sleep-related epileptic seizures must be distinguished from sleep-related attacks or episodes that are of nonepileptic mechanism. The presence of a concomitant EEG seizure discharge during the attack is important to proving an epileptic mechanism. Failing demonstration of an EEG discharge in sleep, its demonstration in the same patient in the waking state during an identical clinical seizure is necessary. A favorable response to "anticonvulsant" medication is not, in itself, absolute evidence that an epileptic mechanism is responsible for the behavior, since such drugs also have effects on many nonepileptic phenomena.

Generalized, tonic–clonic, sleep-related, epileptic seizures seldom offer difficulties in differential diagnosis. Tonic epileptic seizures in sleep, however, must be distinguished from certain nonepileptic tonic reactions seen during sleep in a number of degenerative and demyelinating conditions. Epileptic myoclonus in sleep must be carefully distinguished from physiological myoclonus of sleep onset ("sleep starts"), from sleep-related (nocturnal) myoclonus (A.5.a; B.5.a) and the "restless leg" syndromes (A.5.b; B.5.b), and from other forms of nonepileptic myoclonus observed in sleep such as spinal myoclonus.

Partial epileptic seizures with complex symptomatology, presenting as "psychomotor" attacks with confusion and automatisms, must be differentiated from sleepwalking (D.1). Epileptic seizures during sleep, associated with urinary or fecal incontinence can be separated from nonepileptic sleep-related enuresis (D.3) or rectal incontinence (copracrasia).

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D. 4. c. Sleep-related Bruxism

Key words and phrases: nocturnal teeth grinding, NREMS stage 2 primarily.

Essential features: *Bruxism is a condition in which a rhythmic pattern of masseter muscle activity causes firm contact of the teeth of the upper and lower jaw, often accompanied by a loud grating or clicking sound. The episodes occur primarily in NREMS stage 2 and in stage transitions. They are not related to abnormal EEG discharge and are not allied with vivid or singular mental content. The patient has no awareness of the grinding and is rarely awakened by it.*

Teeth grinding, though preponderant in stage 2 sleep, is evident in all stages. Episodes of bruxism are associated with body movements and increased heart rate. The rhythmic masseter contractions in NREMS 3/4 are followed, but never

preceded, by EEG evidence of partial arousal. These EEG changes do not occur in stage 2. However, bruxism may be considered as a disorder of partial arousal in that it is another example (see D.1,2) of a motor discharge that is released primarily during a light, often alpha-infiltrated NREMS stage, just after K complexes, and occurring with cardiac rate and finger pulse changes. Complaints of insomnia and daytime somnolence are occasionally presented by nocturnal teeth grinders. (If this secondary DIMS is a clinical problem, it should be additionally classified under A.6.) Earlier reports that bruxism is a distinctive REM sleep phenomenon did not adequately distinguish the rhythmic masseter contractions of true bruxism from other oral movements seen in REM sleep.

Associated features and other information: Bruxism causes a sensation of aching jaws in the morning. It often leads to progressive attrition of the occlusal surfaces of the teeth and damage to both bone and soft tissue supporting structures. Bruxism during waking is common. Interestingly, unlike sleep-related teeth grinding, it is unaccompanied by sound.

The incidence of sleep-related teeth grinding is at its peak of 15% in children and adolescents. It is seen in all age groups but is much less frequent in later life. It may be transient or chronic in course. Patients with severe conditions must be fitted with special teeth-protecting prostheses to avoid extreme damage and prevent serious dental problems. In severe cases it can disrupt intimate sleeping arrangements or cause marital discord because of the difficulty of the partner in accommodating to the prominent and disagreeable sound.

No evidence of epileptic activity has been found to underlie bruxism, nor is preexisting dental pathology responsible for the condition. Systematic study of personality and psychological variables does not support the contention that emotional factors contribute to bruxism. Gender differences are not a factor in its incidence, but bruxism has a greater prevalence in certain families.

Differential diagnosis: Sleep-related teeth grinding seldom presents problems in diagnosis. Patients with convulsive disorders leading to masseter activity and dental impairments are identified by the presence of concurrent EEG abnormalities, incontinence, sore tongue, and awake-state seizure activity. Neurological examination is indicated in patients with bruxism to rule out conditions attributable to muscle spasticity (see D.4.b and A.6, respectively).

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D. 4. d. Sleep-related Headbanging (Jactatio Capitis Nocturnus)

Key words and phrases: headbanging, body rocking.

Essential features: *Sleep-related headbanging is the term for a sleep behavior consisting chiefly of rhythmic to-and-fro head rocking, less commonly of*

total body rocking, occurring just before or during sleep. Usually, it is observed in the immediate presleep period and is sustained into light sleep. It uncommonly persists into or occurs in deep NREM sleep.

Associated features and other information: This condition usually appears in childhood and has a self-limited course. It is very rare after adolescence, implicating a maturational lag in the mechanism. Waking impairments are not observed.

The condition is more frequent in children of subnormal intelligence. In the absence of organic factors, the headbanging in sleep appears to be related to environmental stress such as disharmony between parents and pressures for the child to excel in school and elsewhere.

Differential diagnosis: This disorder seldom poses diagnostic problems but requires the ruling out of sleep-related epileptic seizures (D.4.b).

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D. 4. e. Familial Sleep Paralysis

Key words and phrases: isolated sleep paralysis, predormital and postdormital paralysis, not narcolepsy.

Essential features: *Familial sleep paralysis is characterized by a sudden inability to execute voluntary movements either just at the onset of sleep or on awakening during the night or in the morning.*

Sleep paralysis, though most commonly observed as one of the classic symptom tetrad of narcolepsy (B.6) may occur independently in otherwise healthy individuals who may never develop other symptoms of narcolepsy. Generally, in this isolated form of sleep paralysis, consciousness is clear, though hallucinatory phenomena and dreamlike mentation may accompany the immobilized state. Respiration is usually uncompromised. Sleep paralysis episodes endure for several minutes but may be immediately terminated by external stimulation, for example, the voice or touch of another person. Some patients discover that vigorous movement of the eyes—often the only voluntary movement that is spared—will break the paralytic state. The postdormital period is the most favored point of occurrence of the attacks.

Familial sleep paralysis has a heredofamilial incidence. In well-studied families, in which the isolated form of the disorder has affected several generations, it has been shown that the mother transmits the condition as a dominant trait bound to the X-chromosome.

Surveys of normal individuals have identified sleep paralysis in 3–6% of respondents, but this figure is far too high. Some confounding of the samples with narcoleptic individuals and other disorders seems likely. Sleep paralysis may begin in childhood or develop in adulthood. The multigeneration, familial cases

are mostly women, but in the series reporting surprisingly high prevalence figures, many of the cases are males.

Differential diagnosis: Familial sleep paralysis is clinically identical to the sleep paralysis of narcolepsy (both presumably a dissociated REM sleep phenomenon) except that more frequent associations with drowsiness and hallucinatory phenomena occur in narcolepsy. Sleep paralysis attacks are associated with sleep–wake transitions rather than with eruptions of emotion in the awake state, a characteristic of cataplectic paralysis.

The complaint of episodes of complete immobility, in conjunction with seemingly hallucinatory phenomena, must be distinguished from psychotic decompensation or hysterical states.

Another form of familial paralysis, which characteristically affects adolescent males, is familial periodic (hypokalemic) paralysis, occurring in genetically disposed individuals. The pathophysiology in this condition hinges on depletion of tissue potassium in the muscular system owing to an exaggerated 24-hour variation in potassium exchange. The attacks often occur during periods of rest so that the individual may wake up paralyzed as in isolated sleep paralysis. The connection of these attacks with depletion of serum potassium, their periodicity, and their provocation by high carbohydrate meals and alcohol assist in differentiation of familial hypokalemic paralysis from the familial sleep paralysis under review here.

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D. 4. f. Impaired Sleep-related Penile Tumescence

Key words and phrases: erectile impotence, reduced REM sleep erections, organic impotency, psychogenic impotency, nocturnal penile tumescence (NPT).

Essential features: *Impaired sleep-related penile tumescence—compared relative to the expected number and duration of full erections that are objectively recorded during sleep in normally potent men—is a condition that occurs as a result of organic dysfunctions within the chain of complex processes responsible for sustained erection, and possibly occurs also in certain instances of awake-state psychogenic impotence.*

The study of nocturnal penile tumescence (NPT) has definitively established that erections appear in the sleep of normal males, in the main associated with emergence of the REM sleep periods. However, the amount of NPT declines with age: in the third decade, erection is present in almost 200 minutes of sleep time compared to less than 100 minutes by the eighth decade. This diminution corresponds to a decline of one-third in the older group in terms of proportion of total sleep period containing NPT.

Strain gauge sleep monitoring of the penis has demonstrated that manifestation of normal physiological erection during sleep is absent or sharply reduced in virtually all organic conditions that are associated with compromise of waking erectile potency. On the other hand, in psychogenic impotence NPT measurements show little, if any, reduction from the normal standards for age. Accordingly, penile strain gauge monitoring has come into extensive use in the attempt to differentiate organic from psychological etiologies of impotence.

Almost any condition that affects general metabolic efficiency seems to carry a potential for disturbance of normal erective capacity, particularly conditions that compromise vascular, neural, or endocrine functions relating to the penis. The most common organic cause of impotence is diabetes mellitus, but a wide variety of cardiovascular (e.g., hypertension), endocrine (e.g., antiandrogen or estrogen substances), urogenital (e.g., Peyronie's disease, hydrocoele), neurological (e.g., multiple sclerosis, CNS syphilis), hematological (e.g., Hodgkin's disease), respiratory (e.g., pulmonary insufficiency), and pharmacological (e.g., adrenergic blocking) conditions have been implicated in deterioration of the ability to initiate and maintain erection.

Differential diagnosis: NPT monitoring is currently being used to distinguish organic and psychogenic causes of impotence, but research still focuses on whether purely psychological factors may also affect penile tumescence during sleep (though psychogenic factors do not rival organic ones in their effect on NPT). The finding that some individuals with NPT deficiency, in the absence of documented organic findings, experience an increase in sleep erections with resolution of psychological problems and improved sexual functioning suggests this possibility. However, covert organic lesions may also run a fluctuating course.

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D. 4. g. Sleep-related Painful Erections

Key words and phrases: painful erection.

Essential features: *Sleep-related painful erection is diagnosed when the repetitive physiological tumescence of the penis, which occurs in the course of sleep, particularly during REM sleep, is accompanied by painful sensations that awaken the individual.*

Associated features and other information: This disorder is very uncommon but striking when it occurs. The men who have it appear to have little difficulty with sexual functioning and erections in the awake state; that is, pain is generally not present when erections are achieved in waking. Sleep-related painful erection does not *require* pathology of the penis, but it is also often noted with

disease or anatomical defects of the penis such as Peyronie's disease and phymosis.

Anxiety symptoms accompany this condition but are mainly secondary to the physical problem.

This condition may lead to regular, REM-sleep-related awakenings that can progress to a DIMS. In this event, the condition should be doubly classified (also under A.6).

Differential diagnosis: The basic condition must be separated from physical pathologies of the penis that can induce pain with erection. The regular, REM-sleep-related DIMS has a periodic awakening pattern like repeated REM sleep interruptions (A.8.a) in which the arousals are initially provoked by nightmares. However, there should be no difficulty differentiating these two sources of DIMS.

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D. 4. h. Sleep-related Cluster Headaches and Chronic Paroxysmal Hemicrania

Key words and phrases: Cluster headaches, cluster migraine, nocturnal migraine, chronic paroxysmal hemicrania, vascular headaches, REM sleep.

Essential features: *Sleep-related cluster headaches are agonizingly severe, unilateral headaches that appear often during sleep and are marked by an on–off pattern of attacks. Chronic paroxysmal hemicrania is a similar unilateral headache that occurs every day with more frequent, but short-lived, onsets that are without a preponderant sleep distribution. Both types of vascular headache are examples of sleep-exacerbated conditions and appear in association with REM sleep periods, paroxysmal hemicrania being virtually "REM sleep locked."*

The typical onset of the cluster headache is during sleep. One is either awakened with pain during the night or aware of the attack on awakening in the morning. Shifts of sleep schedule reveal that the headaches tend to follow sleep rather than time of day.

Laboratory recording has demonstrated that the awakenings with pain show a bias for REM sleep periods and post-REM sleep. This is invariable in chronic paroxysmal hemicrania. Accordingly, sleep disturbance is much greater towards the latter part of the sleep period, and REM sleep may be reduced. (Unpublished reports have shown reduction and amelioration of attacks when REM-sleep-suppressing agents are taken prior to sleep.) The association of certain kinds of vascular headache with REM sleep may have its origin in the autonomic variability, particularly the wide blood pressure shifts, observed during the REM sleep stage.

Associated features and other information: Cluster headache is characteristically associated with preheadache prodromal symptoms (e.g., "aura," scotomata, drowsiness, mood changes, gastrointestinal disturbances),

and during the attack with tearing, rhinorrhea, visual symptoms (sometimes Horner's syndrome), anorexia, nausea, and an increase in pain when any but the recumbent position is assumed. Cluster headache and chronic paroxysmal hemicrania are members of the vascular group of headaches, known to cause extreme discomfort owing to the exceptional sensitivity of the intracranial arteries to pain when they are distended and squeezed. Though the reason for the onset of attacks in only certain individuals is not yet understood, the mechanism of the pain appears related to initial spasm (prodromal phase) and then dilatation of the extracerebral arteries, leading ultimately to periarterial edema and therefore pressure on the arterial branches.

Neither of these headaches appear before puberty. Women slightly exceed men in incidence and severity of attacks. Predilection for affliction within family lines is striking. Cluster headaches, when they appear, endure many hours, but new attacks are initiated only one to three times per day in contrast to chronic paroxysmal hemicrania, in which up to 24 episodes, lasting 5–15 minutes each, may commence in a 24-hour period.

Differential diagnosis: The differential diagnosis of vascular headache in sleep must include headaches referable to pressure from expanding intracranial masses (e.g., tumors, aneurysms, hematomas). Also to be considered are inflammatory conditions of the bones, nerves, and meninges, referred pain, and psychogenic ("tension") headaches, though the latter are generally bilateral or midline in distribution. All these headache etiologies may be secondarily aggravated by hemodynamic variations induced during the REM sleep stage. Disturbing dreams may also induce psychogenic headaches in REMS.

If the vascular headaches described in this category cause a substantial disturbance of sleep, the condition should be additionally coded as a DIMS under section A.6.

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D. 4. i. Sleep-related Abnormal Swallowing Syndrome

Key words and phrases: deficient swallowing, choking and coughing in sleep, subjective sensation of choking and impaired respiration during sleep, not obstructive sleep apnea, not sleep apnea DIMS syndrome.

Essential features and other information: *Abnormal swallowing syndrome is a condition during sleep in which inadequate swallowing results in aspiration of saliva, coughing, and choking. It is intermittently associated with brief arousals or awakenings.*

Patients report the subjective sense in sleep of choking and blocked breathing, but polysomnographic recording demonstrates no pathological sleep apnea, only

short-lived episodes of coughing and gagging following periods of "gurgling" sounds (the latter are probably due to pooling of saliva in the hypopharynx). The quality of sleep is restless and in certain cases appreciably disturbed. However, symptoms do not occur at sleep onset, and they cease momentarily with awakening, so that return to sleep is unimpeded.

Elderly patients with this condition may be prone to respiratory infections due to aspiration. Vulnerability is increased with administration of hypnotic agents.

Differential diagnosis: The nature of the description of arrested respiration during sleep raises the likelihood of confusion with obstructive upper airway syndrome (see B.4.a). However, in this sleep apnea condition, patient awareness and mention of breathing difficulty is, in fact, unusual. Further, the presenting symptom is almost invariably excessive daytime sleepiness, whereas if a sleep problem is reported in connection with abnormal swallowing syndrome, it is restlessness or insomnia. Polysomnographic monitoring will rule out obstructive sleep apnea and, additionally, central sleep apnea (A.4.a), which—like abnormal swallowing syndrome—may present with disturbed sleep.

Sleep terror attacks (D.2) are characteristically associated with sensations of impaired breathing or choking during sleep (incubus), as well as with panting, agitation, and rapid pulse on awakening. However, they occur in delta sleep and generally only once per night. The inaugural scream and extreme dread of sleep terror separates this condition from the abnormal swallowing syndrome.

Episodes of gastroesophageal reflux (D.4.1) may also lead to coughing and choking during sleep, but typically the awakenings are associated with heartburn and chest pain as well. Evidence of acid reflux and esophagitis suffice to distinguish this disorder from abnormal swallowing syndrome.

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D. 4. j. Sleep-related Asthma

Key words and phrases: asthma, nocturnal asthma, randomly distributed in sleep except stage 4.

Essential features: *Asthma—a chronic respiratory disease consisting of sporadic paroxysms of dyspnea, wheezing, and expectoration of thick sputum because of spasm, swelling of the lining, and resultant narrowing of the small bronchi—appears to be a sleep-exacerbated condition. Its distribution favors sleep and may result in significant sleep disturbance in certain individuals.*

Though a relationship of asthmatic episodes to REM sleep (and to dreaming NREM sleep) has been suggested, studies of greater numbers of adults and children have not confirmed this association. All studies agree that the early portion of the sleep period, particularly deep NREM (stages 3/4) sleep, is spared from attacks. Following the first hour(s) of sleep, attacks occur in evident randomness

with respect to stage of sleep and remaining time of night. Partial sleep loss and reduction of stage 4 sleep is observed, but there is no loss of stage REM sleep.

Associated features and other information: Several purported factors have been advanced to explain the apparent affiliation between sleep and asthma episodes. Among these are the biological rhythms of cholinesterase, catecholamines, and adrenal steroids. (However, the circadian corticosteroid curve would predict the highest and lowest incidences of attacks in the middle and latter thirds of the night, respectively, a prediction not borne out by the data.) Such factors as the recumbent position (elevated airway resistance) and dreaming have also been implicated as triggering phenomena. A clear or logical association of asthma with a presumed, sleep-related instigator is as yet inapparent. The psychogenic theory that relates nocturnal asthma to disturbing dreams also seems to be incompletely documented and, at best, only partially operative.

The findings that stage 4 sleep is devoid of asthma attacks but is reduced in proportion in asthmatic patients may be of relevance in deciding whether asthma does not appear in stage 4 because it is *not triggered* during this stage or *not responded* to in deep NREMS. The latter explanation is suggested by the additional finding that these patients, though partially sleep deprived, have reduced stage 4. This reduction could be caused by the effect of subclinical asthmatic symptoms during stage 4, sufficient to disturb the stage but not sufficiently distressing to awaken the patient.

Differential diagnosis: Bronchial asthma attacks during sleep must be distinguished from cardiovascular problems such as paroxysmal nocturnal dyspnea and "cardiac" asthma (see D.4.k). Polysomnography will help to separate asthma from sleep-induced apnea syndromes (A.4.a and B.4.a), which can also present with snoring, gasping, wheezing, coughing, and interruption of breathing. A sense of inability to breathe, breathing noises, choking, and coughing characterize the abnormal swallowing syndrome (D.4.i) and, with the addition of chest pain, also describe gastroesophageal reflux (D.4.l).

Choking, respiratory distress, anxiety, rapid pulse, and sympathetic discharge are characteristic of sleep terror (D.2), a syndrome that, like asthma, is frequently seen in children. However, the latter is little represented in the first third of the night when sleep terror episodes are at their peak. The respiratory distress of sleep-related asthma may be imitated in brief periods by sleep-related epileptic seizures (D.4.b).

If sleep-related asthma results in a significant complaint of DIMS, the latter, as provoked by asthma, should be additionally coded under DIMS (A.6).

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D. 4. k. Sleep-related Cardiovascular Symptoms

Key words and phrases: sleep-related cardiovascular symptoms, nocturnal cardiac symptoms, angina pectoris, paroxysmal nocturnal dyspnea, orthopnea, congestive heart failure, pulmonary edema, strokes, arrhythmias.

Essential features and other information: *Sleep-related cardiovascular symptoms derive from disorders of cardiac rhythm, myocardial incompetence, coronary artery insufficiency, and blood pressure variability, which may be induced or exacerbated by sleep-altered or sleep-stage-modified cardiovascular physiology.*

Because of cardiac rate and hemodynamic changes in REM sleep noted in normal subjects monitored polysomnographically, e.g., increases and enhanced variability in pulse and blood pressure, the expectation has existed that cardiovascular symptoms would be prominent during the REM sleep of individuals with marginal or compromised cardiac functioning.

Certain cardiovascular signs have been traditionally linked to periods of sleep. In situations of myocardial or valvular incompetence, *paroxysmal nocturnal dyspnea*[1] (perhaps due to recumbency not sleep) is commonly observed, signaling acute worsening of *congestive heart failure*. With a feeling of suffocation, and gasping for breath, the patient wakes from sleep and sits up. On occasion, this is a prelude to *pulmonary edema* manifested by "air hunger," coughing, and wheezing (*cardiac asthma*). *Orthopnea* indicates a sitting-up position during sleep. It is seen in patients in whom heart failure has made gravitational factors sufficiently critical so that they cannot breathe comfortably when recumbent. The incidence of *cerebrovascular accidents* is greater in sleep than in the awake state.

Early observations put the peak time of cardiac deaths (attributable in part to *myocardial infarction*) during sleep at the REM-sleep-rich interval of 5–6 a.m. Observations on *nocturnal angina* have repeatedly associated this syndrome of nocturnal pain due to coronary artery spasm with restless sleep and dreaming, presumably REM sleep.

The first polysomnographic study of angina patients was therefore welcome in finally affirming the decided predilection of anginal episodes for REM sleep. Other investigations yielded similar results, except for discovering that a small proportion of episodes occur in deep NREM (stages 3/4) sleep. The latter attacks were attributed to the low systemic blood pressure prevailing during that sleep stage.

However, recent reports have not consistently supported an association of nocturnal angina and S-T segment EKG changes with REM sleep. Additional research is required. Also, the very few studies of hemodynamic changes during sleep in hypertensive patients have not established that clinically significant increases in blood pressure occur in REM sleep. The incidence in sleep and wakefulness of premature ventricular beats and premature atrial contractions is about

[1] Italicized terms relate to accepted medical diagnoses, symptoms, or signs.

equal (perhaps somewhat smaller in sleep), though premature ventricular beats may be distributed more heavily in REM sleep periods than in other sleep stages.

The growing number of hemodynamic and cardiac studies carried out in patients with sleep-induced respiratory impairment (A.4 and B.4) points to an unmistakable association of reduced blood oxygenation and serious cardiac dysrhythmias during sleep.

It is apparent that the widely divergent data concerning cardiovascular symptoms in sleep partly derives from studies that have used different types of patients, with varying and often unspecified degrees of cardiovascular pathology, subjected to different conditions and techniques of study, and frequently unmonitored for sleep respiration. Nonetheless, it appears that a considerable number of patients at certain times appear to have exacerbations of cardiovascular symptoms during sleep.

Differential diagnosis: *Chest pain on awakening* is typically seen in gastroesophageal reflux (D.4.1). *Difficulties in breathing during sleep* may be observed (or sensed by the patient) in sleep apnea DIMS and DOES syndromes (A.4.a; B.4.a), sleep-related asthma (D.4.j), abnormal swallowing syndrome (D.4.i), sleep-related epileptic seizures (D.4.b), and sleep terror (D.2). In the latter, arousal is accompanied as well by a *marked tachycardia*. Such symptoms may be mimicked in several psychiatric disturbances (see A.1.b and A.2.a,b,c).

Patients with obstructive sleep apnea (B.4.a) and persistent psychophysiological DIMS (specifically, conditioned "insomnia"), for very different reasons, sleep "sitting up." These conditions should be considered in the evaluation of orthopnea. *Dysrhythmias* are characteristic of impaired, sleep-induced respiration (A.4 and B.4) with hypoxemia.

If a cardiovascular symptom results in a complaint of insomnia, it should be additionally coded as provoking DIMS, under A.6.

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D. 4. 1. Sleep-related Gastroesophageal Reflux

Key words and phrases: gastroesophageal reflux, heartburn waking up from sleep, coughing and choking in sleep.

Essential features: *Sleep-related gastroesophageal reflux is a disorder in which the patient awakens from sleep either with burning, substernal pain, a*

feeling of general pain or tightness in the chest, or a sour taste in the mouth. Coughing, choking, and vague respiratory discomfort may also be repetitive occurrences with this syndrome.

The condition may occur during sleep either in individuals devoid of symptoms in the awake state, or in patients who have known difficulties during waking with heartburn (cardialgia), postprandial regurgitation, dysphagia, esophagitis, or laryngopharyngitis. It is diagnosed by means of a positive Bernstein (acid infusion) test; an overnight pH study; a standard acid clearance test to appraise acid clearance during sleep; a standard acid reflux test; or endoscopic or histological evidence of esophagitis.

Associated features and other information: Sleep-related gastroesophageal reflux initially manifests itself only in an occasional awakening from sleep with chest discomfort or heartburn. Over a variable period of time, symptoms become more severe. The number and severity of symptomatic awakenings from sleep are correlated with progression of the reflux towards physical sequelae such as esophagitis or tracheal and pulmonary aspiration. These may develop into serious complications (e.g., esophageal stricture, aspiration pneumonia, bronchiectasis, exacerbation of asthma, or laryngopharyngitis).

This condition is seen almost always in adults, both young and old. An unusually low pressure at the lower esophageal sphincter may predispose to the disorder. Gastroesophageal reflux may be one of the more common gastrointestinal disorders, but it often goes unrecognized. It affects both males and females and is without a familial pattern.

Differential diagnosis: The primary diagnoses to differentiate are chest pain of cardiac origin (see D.4.k) and other peptic disorders such as duodenal ulcer disease. The coughing and choking, which are frequently noted with gastroesophageal reflux, may lead to confusion with sleep-related asthma (D.4.j), obstructive sleep apnea syndrome (B.4.a), abnormal swallowing syndrome (D.4.i), or paroxysmal nocturnal dyspnea (D.4.k).

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D. 4. m. Sleep-related Hemolysis (Paroxysmal Nocturnal Hemoglobinuria)

Key words and phrases: paroxysmal nocturnal hemoglobinuria, sleep-related hemolysis.

Essential features: *Paroxysmal nocturnal hemoglobinuria is a rare, acquired, chronic hemolytic anemia in which intravascular hemolysis results in hemoglobinemia and hemoglobinuria. The hemolysis and consequent hemoglobinuria are accelerated during sleep, coloring the morning urine a brownish-red. Hemolysis is linked to the sleep period even if the latter is acutely shifted.*

The fundamental abnormality is in the membrane of the erythrocyte, rendering the cell extraordinarily sensitive to the normal lytic action of serum complement.

Because of this sensitivity, a traditional diagnostic test for the disease—the Ham test—induces hemolysis in the patient's red blood cells by slight acidification of the serum (any activator of serum complement will serve the same purpose). The serum may come either from the patient or from a normal donor. The patient's erythrocytes do not have a longer life span when transfused into a normal individual than in the patient. Accordingly, no disorder of the complement system exists.

Studies relating the course of hemoglobinemia to specific sleep stages have not been carried out. It is possible that the serum is altered during a particular phase of sleep, rendering complement more active and provoking erythrocyte disintegration. Sleep, not simply recumbency or rest, appears to be necessary for the reaction, but studies have not been performed that incontrovertibly establish the association with sleep.

In classic cases, a clinical picture of weakness, pallor, and anemia is coupled with the complaint of dark urine in the morning after sleep. Hemoglobinuria is sometimes the first sign of the disease.

Associated features and other information: Paroxysmal nocturnal hemoglobinuria, though characterized by irregular exacerbations, is chronic and persistent. The disorder ordinarily appears between ages 20–40 but may occur in childhood and old age. In severe cases, hemolysis is extensive and transfusions are required; in some patients the hemoglobinemia and anemia are seen daily but lessen during waking hours. A few spontaneous remissions have been reported, but generally the disease is fatal after a number of years. Exacerbations are associated with infection, menstruation, surgery, transfusions, vaccination, oral iron, and liver extract injections, but the specific cause of the worsenings has not been determined. Thromboses, infections, and anemia are the causes of death in most instances. There is no racial, geographic, hereditary, or gender predominance.

Differential diagnosis: In most hemolytic anemias, hemolysis takes place extravascularly (in the reticuloendothelial system) and hemoglobinemia is not a finding. Nevertheless, all hematological conditions leading to discoloration of urine must be ruled out, as well as acute porphyria and hematuria.

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D. 4. n. Asymptomatic Polysomnographic Finding

Key words and phrases: snoring without sleep apnea, snoring disclosing asymptomatic sleep apnea, sleep apnea without complaint, alpha–delta sleep without complaint, sleep-related (nocturnal) myoclonus without complaint.

Essential features: *This category is reserved for classifying features,*

noted incidentally in polysomnographic recording, which are usually associated with DIMS or DOES but are not the subject of a complaint and do not appear to be causing symptoms in the individual.

Such polysomnographic disclosures may be made in the course of screening individuals for a sleep research study or during evaluation in a clinical sleep disorder center of a symptom completely unrelated to the unexpected and asymptomatic finding. For example, a patient being monitored for bruxism may be discovered to have brief, infrequent, but definite episodes of central sleep apnea which cause no apparent disturbance and are out of the individual's awareness.

In addition to snoring and sleep apnea (A.4.a or B.4.a), sleep-related (nocturnal) myoclonus (A.5.a or B.4.a) may appear in the absence of excessive daytime sleepiness or insomnia. Alpha–delta sleep (A.8.b) is another phenomenon revealed in sleep recordings that may have no symptom counterpart.

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Sleep, Volume 2, Number 1

Glossary of Terms Used in the Sleep Disorders Classification

Alpha activity The presence of alpha waves or alpha rhythm (i.e., electrical oscillations in the alpha frequency) in the EEG of humans. *See* Alpha rhythm.

Alpha intrusion (-infiltration, -insertion, -interruption; "riddled with" alpha) A brief interposition of alpha activity during a stage of sleep.

Alpha rhythm (-activity, -frequency) EEG oscillations with a frequency of 8–13 Hz in adults, prominent over the occipital cortex; indicative of the awake state in humans; present in most, but not all, normal individuals; most consistent and predominant during relaxed wakefulness, particularly with reduction of visual input. The alpha frequency has a range in each individual: the low end is exhibited in drowsiness or sleep and the upper end with alertness. The frequency range also varies with age; it is slower in children and older age groups relative to young and middle-aged adults.

Arousal An abrupt change from a "deep" stage of NREM sleep to a "lighter" stage, or from REMS to awake, with the possibility of awakening as the final outcome. Arousal may be accompanied by increased tonic EMG activity and heart rate, as well as body movements.

Awakening (full arousal) The return to the polysomnographically defined awake state from any of the NREM stages or REMS; characterized by alpha and beta waves, rise in tonic EMG, voluntary REMs, and eye blinks. This definition of awakening is valid only insofar as the polysomnogram is matched by a resumption of a reasonably alert state of consciousness.

Base line The normative (i.e., typical) state of an individual or of an investigative parameter prior to an experimental manipulation.

Bedtime Defined as the time when one attempts to fall asleep (as distinguished from the time when one gets into bed).

Beta rhythm (-waves, -activity) EEG frequency in the range of 13–35 Hz; when the predominant frequency, beta rhythm is usually associated with alert wakefulness or vigilence and is accompanied by a high tonic EMG.

Cataplexy A sudden, dramatic decrement in muscle tone and loss of deep reflexes leading to muscle weakness, paralysis, or postural collapse; usually precipitated by an outburst of emotional expression—notably laughter, startle, or sudden physical exercise; one of the tetrad of symptoms of narcolepsy. During cataplexy, respiration is not compromised.

Cheyne-Stokes respiration A breathing pattern characterized by regular "crescendo–decrescendo" fluctuations in respiratory rate and tidal volume.

Circadian rhythm An innate, daily, fluctuation of physiological and behavioral functions, including sleep–waking; generally tied to the 24 hour day–night cycle but sometimes to a measurably different (e.g., 23 or 25 hour) periodicity when light/dark and other time cues are removed.

Conditioned insomnia An easily overlooked form of chronic insomnia (sometimes a component of psychophysiological DIMS) caused by the development—during an earlier experience of sleeplessness—of a negative association between characteristics of the customary sleep environment and sleeping.

"Deep" sleep stage Common term for NREM stages 3 and 4 sleep. In some European sleep literature, "deep" sleep is applied to REM sleep because of its high awakening threshold. *See* "Intermediary" sleep stage; "Light" sleep stage.

Delayed sleep phase A condition that occurs when the clock hour at which sleep normally occurs is moved back in time in a given, 24 hour sleep–wake cycle. This results in a temporarily displaced (delayed) occurrence of sleep within the 24 hour cycle. The same term denotes a chronic sleep schedule disturbance (category C.2.b).

Delta sleep stage(s) Indicative of the stage(s) of sleep in which EEG delta waves are prevalent or predominant (sleep stages 3 and 4, respectively). *See* Slow wave sleep.

Delta waves EEG activity with a frequency of less than 4 Hz. In human sleep scoring, the minimum characteristics for scoring delta waves is conventionally 75 μV (peak-to-peak) amplitude, and 0.5 second duration (2 Hz).

Dyssomnia Any disorder of sleep or wakefulness per se; not a parasomnia.

Early a.m. arousal Synonymous with premature morning awakening.

Electroencephalogram (EEG) A recording through the scalp of the electrical potentials from the brain and the moment-to-moment changes in these potentials. With the EMG and EOG, the EEG is one of the three basic variables used to score sleep stages and waking. Sleep recording in humans utilizes surface electrodes to record potential differences between brain regions and a neutral reference point, or simply between brain regions. Either the C3 or C4, central region, placement in the International 10-20 system is the standard electrode from which stage scoring is done.

Electromyogram (EMG) A recording of electrical activity from the muscular system; in sleep recording, synonymous with resting muscle activity or potential. The chin/cheek EMG, along with EEG and EOG, is one of the three basic variables used to score sleep stages and waking. Sleep recording in humans utilizes surface electrodes to measure activity from the submental or masseter muscles. These reflect maximally the changes in resting muscle activity. The chin/cheek EMG is tonically inhibited during REM sleep.

Electro-oculogram (EOG) A recording of voltage changes resulting from shifts in position of the eyeball—possible because each globe is a positive (anterior) and negative (posterior) dipole; along with the EEG and the EMG, one of the three basic variables used to score sleep stages and waking. Sleep recording in humans utilizes surface electrodes placed near the eyes to record the movement (incidence, direction, and velocity) of the eyeballs. Rapid eye movements in sleep indicate a certain stage of sleep (REM sleep).

Excessive daytime sleepiness or somnolence A subjective report of difficulty in maintaining the awake state, accompanied by a ready entrance into sleep when the individual is sedentary; may be quantitatively measured by use of subjectively defined rating scales of sleepiness.

Fragmentation (pertaining to sleep architecture) The interruption of any stage of sleep due to appearance of another stage or waking, leading to disrupted NREMS–REMS cycles; often used to refer to the interruption of REMS by movement arousals or stage 2 activity. *Sleep* fragmentation connotes repetitive interruptions of sleep by arousals and awakenings.

Hertz (Hz) A unit of frequency; synonymous with cycles per second (cps).

Hypercapnia Elevated carbon dioxide level in blood.

Hypersomnia Excessive or prolonged sleep. Sometimes associated with difficulty in awakening or sleep drunkenness.

Hypnagogic imagery (-hallucinations) Vivid sensory images occurring at sleep onset but particularly vivid with sleep-onset REMS periods. A feature of narcoleptic REMS naps.

Hypnagogic startle A "sleep start" or sudden body jerk, observed normally just at sleep onset and resulting in at least momentary awakening.

Insomnia Difficulty in sleeping. A confusing term—though ubiquitously employed—because it is used to indicate any and all gradations and types of sleep loss.

"Intermediary" sleep stage A term used for NREM stage 2 sleep. *See* "Deep" sleep stage; "Light" sleep stage.

Internal arousal insomnia A form of chronic insomnia (sometimes a component of psychophysiological DIMS) resulting from excessive mental activity; induced by too conscious efforts to sleep and underlying apprehension that all attempts will fail.

K complex A sharp, negative, high-voltage EEG wave, which is followed by a slower, positive component. K complexes occur spontaneously during NREM sleep, beginning in (and defining) stage 2. They are thought to be CNS-evoked responses to internal stimuli. They can be elicited during sleep by external (particularly auditory) stimuli as well.

"Light" sleep stage Common term for NREMS stage 1 (and sometimes stage 2). *See* "Deep" sleep stage; "Intermediary" sleep stage.

Microsleep(s) A period lasting up to a few seconds during which the polysomnogram suddenly shifts from waking characteristics to sleep and external stimuli are not perceived; associated with excessive daytime sleepiness and automatic behavior, which are symptoms of DOES.

Movement arousal A body movement associated with arousal or awakening; a sleep scoring variable.

Movement time The term used in sleep record scoring to denote when EEG and EOG tracings are obscured for more than 15 seconds because of movement. Usually combined with awake time.

Multiple sleep latency test A series of measurements of the interval from "lights out" to sleep onset that is utilized in the assessment of excessive daytime sleepiness. Subjects are allowed a fixed number of opportunities to fall asleep during their customary awake period. Long latencies are helpful in distinguishing physical tiredness or fatigue from true sleepiness.

Muscle tone A term sometimes used for resting muscle potential or resting muscle activity. *See* Electromyogram (EMG).

Myoclonus Muscle contractions in the form of "jerks" or twitches. In sleep-related (nocturnal) myoclonus, the jerks are primarily of the flexor groups in the lower extremities and have a characteristic frequency of 20–40 seconds.

Nightmare Used to denote a dream anxiety attack, not a sleep (night) terror. In the past—and still in the European sleep literature—nightmare is used to indicate both sleep terror and anxiety dream.

Nocturnal confusion Episodes of delirium and disorientation close to or during nighttime sleep; often seen in the elderly and indicative of organic CNS deterioration; now referred to as the "sundown syndrome."

Nocturnal dyspnea Respiratory distress, minimal during the day but becoming quite disturbing in sleep.

Nocturnal sleep Indicative of the typical "nighttime," or major, sleep period dictated by one's circadian rhythm of sleep and wakefulness; the conventional time for sleeping.

Non-rapid eye movement sleep (NREMS, also written as non-REMS) *See* Sleep stages.

NREMS intrusion An interposition of NREM sleep, or a component of NREMS physiology (e.g., elevated EMG, K-complex, sleep spindle, delta waves), in REMS; a portion of NREMS not appearing in its usual sleep cycle position.

NREMS period The NREMS portion of NREMS–REMS cycle; such a period consists primarily of sleep stages 3/4 early in the night and of sleep stage 2 later. *See* Sleep cycle; Sleep stages.

NREMS–REMS cycle (synonymous with sleep cycle) A period during sleep composed of a NREMS period and the subsequent REMS period; each NREMS–REMS couplet is equal to one cycle. Any NREM sleep stage suffices as the NREMS portion of a cycle. A sleep period of 6.5–8.5 hr generally consists of four to six cycles.

Parasomnia Not a disorder of sleep or wakefulness per se; rather, an event happening *during* sleep, or induced or exacerbated *by* sleep, such as sleepwalking or asthma; not a dyssomnia.

Paroxysmal nocturnal dyspnea (PND) Respiratory distress and shortness of breath due to pulmonary edema, which appear suddenly and often awaken the sleeping individual.

Period length The duration of time encompassed by an individual's full, daily sleep–wake cycle; conventionally, but not always, 24 hours. *See* Circadian rhythm.

Phase advance The movement to a position *earlier* in the 24 hour sleep–wake cycle of a period of sleep or wake; for example, a shift of the sleep phase from 11 p.m.–7 a.m. to 8 p.m.–4 a.m. *See* Phase delay.

Phase delay Phase delay is exactly the opposite of phase advance, i.e., a shift *later* in time. (Confusion is sometimes introduced into these concepts because clock language is reversed; to effect a phase *delay*, the clock is moved *ahead* or *advanced*. Contrariwise, as in the example in phase *advance*, the change from the 11 p.m.–7 a.m. to the 8 p.m.–4 a.m. position requires a movement of the clock *backward*.) *See* Phase advance.

Phase transition One of the two junctures of the major sleep and wake phases in the 24 hour sleep–wake cycle.

Phasic event (-activity) Brain, muscle, or autonomic events of an episodic or fluctuating nature occurring in sleep; characteristic of REMS (e.g., eye movements, muscle twitches); usually enduring for milliseconds to 1–2 seconds.

Polysomnogram The continuous and simultaneous recording of physiological variables during sleep, i.e., EEG, EOG, EMG (these are the three basic stage scoring parameters), EKG, respiratory air flow, respiratory excursions, lower limb movement, and other electrophysiological variables.

Polysomnographic (as in -recording, -monitoring, -registration, or -tracings) Describes a paper or FM tape record of a polysomnogram.

Premature morning awakening Early termination of the sleep period in a sleep-maintenance DIMS due to inability to return to sleep after the last of several awakenings; typifies the failure to accomplish a normal length of nocturnal sleep because of interference at the end rather than at the commencement of sleep; the DIMS characteristic of depressed individuals.

Rapid eye movement sleep (REMS) *See* Sleep stages.

REM density (-intensity) A function that expresses the frequency of eye movements during sleep stage REM.

REMS intrusion A brief interval of REMS appearing out of its usual position in the NREMS–REMS cycle; an interposition of REMS in NREMS; sometimes appearance of a single, dissociated component of REMS (e.g., eye movements, or "drop out" of muscle tone) rather than all REMS parameters.

REMS latency The period of time in the sleep period from sleep onset to the first appearance of stage REMS.

REMS onset The designation for commencement of a REMS period. Sometimes also used as a shorthand term for a sleep-onset REMS period. *See* Sleep onset; Sleep-onset REMS period.

REMS percent The proportion of total sleep time constituted by the REM stage of sleep.

REMS period The REMS portion of a NREMS–REMS cycle; early in the night it may be as short as a half-minute, whereas in later cycles longer than an hour. *See* Sleep stage REM.

REMS rebound or recovery Lengthening and increase in frequency and density of REMS periods, which results in an increase in REMS percent above base line. REMS rebound follows REMS deprivation once the inhibitory influence is removed.

Restlessness (referring to a quality of sleep) Persistent or recurrent body movements, arousals, and brief awakenings in the course of sleep.

Sleep architecture The NREMS/REMS stage and cycle infrastructure of sleep understood from the vantage point of the quantitative relationship of these components to each other. *See* Sleep structure.

Sleep cycle Synonymous with NREMS–REMS cycle.

Sleep efficiency (or sleep efficiency index) The proportion of sleep in the period potentially filled by sleep; that is, the ratio of *total sleep time* to *time in bed*.

Sleep hygiene The conditions and practices that promote continuous and effective sleep. These include regularity of bedtime and arise time; conformity of time spent in bed to the time necessary for sustained and individually adequate sleep (i.e., the total sleep time sufficient to avoid sleepiness when awake); restriction of alcohol and caffeine beverages in the period prior to bedtime; employment of exercise, nutrition, and environmental factors so that they enhance, not disturb, restful sleep.

Sleep interruption Breaks in the sleep architecture resulting in arousal and wakefulness. *See* Fragmentation; Restlessness.

Sleep latency The period of time measured from "lights out," or bedtime, to the commencement of sleep.

Sleep log (-diary) A daily, written record of an individual's sleep–wake pattern containing such information as time of retiring and arising, time in bed, estimated total sleep period, number and duration of sleep interruptions, quality of sleep, daytime naps, use of medications or caffeine beverages, nature of waking activities, and other data.

Sleep-maintenance DIMS or insomnia A disturbance in maintaining sleep once achieved; persistently interrupted sleep without difficulty falling asleep. Synonymous with sleep continuity disturbance.

Sleep mentation The imagery and thinking (and emotion) experienced during sleep. Sleep mentation consists of individual representations—but usually combinations of—images and thoughts. Imagery is vividly expressed in dreams during REMS in all the senses in approximate proportion to their waking representations. Mentation is experienced generally less distinctly in NREM sleep, but it may be quite vivid in stage 2 sleep, especially toward the end of the sleep period.

Sleep onset The transition from the awake to the sleep state, normally into NREM stage 1 (but in certain conditions, such as infancy and narcolepsy, into stage REMS). Most polysomnographers accept EEG slowing, reduction, and eventual disappearance of alpha activity, presence of EEG vertex spikes, and slow rolling eye movements (the components of NREM stage 1) as sufficient for sleep onset; others require appearance of stage 2 wave forms. Consciousness has been shown to be lost as alpha activity fragments. *See* Sleep latency; Sleep stages.

Sleep-onset REMS period The atypical beginning of sleep by entrance directly into stage REMS.

Sleep pattern (24 hour sleep–wake pattern) An individual's clock hour schedule of bedtimes and rise times as well as nap behavior; may also include time and duration of sleep interruptions. *See* Sleep–wake, 24-hour cycle; Circadian rhythm; Sleep log.

Sleep spindle An episodically appearing, spindle-shaped aggregate of 12–14 Hz waves

with a duration of 0.5–1.5 seconds; one of the identifying EEG phenomena of NREM stage 2 sleep; may persist into NREM stages 3/4; not seen in REMS.

Sleep stage demarcation The significant polysomnographic characteristics that distinguish the boundaries of the sleep stages. In certain conditions and with drugs, sleep stage demarcations may be blurred or lost, making it difficult to identify certain stages with certainty or to distinguish the temporal limits of sleep stage lengths.

Sleep stage period A sleep stage interval that represents the stage in a NREMS–REMS cycle; easiest to comprehend in relation to REMS, which is a homogeneous stage, i.e., the fourth REMS period is in the fourth sleep cycle. If one interval of REMS separated from another by more than 20 minutes, they constitute separate REMS periods (and are in separate sleep cycles); a sleep stage period may be any duration.

Sleep stages:

Sleep stage NREM (NREMS) The other major sleep state apart from REMS; comprises sleep stages 1–4, which constitute areas in the spectrum of NREMS "depth" or physiological intensity.

Sleep stage 1 (NREMS stage 1) A stage of NREM sleep that ensues directly from the awake state. Its criteria consist of a low-voltage EEG with slowing to theta frequencies, alpha activity less than 50%, EEG vertex spikes, and slow rolling eye movements; no sleep spindles, K complexes, or REMs. Stage 1 normally assumes 4–5% of total sleep.

Sleep stage 2 (NREMS stage 2) A stage of NREM sleep characterized by the advent of sleep spindles and K complexes against a relatively low-voltage, mixed-frequency EEG background; high-voltage delta waves may comprise up to 20% of stage 2 epochs; usually accounts for 45–55% of total sleep time.

Sleep stage 3 (NREMS stage 3) A stage of NREM sleep defined by at least 20 and not more than 50% of the period consisting of EEG waves less than 2 Hz and more than 75 μV (high-amplitude delta waves); a "delta" sleep stage; with stage 4, it constitutes "deep" NREM sleep; often combined with stage 4 into NREMS stage 3/4 because of the lack of documented physiological differences between the two; appears usually only in the first third of the sleep period; usually comprises 4–6% of total sleep time.

Sleep stage 4 (NREMS stage 4) All statements concerning NREMS stage 3 apply to stage 4 except that high-voltage, slow EEG waves, cover 50% or more of the record; NREMS stage 4 usually takes up 12–15% of total sleep time. Somnambulism, sleep terror, and sleep-related enuresis episodes generally start in stage 4 or during arousals from this stage. *See* Sleep stage 3.

Sleep stage REM (REMS) The stage of sleep (i.e., state of the CNS) found in all mammals studied, including man, in which brain activity is extensive, brain metabolism is increased, and vivid hallucinatory imagery, or dreaming occurs (in humans). It is also called "paradoxical sleep" because, in the face of this intense excitation of the CNS and presence of spontaneous rapid eye movements, resting muscle activity is suppressed. The EEG is a low-voltage, fast-frequency, nonalpha record. Stage REMS is usually 20–25% of total sleep time.

Sleep structure Similar to sleep architecture. However, sleep structure—in addition to encompassing sleep stage and cycle relationships—assesses the within-stage qualities of the EEG and other physiological attributes.

Sleepiness (somnolence, drowsiness) Difficulty in maintaining the wakeful state so that the individual falls asleep if not actively kept aroused; not simply a feeling of physical tiredness or listlessness. *See* Excessive daytime sleepiness.

Sleep talking Talking in sleep takes place during stage REMS, at which time it represents a motor breakthrough of dream speech, or in the course of transitory arousals from NREMS and other stages. Full consciousness is not achieved and no memory of the event remains.

Sleep–wake, 24 hour cycle Basically, the clock hour relationships of the major sleep and wake phases in the 24 hour cycle; similar to sleep pattern. *See* Phase transition; Circadian rhythm.

Sleep–wake shift (-change, -reversal) When sleep–wholly or partially–is moved to a time of customary waking activity, and the latter is moved to the habitual sleep period; common in jet lag and shift work.

Slow wave sleep (SWS) Synonymous with sleep stages 3 and 4. *See* Delta sleep stage(s).

Snoring A noise produced primarily with inspiratory respiration during sleep owing to vibration of the soft palate and the pillars of the oropharyngeal inlet. Many snorers have incomplete obstruction of the upper airway, and may in time develop frank obstructive sleep apnea.

Spindle REMS A condition in which sleep spindles persist atypically in REMS; seen in chronic DIMS conditions.

Subwakefulness syndrome A syndrome postulated as a defect in the CNS support system for waking. The few individuals reported with subwakefulness syndrome have daytime drowsiness and daytime sleep episodes that are always composed of NREMS stages 1 or 2. The naps occur repetitively.

Theta waves EEG activity with a frequency of 4–8 Hz, maximal over temporal cortex.

Total sleep period The period of time measured from sleep onset to final awakening. In addition to total sleep time, it is comprised of the time taken up by arousals and movement time until wake-up. *See* Sleep efficiency.

Total sleep time The amount of actual sleep time in a sleep period; equal to total sleep period less movement and awake time. Total sleep time is the total of all REMS and NREMS in a sleep period.

Tumescence (penile) Hardening and expansion of the penis; penile erection. Commonly referred to as nocturnal penile tumescence (NPT) in sleep recordings.

Twitch (body twitch) A very small body movement such as a facial grimace or finger jerk; not usually associated with arousal.

Wake time The total time that is scored awake in a polysomnogram occurring between sleep onset and final wake-up.

List of Abbreviations

CNS	Central nervous system	EOG	Electro-oculogram
cps	Cycles per second	Hz	Hertz
DIMS	Disorders of initiating and maintaining sleep	NPT	Nocturnal penile tumescence
		NREMS	Non-rapid eye movement sleep
DOES	Disorders of excessive somnolence	PND	Paroxysmal nocturnal dyspnea
		REM(s)	Rapid eye movement(s)
EEG	Electroencephalogram	REMS	Rapid eye movement sleep
EMG	Electromyogram	SWS	Slow wave sleep

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ASDC–APSS Diagnostic Classification of Sleep and Arousal Disorders in Relation to the International Classification of Diseases (ICD-9-CM)

In the following two tables, the codings of the sleep disorders classification system of the Association of Sleep Disorders Centers (ASDC) and the Association for the Psychophysiological Study of Sleep (APSS) are paired with recommended equivalent codes from the *International Classification of Diseases, 9th Revision, Clinical Modification* (ICD-9-CM).* ICD-9-CM is an adaptation of the World Health Organization's *International Classification of Diseases, 9th Revision.* The ICD-9-CM was developed by the Council on Clinical Classifications under the guidance of a broadly representative committee authorized by the WHO Center for Classification of Diseases for North America sponsored by the National Center for Health Statistics. It was intended to provide more clinical detail than ICD-9 in the classification and indexing of conditions of illness. The coding in ICD-9-CM is entirely compatible with ICD-9 except for the use of four and five digits—in place of three or four, respectively—which allows inclusion of additional conditions and clinical variations of disease entities.

The sleep disorders listings, with the exception of narcolepsy and several other conditions, appear in ICD-9-CM, Tabular List (volume 1), in two sections: chapter 5. *Mental Disorders,* under codes 307.40–307.49, Specific disorders of sleep of nonorganic origin (pp. 237–238), and in chapter 16. *Symptoms, Signs, and Ill-Defined Conditions,* under codes 780.50–780.59, Sleep disturbances (pp. 708–709).

In ICD-9-CM, sleep disorders of a functional nature are contained in category 307.4, whereas nonspecific, or presumably organically based, sleep symptoms are placed in category 780.5. By the time, in 1977, that the committee on sleep disorders classification of the ASDC began consulting with the editors of ICD-9-CM in an effort to include our classification schema, the basic organization of the sleep disturbances into the same two sections in ICD-9 and ICD-9-CM had already been established. Nevertheless, because of the cooperation of the staff of ICD-9-CM, a few changes and additions still proved possible in the two sections. The alterations, though few in number, were significant. They allowed entry of new terminology, and for the first time, because of the use of all the fifth digits, inclusion of a number of important, additional conditions in the published version of ICD-9-CM, such as sleep apnea DIMS and DOES syndromes, DIMS with atypical polysomnographic features, repeated REM sleep interruptions, and others.

Equally important for an often computerized, internal data base, a method was devised so that the ICD-9-CM coding system, which still does not allow separate discrimination of the full range of ASDC-APSS conditions, may be expanded,

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when necessary, by addition of a suffix numeral. This system now permits *every* condition contained in the ASDC-APSS nosology to be matched by a unique ICD-9-CM–based designation. In the expanded all-numeral system, that portion of a code that appears after a hyphen (the suffix numeral) represents an unauthorized designation—an addition grafted onto the official ICD-9-CM code to increase nosological specificity. Accordingly, the ASDC-APSS diagnoses must be entered only in the form of the compatible, authentic ICD-9-CM rubrics when diagnostic information is filed in hospital records and with insurers and public health agencies; however, diagnoses may be entered as individualized ICD-9-CM–rooted code numbers (in the place of ASDC-APSS outline designations) when the recording is into internal data systems.

It is important that certain limitations, as well as liberties, in the creation of these comparative code classifications be pointed out. It was at times difficult for the ASDC committee to decide how to fit the ASDC classification conditions in logical positions within the current, two-category ICD-9-CM sleep disorders framework. The ICD-9-CM partitioning of conditions presents problems for a modern system of diagnosis of sleep disorders for it unintentionally requires the combination of specificity of condition and nonorganic origin (307.4), on the one hand, and of nonspecificity of condition and organic origin (780.5), on the other (see p. 707 of volume 1). This unfortunate dichotomy appears to leave no room for a specific *as well as* organic sleep disorder, of which there are at least several. In line with this incongruity, ICD-9-CM and ICD-9 treat entities such as sleepwalking and night (i.e., sleep) terrors—now believed to derive from organic pathophysiology—as nonorganic by listing them in 307.4. Accordingly, in the interest of developing an appropriate, corresponding ICD-9-CM code for every ASDC-APSS sleep disorders syndrome, the ASDC committee was forced to overlook the placement criteria of organicity and specificity in several instances; that is, a somewhat flexible approach to assigning ICD-9-CM designations was assumed for the purpose of establishing an ICD-9-CM-compatible entry for the sleep conditions in the ASDC-APSS nosology. The committee was guided by the standard that unless clearly organic, a condition is regarded as nonorganic. In other words, with few exceptions, conditions that are, or even may be, functional or psychogenic are put in 307.4 (where, also, more categories are available), whereas conditions that are clearly organic are incorporated in 780.5. In several cases, in which a syndrome may have either a functional or organic origin, functional (307.4) and organic (780.5) codes are both provided.

As discussed in the introduction to this nosology, an attempt was made in the ASDC-APSS diagnostic system to avoid the categorization of conditions according to etiology, which for many disorders is altogether unknown or unclear. However, as indicated, such decisions were required in the exercise of assigning compatible ICD-9-CM codes. Nevertheless, the ASDC committee is of the opinion that establishing ICD-9-CM entry codes for all conditions outweighs in importance the questions that such categorization may raise referable to etiology. The codes, in the view of the committee, do not carry an implication for etiology.

Additional ICD-9-CM designations are suggested in this section, as the footnote statement points out, wherever the possibility of double coding may be desirable. This is primarily the case when a sleep condition requiring diagnosis is part of the clinical picture caused by an underlying condition. Accordingly, the tables of comparative codes list the sleep condition entries, whereas the footnotes provide a range of non-sleep-related ICD-9-CM codes for the primary disorders (see footnote 7, for example). Note that the names of some of the ASDC-APSS diagnostic conditions have been altered or shortened to accommodate the tables.

TABLE OF COMPARATIVE CODES—I[a]

	ASDC-APSS Diagnostic codes and conditions	Recommended compatible ICD-9-CM codes[b]
	A. DIMS: Disorders of Initiating and Maintaining Sleep (Insomnias)	
A.1.a	Transient and situational psychophysiological DIMS	307.41-0
A.1.b	Persistent psychophysiological DIMS	307.42-0
A.2.a	DIMS with symptom and personality disorders	307.42-1[1]
A.2.b	DIMS with affective disorders	307.42-2[2]
A.2.c	DIMS with other functional psychoses	307.42-3[3]
A.3.a	DIMS with tolerance to or withdrawal from CNS depressants	780.52-0[4]
A.3.b	DIMS with sustained use of CNS stimulants	780.52-1[5]
A.3.c	DIMS with sustained use of or withdrawal from other drugs	780.52-2[6]
A.3.d	DIMS with chronic alcoholism	780.52-3[7]
A.4.a	Sleep apnea DIMS syndrome	780.51-0
A.4.b	Alveolar hypoventilation DIMS syndrome	780.51-1
A.5.a	Sleep-related (nocturnal) myoclonus DIMS syndrome	780.52-4[8]
A.5.b	"Restless legs" DIMS syndrome	780.52-5[9]
A.6	DIMS with other medical, toxic, and environmental conditions	780.52-6[10]
A.7	Childhood–onset DIMS	780.52-7
A.8.a	Repeated REM sleep interruptions	307.48-0
A.8.b	Atypical polysomnographic features	307.48-1
A.8.c	Not otherwise specified DIMS disorder	307.42-9 or 780.52-9[11]
A.9.a	Short sleeper	307.49-0
A.9.b	Subjective DIMS complaint without objective findings	307.49-1
A.9.c	Not otherwise specified non-DIMS condition	307.40-1
	B. DOES: Disorders of Excessive Somnolence	
B.1.a	Transient and situational psychophysiological DOES	307.43-0
B.1.b	Persistent psychophysiological DOES	307.44-0
B.2.a	DOES with affective disorders	307.44-1[12]
B.2.b	DOES with other functional psychiatric disorders	307.44-2[1,2]
B.3.a	DOES with tolerance to or withdrawal from CNS stimulants	780.54-0[13]

B.3.b	DOES with sustained use of CNS depressants	780.54-1[14]
B.4.a	Sleep apnea DOES syndrome	780.53-0
B.4.b	Alveolar hypoventilation DOES syndrome	780.53-1
B.5.a	Sleep-related (nocturnal) myoclonus DOES syndrome	780.54-4[15]
B.5.b	"Restless legs" DOES syndrome	780.54-5[16]
B.6	Narcolepsy	347
B.7	Idiopathic CNS hypersomnolence	780.54-7
B.8	DOES with other medical, toxic, and environmental conditions	780.54-6[17]
B.9.a.i	DOES (periodic) with Kleine-Levin syndrome	780.54-2[18]
B.9.a.ii	DOES (periodic) with menstrual-associated syndrome	780.54-3[19]
B.9.b	Insufficient sleep	307.49-4
B.9.c	Sleep drunkenness	307.47-1
B.9.d	Not otherwise specified DOES disorder	307.449 or 780.54-9[20]
B.10.a	Long sleeper	307.49-2
B.10.b	Subjective DOES complaint without objective findings	307.49-3
B.10.c	Not otherwise specified non-DOES condition	307.40-2

C. Disorders of the Sleep–Wake Schedule

C.1.a	Rapid time zone change ("jet lag") syndrome	307.45-0
C.1.b	"Work shift" change in conventional sleep–wake schedule	307.45-1
C.2.a	Frequently changing sleep–wake schedule	307.45-2
C.2.b	Delayed sleep phase syndrome	780.55-0
C.2.c	Advanced sleep phase syndrome	780.55-1
C.2.d	Non-24-hour sleep–wake schedule	780.55-2
C.2.e	Irregular sleep–wake pattern	307.45-3
C.2.f	Not otherwise specified sleep–wake schedule disturbance	307.45-9 or 780.55-9[21]

D. Dysfunctions Associated with Sleep, Sleep Stages, or Partial Arousals (Parasomnias)

D.1	Sleepwalking (somnambulism)	307.46-0
D.2	Sleep terror (pavor nocturnus, incubus)	307.46-1
D.3	Sleep-related enuresis	307.46-2 or 780.56-0[22]
D.4.a	Dream anxiety attacks (nightmares)	307.47-0
D.4.b	Sleep-related epileptic seizures	780.56-1[23]
D.4.c	Sleep-related bruxism	306.8
D.4.d	Sleep-related headbanging (jactatio capitis nocturnus)	307.3
D.4.e	Familial sleep paralysis	780.56-2
D.4.f	Impaired sleep-related penile tumescence	780.56-3[24]
D.4.g	Sleep-related painful erections	780.56-4[25]
D.4.h	Sleep-related cluster headaches and chronic paroxysmal hemicrania	780.56-5[26]
D.4.i	Sleep-related abnormal swallowing syndrome	780.56-6
D.4.j	Sleep-related asthma	780.56-7[27]
D.4.k	Sleep-related cardiovascular symptoms	780.56-8[28]
D.4.l	Sleep-related gastroesophageal reflux	780.56-9[29]
D.4.m	Sleep-related hemolysis (paroxysmal nocturnal hemoglobinuria)	283.2
D.4.n	Asymptomatic polysomnographic finding	780.59
D.4.o	Not otherwise specified parasomnia	307.47-9 or 780.56[30]

[a] For explanation of superscripts to the ICD-9-CM code numbers, see footnote to Table II.

[b] Numbers following hyphens are not part of the authorized ICD-9-CM codes.

TABLE OF COMPARATIVE CODES—II[a]

Recommended compatible ICD-9-CM codes[b]	ASDC-APSS Diagnostic codes and conditions	
283.2	D.4.m	Sleep-related hemolysis (paroxysmal nocturnal hemoglobinuria)
306.8	D.4.c	Sleep-related bruxism
307.3	D.4.d	Sleep-related headbanging (jactatio capitis nocturnus)
307.41-0	A.1.a	Transient and situational psychophysiological DIMS
307.42-0	A.1.b	Persistent psychophysiological DIMS
307.42-1[1]	A.2.a	DIMS with symptom and personality disorders
307.42-2[2]	A.2.b	DIMS with affective disorders
307.42-3[3]	A.2.c	DIMS with other functional psychoses
307.42-9[11]	A.8.c	Not otherwise specified DIMS disorder (also 780.52-9)
307.43-0	B.1.a	Transient and situational psychophysiological DOES
307.44-0	B.1.b	Persistent psychophysiological DOES
307.44-1[12]	B.2.a	DOES with affective disorders
307.44-2[13]	B.2.b	DOES with other functional psychiatric disorders
307.44-9[20]	B.9.d	Not otherwise specified DOES disorder (also 780.54-9)
307.45-0	C.1.a	Rapid time zone change ("jet lag") syndrome
307.45-1	C.1.b	"Work shift" change in conventional sleep–wake schedule
307.45-2	C.2.a	Frequently changing sleep–wake schedule
307.45-3	C.2.e	Irregular sleep–wake pattern
307.45-9[21]	C.2.f	Not otherwise specified sleep–wake schedule disturbance (also 780.55-9)
307.46-0	D.1	Sleepwalking (somnambulism)
307.46-1	D.2	Sleep terror (pavor nocturnus, incubus)
307.46-2[22]	D.3	Sleep-related enuresis (also 780.56-0)
307.47-0	D.4.a	Dream anxiety attacks (nightmares)
307.47-1	B.9.c	Sleep drunkenness
307.47-9[30]	D.4.o	Not otherwise specified parasomnia (also 780.56)
307.48-0	A.8.a	Repeated REM sleep interruptions
307.48-1	A.8.b	Atypical polysomnographic features
307.49-0	A.9.a	Short sleeper
307.49-1	A.9.b	Subjective DIMS complaint without objective findings
307.49-2	B.10.a	Long sleeper
307.49-3	B.10.b	Subjective DOES complaint without objective findings
307.49-4	B.9.b	Insufficient sleep
307.40-1	A.9.c	Not otherwise specified non-DIMS condition
307.40-2	B.10.c	Not otherwise specified non-DOES condition
347	B.6	Narcolepsy
780.51-0	A.4.a	Sleep apnea DIMS syndrome
780.51-1	A.4.b	Alveolar hypoventilation DIMS syndrome
780.52-0[4]	A.3.a	DIMS with tolerance to or withdrawal from CNS depressants
780.52-1[5]	A.3.b	DIMS with sustained use of CNS stimulants
780.52-2[6]	A.3.c	DIMS with sustained use or withdrawal from other drugs
780.52-3[7]	A.3.d	DIMS with chronic alcoholism
780.52-4[8]	A.5.a	Sleep-related (nocturnal) myoclonus DIMS syndrome
780.52-5[9]	A.5.b	"Restless legs" DIMS syndrome
780.52-6[10]	A.6	DIMS with other medical, toxic, and environmental conditions
780.52-7	A.7	Childhood–onset DIMS
780.52-9[11]	A.8.c	Not otherwise specified DIMS disorder (also 307.42-9)

780.53-0	B.4.a	Sleep apnea DOES syndrome
780.53-1	B.4.b	Alveolar hypoventilation DOES syndrome
780.54-0[13]	B.3.a	DOES with tolerance to or withdrawal from CNS stimulants
780.54-1[14]	B.3.b	DOES with sustained use of CNS depressants
780.54-2[18]	B.9.a.i	DOES (periodic) with Kleine-Levin syndrome
780.54-3[19]	B.9.a.ii	DOES (periodic) with menstrual-associated syndrome
780.54-4[15]	B.5.a	Sleep-related (nocturnal) myoclonus DOES syndrome
780.54-5[16]	B.5.b	"Restless legs" DOES syndrome
780.54-6[17]	B.8	DOES with other medical, toxic, and environmental conditions
780.54-7	B.7	Idiopathic CNS hypersomnolence
780.54-9[20]	B.9.d	Not otherwise specified DOES disorder (also 307.44-9)
780.55-0	C.2.b	Delayed sleep phase syndrome
780.55-1	C.2.c	Advanced sleep phase syndrome
780.55-2	C.2.d	Non-24-hour sleep–wake schedule
780.55-9[21]	C.2.f	Not otherwise specified sleep–wake schedule disturbance (also 307.45-9)
780.56[30]	D.4.o	Not otherwise specified parasomnia (also 307.47-9)
780.56-0[22]	D.3	Sleep-related enuresis (also 307.46-2)
780.56-1[23]	D.4.b	Sleep-related epileptic seizures
780.56-2	D.4.e	Familial sleep paralysis
780.56-3[24]	D.4.f	Impaired sleep-related penile tumescence
780.56-4[25]	D.4.g	Sleep-related painful erections
780.56-5[26]	D.4.h	Sleep-related cluster headaches and chronic paroxysmal hemicrania
780.56-6	D.4.i	Sleep-related abnormal swallowing syndrome
780.56-7[27]	D.4.j	Sleep-related asthma
780.56-8[28]	D.4.k	Sleep-related cardiovascular symptoms
780.56-9[29]	D.4.l	Sleep-related gastroesophageal reflux
780.59	D.4.n	Asymptomatic polysomnographic finding

[a] Codes for non-sleep-related or more specific disorders may be required or desired in certain cases in addition to the *ICD-9-CM* sleep-related disorder codes listed in the tables of comparative codes above. The diagnostician should consult *ICD-9-CM* for supplementary and alternative codes to fit the variety of clinical manifestations of the disorders. Suggestions concerning such additional code assignments, where indicated, are provided in the footnotes below.

[b] Numbers following hyphens are not part of the authorized ICD-9-CM codes.

1. Use additional code to identify:
   anxiety (300.00-300.09)
   personality disorder (301.0-301.9)
   symptom disorders (306.0-306.9, 307.0-307.3, 307.5-307.9, 316)
2. Use additional code to identify:
   major affective disorder (296.0-296.9, 296.82, 298.0)
   minor affective disorder (300.4, 301.10-301.13, 311)
3. Use additional code to identify:
   schizophrenia (295.0-295.9)
   other functional psychosis (297.0-298.9)
4. Use additional code to identify barbiturate, sedative, or hypnotic:
   dependence (304.1)
   withdrawal (292.0)
5. Use additional code to identify:
   amphetamine or other psychostimulant abuse (305.7)

6. Use additional code to identify:
   drug withdrawal (292.0)
   tranquilizer dependence (304.1)
   unspecified drug dependence (304.9)
7. Use additional code to identify:
   alcohol amnestic syndrome (291.1)
   alcohol dementia (291.2)
   chronic alcoholism (303.9)
8. Use additional code to identify:
   abnormal involuntary movements (781.0)
9. Use additional code to identify:
   restless legs (333.99)
10. Code also underlying condition, as:
    Cushing's syndrome (255.0)
    Use additional code for environmental condition, as:
    exposure to noise (E928.1)
11. This category is set aside for DIMS that are as yet undesignated and undescribed; use codes 307.42-9 if the disorder is functional and 780.52-9 if it has an organic etiology.
12. Use additional code to identify:
    major depression (296.2-296.3, 296.5, 296.82, 298.0)
    minor depression (300.4, 301.11-301.12, 311)
13. Use additional code to identify:
    amphetamine and other psychostimulant dependence (304.4)
14. Use additional code to identify:
    barbiturate and similarly acting sedative or hypnotic abuse (305.4)
15. See footnote 8.
16. See footnote 9.
17. See footnote 10.
18. Use additional code to identify:
    Kleine-Levin syndrome (349.89)
19. Use additional code to identify:
    menstrual-associated syndrome (625.4, 626.9)
20. This category is set aside for DOES (as is 11 for DIMS) that are as yet undesignated and undescribed; use codes 307.44-9 if the disorder is functional, and 780.54-9 if it has an organic etiology.
21. As in footnotes 11 and 20 but for sleep-wake schedule disturbance; use codes 307.45-9 for functional and 780.55-9 for organic conditions.
22. For sleep-related enuresis, code as follows:
    psychogenic (307.46 and 307.6)
    organic (780.56 and 788.3)
23. Use additional code to identify type of epilepsy:
    automatism (345.4)
    generalized convulsive (345.1)
    partial, with impairment of consciousness (345.4)
    temporal lobe type (345.4)
    unspecified (345.9)
24. Use additional code for impotence:
    psychogenic (302.72)
    organic (607.84)
25. Use additional code to identify:
    persistent painful erection (priapism) (607.3)
26. Use additional code to identify:
    classical migraine (346.1)
    cluster headache (346.2)
    common (atypical) migraine (346.0)
    migraine variants (346.2)
    other specified forms of migraine (346.8)
27. Use additional code to identify asthma:
    extrinsic (493.0)
    intrinsic (493.1)

28. Use additional code to identify cardiovascular symptoms:
    cardiac dysrhythmias (427.0-427.9)
    nocturnal angina (413.0)
    orthopnea (786.02)
    paroxysmal nocturnal dyspnea (786.09)
    pulmonary edema (with heart failure) (428.1)
29. Use additional code to identify:
    gastroesophageal reflux (530.1)
30. See footnotes 11, 20, and 21. For parasomnias, use sleep disorder code 307.47-9 for functional origin, or code 780.56 for organic-related conditions.