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# The Use of Polysomnography in the Evaluation of Insomnia

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## 1.0 INTRODUCTION

Polysomnography (PSG) permits a quantified assessment of nocturnal sleep. It was originally hoped that PSG would be a valuable tool in facilitating our assessment and treatment of patients with insomnia complaints. This review considers the medical literature of the past 25 years in which PSG has been used in evaluating insomnia complaints. It addresses the issues of whether PSG is a useful, valuable and cost-effective strategy for the evaluation and treatment planning of patients with insomnia complaints.

## 2.0 METHODOLOGY

This review was commissioned by the Standards of Practice Committee of the American Sleep Disorders Association. The review was developed in the following manner: 1) A MEDLINE search on relevant topics was conducted for the period 1966–1994, as well as additional literature reviews as appropriate; 2) published peer-reviewed literature was emphasized in the review; 3) outside experts were consulted concerning thoroughness and comprehensiveness of the literature reviewed, as well as its accurate interpretation; and 4) the final document was agreed to by all authors and reflects the opinion of all authors.

## 3.0 HISTORICAL BACKGROUND

### 3.1 The insomnia complaint

Transient insomnias in response to illness, stress, schedule changes and long-distance travel are ubiquitous. Long-lasting or chronic insomnia (e.g. $\geq 3$ weeks) is a significant public health problem affecting a substantial percentage of the population. In a 1991 Gallup survey, 36% of respondents reported insomnia complaints, with 9% reporting that it was a chronic problem (1). Additionally, Campbell and colleagues indicated that half of the population over age 65 suffer from chronic sleep disturbances (2).

In the six-volume compendium of *Osler's Modern Medicine* published in 1925 (3), the mention of insomnia is limited to a symptom accompanying other disorders. Prominent causes were infectious diseases such as influenza, tuberculosis and encephalitis lethargica. Insomnia also occasionally accompanied neurasthenia (in distinction to hysteria, where sleep was reported as good) and was a frequent symptom of general paresis, delirium tremens and manic depressive psychosis. Disorders of sleep per se, however, were not isolated as medical entities.

Over 30 years later, in the third edition of Harrison's *Principles of Internal Medicine,* Raymond Adams wrote a chapter entitled "Sleep and its abnormalities" (4). Insomnia was again discussed as a symptom, with the most common etiologies being the presence of pain or discomfort, or the presence of anxiety or other "nervous" (i.e. psychiatric) disorders. Adams cautioned that, with respect to insomnia, "whatever the cause may be, the physician should always be on his guard when listening to reports of the amount of sleep lost by sufferers of insomnia because they are usually exaggerated" (p. 319). The existence of possible primary sleep disorders manifesting themselves as insomnia was not considered.

The most recent *International Classification of Sleep Disorders* (ICSD) (5) lists several dozen types of insomnia complaints, in many of which the sleep-disorder is thought to be primary. Although polysomnographic findings are mentioned, polysomnographic criteria pathognomonic for specific disorders are not detailed.

There has therefore been a progression from disturbed sleep representing solely a symptom of other

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disorders, to a contemporary viewpoint that there are primary sleep disorders whose symptoms include, or may consist primarily of, insomnia. This change in viewpoint has resulted in large part from the increasing attention given to sleep by the scientific community following the development of PSG as a method for measuring and quantifying sleep, and which, perhaps more importantly, encouraged clinicians to listen more closely to their patients' sleep complaints.

### 3.2 Development of PSG as a measure for sleep assessment

As in many areas, knowledge about sleep emerged with the development of technologies that permit its measurement and quantification. Shortly after development of the electroencephalogram (EEG), Loomis and colleagues found that in humans brain electrical activity during sleep differed from that during wakefulness (6,7). The discovery of rapid eye movement (REM) or paradoxical sleep (8) and its associated mental content (9) heightened interest in objective studies of sleep and insomnia. Recordings of additional physiological variables, including measures of respiration and leg movements (10,11), shortly followed.

One of the earliest polysomnographic studies of insomniacs was performed by Monroe (12), who found that poor sleepers had longer sleep latencies, less total sleep and more awakenings than good sleepers, but also that they tended to overestimate the severity of their sleep complaint. Poor sleepers also had less REM sleep and marginal evidence of higher levels of physiological arousal. These early findings were promising in terms of suggesting PSG recordings may be helpful in the assessment of sleep complaints.

Two major questions remain unanswered at the present time, however: 1) How valuable is PSG in the differential diagnosis and treatment planning of insomnia complaints?, and 2) In which patients might PSG prove to be cost effective? This review attempts to provide a summary of current information addressing these questions.

### 3.3 What polysomnography measures—and what it (usually) does not measure

Conventional PSG is most often limited to obtaining the minimal data required for assessing sleep state by conventional criteria (13), and to recording some autonomic or skeletal muscle variables related to specific sleep disorders or complaints, such as measures of breathing, cardiac rate and rhythm, and leg movement activity.

There are certain limitations associated with conventional recording and scoring. A single page of polygraphic data constituting 20 seconds of data at a speed of 15 mm/second, or 30 seconds of data at a speed of 10 mm/second, is usually scored as representing only one stage, which is that stage that represents >50% of the epoch. Thus stage information contained in up to 49% of the epoch may not be assessed, possibly providing a skewed view of the record as a whole. Sleep scoring based upon epochs of 20–30 seconds has contributed to lack of uniformity in the manner in which awakenings or arousals from sleep have been scored and quantified. A proposed scoring system for arousals has been recently published (14), but has not yet been routinely incorporated into most clinical polysomnographic recordings.

Basing nonrapid eye movement (NREM) slow-wave sleep stages 3 and 4 on both frequency and absolute amplitude criteria has meant that slow activity not reaching formal amplitude criteria of 75 $\mu$V is disregarded, even though it may be present in substantial amounts at amplitudes nearly meeting criteria. PSG reports with no stage 3 or stage 4 sleep represented, for example, may suggest that no slow-wave sleep was present, yet substantial amounts of 1–4-Hz activity not meeting formal amplitude criteria may be present. This may have significant implications for the elderly, in whom stages 3 and 4 are often reported to be decreased.

Limiting EEG recording to central leads means that topographic information is lacking, and events with a limited topographic representation are not recorded at all. Recently published guidelines for PSG recordings by the American EEG Society recommend six or more channels of EEG be recorded (including bilateral frontal, central, occipital and temporal leads), although this recommendation is based primarily upon looking for evidence of seizure activity or asymmetry, rather than for information on topographic distribution of normal sleep activity (15).

These and other concerns have been outlined by Kubicki et al. (16) and address limitations that impede progress in areas that may be important to insomnia research. Until the research is done, however, these concerns remain speculative. Rechtschaffen (17) has recently pointed out that procedures for scoring by visual examination are unwieldy and time consuming, and should be able to be replaced by some automated system that could accurately approximate epoch-based visual scoring. Not clear, however, is the value of efforts currently in place to make computerized sleep scoring result in correct Rechtschaffen and Kales scores, because the value of these scores in better understanding insomnia complaints is unclear. Recognizing the problems inherent in the original system, Rechtschaffen also cautions, "In the absence of a powerful rationale for a blanket emphasis on wavelength or am-

plitude, we might as well retain the current criteria" (17, p. 27).

Another concern is that sleep is most often quantified and defined by use of EEG measures alone. As a result, important physiological information that contributes to our understanding of sleep is ignored, including heart rate, heart rate variability and other measures of physiology, such as body temperature and other biological rhythms, electromyographic activity, autonomic nervous system physiology, and phasic REM activity (18–20).

In spite of these limitations, standard scoring criteria and a few limited additional measures of physiology remain the basis of conventional PSG as is usually performed in ASDA-accredited sleep disorders centers.

## 4.0 POLYSOMNOGRAPHIC FINDINGS IN CHRONIC INSOMNIA

### 4.1 Patients with intrinsic and unspecified chronic insomnias

Many early polysomnographic studies, and several more recent ones as well, made no attempt to discriminate an etiologic diagnosis in patients complaining of chronic insomnia. In many of these, selection criteria included only a complaint of chronic insomnia (sleep onset or sleep continuity difficulties) without specific exclusion criteria for other primary sleep disorders, psychiatric disorders or occult medical and neurological conditions (12,21–29). Other studies have further specified inclusion criteria for polysomnographic studies of patients with insomnia. In most of these, the patients could be characterized as having idiopathic or psychophysiological insomnia, or sleep state misperception (in ICSD nosology) (30–38).

Polysomnographic studies of either unselected insomnia patients or selected patients with intrinsic forms of insomnia have generally demonstrated three types of findings: 1) altered sleep/wake ratios, 2) normal sleep stage architecture and 3) altered patterns of night-to-night variability. Altered sleep/wake ratios can take the form of prolonged sleep latencies, decreased total sleep time and sleep continuity disturbances, indicated by an increased number and/or duration of awakenings. The magnitude of insomniac–control differences is not always striking, and several investigators have noted that many self-identified insomniacs do not demonstrate objective sleep abnormalities (35,39). In part, this may reflect greater discrepancies between subjective and objective estimates of sleep among insomniac patients compared to controls (e.g. 29,31,36,40). Possible individual differences in how long consciousness must be interrupted to be perceived as interrupted (e.g. by sleep) may also play a role (41). Whether some individuals may continue with subjective awareness of conscious mentation during NREM sleep stages is not clear.

In contrast to sleep continuity measures, most reports of sleep stage architecture have not revealed clear differences between patients with chronic insomnia and control groups, although a few studies have found decreased amounts of REM sleep (32,34).

Greater night-to-night variability usually characterizes patients with chronic intrinsic or unselected insomnias. Some, but not all, patients with persistent psychophysiological insomnia have a "reverse first night effect", that is, better sleep on the initial night of studies than on subsequent nights (12,22,23,42).

Prolonged sleep latency or frequent awakenings in self-identified insomnia patients often confirm patients' reports without providing additional information. Severe discrepancies between subjective and objective data may suggest sleep state misperception (43). However, such findings do not in themselves justify the expense of PSG. More critical questions are: 1) How often do patients with chronic insomnia have diagnoses that can be established only with conventional PSG (e.g. sleep apnea, periodic limb movements); 2) How often do patients with chronic insomnia present "unexpected" findings at PSG?; and 3) Can PSG differentiate subtypes of chronic insomnia?

#### 4.1.1 *How often do patients with chronic insomnia have diagnoses that can be established only with polysomnography (e.g. sleep apnea, periodic limb movements)?*

The prevalence of insomnia-dependent diagnoses shows considerable variability among studies. Kales and colleagues (25) reported that none of their 200 consecutive insomnia patients demonstrated clinically significant sleep apnea (defined as >30 events per night) and that only 21/200 patients (10.5%) demonstrated any sleep apnea events. The same study reported that 10/200 insomnia patients (5%) had significant nocturnal myoclonus, which was not different from the 6% figure found in healthy controls. The definition of nocturnal myoclonus in this study required at least three episodes of >30 isolated leg movements (each lasting 0.5–5.0 seconds, occurring with a periodicity of 5–120 seconds), which constitutes a stringent definition. By contrast, Zorick et al. (44) evaluated 84 insomnia patients and diagnosed six (7%) with sleep apnea, nine (11%) with nocturnal myoclonus, nine (11%) with restless legs syndrome and 16 (19%) with subjective insomnia (i.e. sleep state misperception). The exact procedures for scoring nocturnal myoclonus were not specified. Coleman et al. (45) found that 13% of 409

sleep disorder patients had periodic (leg) movements in sleep (PLMS), but that they occurred in a wide variety of sleep disorders and were not limited to patients with insomnia complaints. More recently, Edinger et al. (46) assessed 100 patients with chronic insomnia and diagnosed sleep apnea in 3%, periodic movements of sleep (PMS)/restless legs syndrome in 25% and subjective insomnia in 6%. PMS was scored according to the method of Coleman et al. (47), with a lenient criterion level of five movements per hour of sleep associated with arousals; the mean was 23.3 movements with arousal per hour of sleep. Apnea scoring was not specified, but the three patients diagnosed with sleep apnea had an average of 22.2 apneas per hour. Thus, 34% of the sample received a diagnosis that depended heavily on polysomnographic results. The investigators also noted a strong age relationship in their findings; 15% of insomnia patients <40 years of age had a final diagnosis of apnea, PMS or subjective insomnia, compared to 46% of patients >40 years of age. In a study using ambulatory PSG, McCall et al. (48) found that 15 of 24 older men (60–72 years of age) with no sleep complaints would have met ICSD (5) criteria for a "mild" periodic limb movement disorder (PLMD), and two of the 24 would have met criteria for a "severe" disorder, if they had had a sleep complaint. Thus PLMS may be a common finding in asymptomatic older men, although PLMS with arousals are less common.

Coleman et al.'s national cooperative study (47) included 1,214 patients with a primary complaint of insomnia. Sleep apnea was diagnosed in 75 patients (6.2%), sleep-related myoclonus and restless legs syndrome occurred in 148 patients (12.2%) and no insomnia abnormality was found in 112 (9.2%). Clearly, individual sites participating in the cooperative study had different referral biases; some focused on evaluation of sleep apnea and others on psychiatric disorders. Furthermore, only 80% of referred patients had PSG, and Coleman et al. reported only on those who had undergone the procedure. As a result, the "polysomnography-dependent diagnoses" were probably overrepresented.

Studies addressing the prevalence of polysomnography-dependent diagnoses probably differ in part because of the differing scoring conventions. Although Kales et al. (25) and Edinger et al. (46) report consecutive patients, Zorick et al. (44) and Coleman et al. (47) do not specify this feature and may have estimates biased upward as a result. Comparison groups of noncomplaining control subjects were included in the Kales et al. (25) study, but not in others. Finally, the studies do not consistently address an even more pressing question: How often were the final diagnoses suspected on clinical grounds alone?

The finding of sleep-related breathing disturbances must also take into account how often such findings might occur in normal individuals. Kripke et al. found middle-aged adults have a median value of five respiratory events per hour (49). This of course raises the question of what population norms do we compare such findings to; such norms are sparse indeed.

#### 4.1.2 *How often do patients with chronic insomnia present "unexpected" findings at polysomnography?*

Jacobs et al. (50) reported the results of PSG in 123 patients referred for evaluation of chronic insomnia. Final diagnoses according to the Association of Sleep Disorders Centers nosology included difficulties initiating and maintaining sleep (DIMS) associated with affective disorder (29%), persistent psychophysiological DIMS (19%), DIMS associated with periodic limb movements (12%) and DIMS associated with other medical conditions (11%). More importantly, the authors reported that initial clinical diagnoses in 49% of patients "required substantial modification based on laboratory findings". In 41% of the 123 patients, PSG provided additional and unsuspected evidence; in 20%, the studies failed to confirm an initial clinical diagnosis; and in 11%, PSG both added new information and failed to support initial clinical diagnoses. The major point of contention with such results relates to the definition of what constitutes "significant" polysomnographic information. Edinger and colleagues (46) also addressed the question of prior expectations versus polysomnographic results. They reported that only 14/25 cases of periodic limb movements/restless leg syndrome were identified clinically, and nine additional cases suspected clinically were not confirmed. For apnea, 1/3 final diagnoses was clinically suspected, and seven additional clinical diagnoses were ruled out by PSG. Each of the aforementioned articles, however, were followed by letters to the editor that raised additional criticisms. Additionally, such studies may be intrinsically biased by virtue of studying subjects referred for sleep evaluations.

#### 4.1.3 *Do different types of insomnia patients have different EEG sleep findings?*

In one of the earliest studies to address this question, Gillin and colleagues (32) found that a discriminant function analysis of EEG sleep measures (using total sleep time, total recording period, sleep latency, sleep efficiency, awake time, early morning awake time, REM time and REM %) correctly discriminated 82% of patients with "chronic insomnia" or depression, and healthy controls. Hauri and Olmstead (51) and Hauri (38) reported EEG sleep differences between healthy controls and patients with "childhood-onset" insom-

nia, adult-onset "psychophysiological insomnia" and "subjective insomnia". The latter report used the multivariate statistical technique of cluster analysis and identified eight different clusters. This clearly suggests that different types of EEG sleep patterns can be identified in "chronic insomniacs". A high frequency of psychiatric symptoms and disorders has often been noted in studies of patients with insomnia (50,52). In this context, distinctions in EEG sleep patterns between patients with psychophysiological insomnia, generalized anxiety disorder and major depression are noteworthy (37,38). Zorick and colleagues (44) also noted EEG sleep differences between chronic insomnia patients with different diagnoses (including psychiatric disorders, respiratory impairment, periodic limb movements and atypical polysomnographic features).

Therefore, published evidence suggests that patients with different types of chronic insomnia might be distinguishable on the basis of EEG sleep findings. However, closer consideration of these findings reveals further questions and problems. For instance, several studies used multivariate statistical techniques to distinguish patient groups (38,53,54). Such techniques cannot be generalized readily to other laboratories and other populations. On the other hand, most studies report group differences, but do not indicate any criterion values for evaluating the sensitivity and specificity of particular parameters. One reason is that group differences tend to be small in magnitude and inconsistent from one study to the next. Furthermore, most studies have defined different groups on clinical grounds, and even if group differences can be demonstrated polysomnographically, it is not clear whether this information helps in elucidating pathophysiology or treatment course.

Hudson et al. (53) recently reported a meta-analysis of polysomnographic findings in depression, primary insomnia and narcolepsy. A summary measure constructed from five major variables (wake after sleep onset, REM latency, REM density, percent stage 3 + 4 sleep and percent stage 1 sleep) was used in estimating degree of sleep disturbance. On both individual measures and the summary index, sleep disturbance demonstrated a progression of disturbance from insomnia through depression to narcolepsy, but did not otherwise distinguish the groups. These findings suggested present polysomnographic measures may not be sufficiently robust to separate these diagnostic entities and, in turn, their ability to accurately define subtypes of insomnia is called into question.

### 4.2 Patients with psychiatric disorders

Many patients with psychiatric disorders present with, or complain of, insomnia. Depression especially is characterized by insomnia complaints, perhaps most characteristically early morning awakenings. Numerous studies devoted to the PSG characteristics of patients with major depression have been reviewed elsewhere (55–57). The EEG sleep characteristics of depression are best considered as a "constellation" characterized by reduced REM latency, increased phasic REM activity, altered temporal distribution of REM sleep and reduced amounts of slow-wave sleep with altered temporal distribution. In terms of differential diagnosis, most attention has focused on the sensitivity and specificity of REM latency as a "marker" of depression. Clearly, the sensitivity and specificity of EEG sleep measures as "markers" of depression vary according to which test is used, what criterion value is chosen, whether univariate or multivariate techniques are used and, most importantly, what reference group is studied (56). Benca et al.'s (55) recent meta-analysis showed that reduced REM latency characterizes affective disorders and that REM latency is shorter for affective disorders than for other conditions, but that it does not always distinguish affective disorders from other psychiatric or sleep disorders in a qualitative sense. The meta-analysis also shows that affective disorders are characterized by an increased amount of REM sleep, increased phasic REM activity, and decreased slow-wave sleep. Unfortunately, in direct comparisons, these features do not consistently distinguish affective disorders from other psychiatric disorders. Several studies (55,58) have reviewed the EEG sleep findings of other psychiatric disorders, including anxiety disorders, borderline personality, eating disorders and schizophrenia. Sleep onset difficulties and disrupted sleep continuity characterize each of these (with the exception of eating disorders). Reduced slow-wave sleep and reduced REM latency have characterized some studies of patients with schizophrenia, and reduced REM latency has also been found in borderline personality. None of these findings can be considered "diagnostic" or specific to the individual disorders, however; thus PSG findings may be useful to clinicians evaluating patients with such psychiatric disorders, but they are not sufficiently specific to merit PSG as a primary diagnostic instrument.

### 4.3 Patients with substance-related insomnias

Patients taking prescribed medications or drugs of abuse can have a variety of sleep complaints, including insomnia (for reviews, see references 59–61). Many medications have relatively consistent effects on sleep. Examples include the increase in frequency and duration of sleep spindles with benzodiazepines and barbiturate sedative-hypnotics; the suppression and subsequent rebound of REM seen with alcohol; and the

appearance of both rapid and slower eye movements during NREM sleep caused by clomipramine, fluoxetine and other antidepressant drugs. Many other drug effects, such as increased wakefulness and REM sleep suppression, are nonspecific.

Medications can affect sleep differently during periods of acute ingestion, chronic use and withdrawal. For instance, barbiturates acutely increase sleep and decrease awakenings, but with continued use tolerance develops and wakefulness returns during sleep. During abrupt withdrawal, sleep is more dramatically disturbed, and there is a large increase in wakefulness and REM sleep rebound. In addition to differences according to phase of use, many drugs also show differential effects due to dose and timing of administration. Each of these factors makes it difficult to use PSG clinically to "diagnose" insomnia related to substance abuse. Finally, there is a question of persistent effects from substance abuse. For instance, Adamson and Burdick (62) suggested possible impairment of slow-wave sleep in alcoholics who had been abstinent for up to 1–2 years.

Although medications and drugs clearly affect polysomnographic sleep measures, it is less clear whether the specific findings PSG might provide have significant treatment or prognostic implications. A recent study by Gillin et al., however, addresses this issue, suggesting REM sleep measures obtained on admission to an alcohol treatment program predict relapse in chronic alcoholics (63). In this study, three REM sleep variables (short REM latency, increased REM % and increased REM density) correctly predicted relapse at 3 months in 80% of a group of 45 patients with primary alcoholism.

Overall, an accurate clinical history and urine drug screen are clearly less expensive, and arguably more specific, indicators of substance-related insomnia problems. Furthermore, it may be difficult to distinguish effects of a substance from preexisting characteristics of the individual. For example, the persistent reduction of slow-wave sleep noted in some alcoholics may reflect the individual's premorbid characteristics rather than persistent effects of alcohol. The role of PSG in evaluation of insomnia related to substance use is probably limited to cases in which it is necessary to verify or quantify sleep difficulties; cases presenting with atypical features; and cases in which there is reason to suspect another intrinsic sleep pathology, for example, sleep apnea. The possible predictive value of PSG findings in alcoholism remain to be determined.

### 4.4 Patients with medical and neurological disorders

Patients with medical and neurological disorders frequently complain of disrupted nocturnal sleep (for reviews see references 64–66). The concurrent use of medications can further complicate the evaluation of medically ill patients. The critical issue in PSG of insomnia patients with medical disorders is whether specific information can be gained, and whether such information is helpful in terms of unsuspected diagnoses and/or additional treatment recommendations. Some disorders have specific findings. For example, a variety of spontaneous movement disorders can occur during sleep and may have specific polysomnographic findings very useful in diagnosis (67). Alzheimer's type dementia is associated with a loss of REM sleep and slow-wave sleep that is proportional to the degree of cognitive impairment (68–70). In addition, patients show deterioration of 24-hour sleep–wake consolidation. Finally, patients with dementia develop "indeterminate" NREM sleep, which resembles stage 2 sleep but without the normal transients such as K complexes and sleep spindles. These features are sufficient to distinguish elderly patients with dementia from those with depression with 80% accuracy (71). Even in this case, however, the diagnosis of dementia is established clinically, and EEG sleep does not appear to be abnormal in very early cases (68). Indeed, Vitiello et al. (72) state "sleep impairment is a rather nonspecific concomitant of each dementing condition and does not easily differentiate the various causes of dementia" (p. 86).

Another finding is the "alpha-delta" ("Alpha-NREM", "alpha sleep") pattern found in patients with fibromyalgia (73). The amount of alpha can be graded on a simple ordinal scale. However, this finding is not specific, and it has been noted in other patients with complaints of nonrestorative sleep, including patients with psychiatric disorders, rheumatoid arthritis and chronic fatigue syndrome (e.g. 74–77).

A much larger number of disorders can be exacerbated by sleep or occur exclusively during sleep. Examples include certain headache disorders (e.g. cluster headaches), seizure disorders, asthma, congestive heart failure and peptic ulcer disease. In most cases of these disorders, sleep exacerbates an existing disorder that can be diagnosed during waking hours, and routine PSG does not produce specific or unsuspected findings. Some cases of nocturnal seizures may be diagnosed for the first time during sleep studies, but not without a prior clinical suspicion based on history, and not with the usual routine polysomnographic sleep EEG consisting of only one or two monopolar electrodes. There are cases of unsuspected sleep-related breathing disorders found only on PSG, especially in patients with certain neurological and cardiac disorders, and careful medical screening must be used to be certain such cases are not missed.

In summary, sleep studies often confirm the insomnia complaint of patients with medical or neurological

disorders, but they rarely lead to a new diagnosis unsuspected by clinical history or routine laboratory testing.

### 4.5 Periodic limb movements and sleep-related breathing disorders

The question with these conditions is not whether they can lead to insomnia complaints (they can), but rather how often they appear as unsuspected clinically significant findings in patients with chronic insomnia. Kales and colleagues (25) found few such cases in their chronic insomnia patients, whereas Zorick et al. (44), Coleman et al. (47) and Edinger et al. (46) found 3–25% of cases with either PLMS or apnea. In the study by Jacobs et al. (50), 4/123 patients (3.3%) had unsuspected apnea, and 10/123 (8%) had unsuspected myoclonus. Additionally, in 9/123 (7%) cases, initial impression of apnea, myoclonus or cardiac arrhythmia was not supported by polysomnographic findings. Thus, in approximately 18% of cases, PSG either indicated the unsuspected presence of, or failed to confirm, the clinical impression with regard to these conditions. Edinger et al. (46) had similar findings, with 11/100 cases showing clinically unsuspected PLMS and 2/100 showing unsuspected apnea. Conversely, in 9/23 suspected cases of PLMS, the diagnosis was not supported, and in 7/8 suspected cases of apnea, the diagnosis was not supported. Overall, 29/100 cases revealed unexpected findings with regard to PLMS and apnea. This issue of whether PLMS contribute to an insomnia complaint is debated (e.g. see reference 45). There is evidence, however, that experimentally induced sleep fragmentation leads to decreases in daytime vigilance and performance (78) and to an increase in daytime sleepiness (79) symptoms also seen with insomnia complaints. Thus it is reasonable to conclude that frequent PLMS associated with arousals may represent the etiology of some insomnia complaints. In summary, apnea and myoclonus are occasional incidental and unsuspected findings of PSG, but such appear to represent exceptions rather than the rule.

Patients with sleep-related seizure disorders, or other disruptions of sleep related to abnormal motor activity during sleep, such as a REM behavior disorder, may also have an insomnia complaint due to their disrupted sleep. Although the history may provide a strong index of suspicion in such cases, PSG is required for a definitive diagnosis (80–82).

### 4.6 Circadian rhythm sleep disorders

By definition, polysomnographic studies can only lend or detract support for these disorders; their diagnosis rests on a consistent clinical history supplemented by the use of sleep diaries. We know of no data suggesting that circadian rhythm sleep disorders are frequently diagnosed de novo based on polysomnographic results, although Thorpy and collaborators found PSG recordings useful in confirming the diagnosis of delayed sleep phase syndrome in adolescents (83). The Zorick et al. (44) study included 7/84 (8%) chronic insomnia patients with a final diagnosis of circadian rhythm sleep disorders, but these diagnoses were established from both clinical and polysomnographic data. Jacobs et al. (50) report 1/123 cases with unsuspected delayed sleep phase syndrome discovered at PSG. The Edinger et al. (46) series of 100 patients and the Kales et al. (25) study do not mention circadian rhythm sleep disorders among their patients. These findings all support the belief that circadian rhythm sleep disorders are unlikely to be diagnosed on the basis of conventional polysomnography. PSG findings could be helpful, however, in cases where a delayed sleep phase syndrome occurs in connection with another sleep disorder in which PSG findings might be helpful. Conventional PSG, which does not routinely record core body temperature, may miss some circadian rhythm disorders that present as sleep onset insomnia (see section 6.1).

## 5.0 ARE POLYSOMNOGRAPHIC FINDINGS HELPFUL?

Clearly, patients with chronic insomnia frequently demonstrate abnormal polysomnographic features when any type of abnormality is considered. But are such findings clinically helpful? In this context, we shall define "helpful" as indicating that a finding helps to elucidate pathophysiology, establish a diagnosis, understand severity or guide treatment recommendations.

### 5.1 Sleep-disordered breathing

The finding of sleep-disordered breathing (SDB) has diagnostic and treatment implications. Although no single criterion serves to define a "significant" or "abnormal" amount of apnea, frequent awakenings, oxyhemoglobin desaturations $\leq 85\%$ and apnea frequency $\geq 10$ apneas per hour of sleep should prompt consideration of treatment.

### 5.2 Periodic limb movements in sleep

Periodic limb movements in sleep or associated arousals may have direct implications for etiology, diagnosis and treatment. As with apnea, there is as yet no clear criterion to establish an "abnormal" number of PLMS in patients with chronic insomnia. In most

cases, a finding of ≥10–15 PLMS per hour of sleep associated with transient EEG arousals would seem to warrant consideration of treatment, given that sleep fragmentation diminishes its recuperative value (79). The occurrence of PLMS without evidence of EEG arousals may not contribute to insomnia complaints, although additional work is needed in this area.

### 5.3 Prolonged sleep latency

In most cases, prolonged sleep latency does not yield specific information regarding etiology, diagnosis or treatment. It is rarely found in the absence of a subjective complaint and would have uncertain significance in such a situation. Conversely, a complaint of prolonged sleep latency in the presence of "normal" polysomnographic findings strongly suggests a diagnosis of sleep state misperception. A complaint of prolonged sleep latency verified by PSG may aid in the selection of specific hypnotic agents (e.g. choosing a hypnotic with rapid onset of action).

Downey and Bonnet (84) recently described how objective information on sleep latency obtained by PSG can be used in a feedback procedure to improve the accuracy of sleep onset perception in a group of subjective increased sleep latency insomniacs. In a smaller number of cases, specific causes for sleep onset difficulty, such as limb movements or apnea, may be identified.

### 5.4 Measures of continuity, arousals, fragmentation

Polysomnography may confirm or quantify a subjective complaint. The absence of awakenings in the presence of a complaint may also be useful diagnostically (for sleep state misperception disorder). The actual number and duration of full awakenings have little diagnostic significance, although quantification may suggest more or less intensive treatment recommendations. A large number of very brief arousals (0.5–3.0 seconds) should alert the clinician to the possibility of other pathologies such as PLMS or apnea. Awakenings from specific sleep stages may also suggest specific diagnoses or treatments: for instance, a diagnosis of sleep terrors or sleep panic when awakenings occur from slow-wave sleep or a diagnosis of nightmare disorder with awakenings from REM sleep. However, in these cases, it would be unusual for chronic insomnia to be the presenting complaint.

To some extent, the failure of awakenings to suggest specific diagnoses or treatments may result from methodological shortcomings in previous studies. For example, few studies report the stage of sleep from which awakenings occur, the timing of such awakenings with reference to NREM/REM sleep cycles, or other psychophysiological correlates of awakenings, such as heart rate variability or skin conductance. When such details are reported, they may provide important characteristics of a syndrome. An example is the careful description of sleep panic attacks reported by Hauri et al. (85).

### 5.5 Special cases: medical disorders and the elderly

Patients with medical and neurological illness frequently have insomnia complaints, but there is little evidence that PSG offers special information to help with diagnosis or treatment. Sleep studies are more likely to be helpful if there is a specific suspicion of an intrinsic sleep pathology, if patients are elderly (86–88), or if the sleep complaints are not adequately explained by the type or degree of medical illness and medications. Examples would include a suspicion of PLMS in a patient with chronic renal disease, a suspicion of apnea in a patient with neuromuscular or chronic pulmonary disease, or a suspicion of a seizure disorder in a patient who has suffered head trauma or stroke.

The study by Edinger et al. (46) suggests that patients >40 years old more frequently demonstrate unsuspected PLMS and apnea. Obviously, the incidence of substance-related and secondary sleep disorders due to medical illness also increase with age. Therefore, polysomnographic findings are likely to be more helpful in older patients. There are no comprehensive data by which to define "older" in this context.

### 5.6 When PSG is normal

On occasion patients with insomnia complaints are studied by PSG and findings are within normal limits. This does not necessarily mean that the patients do not have bona fide sleep disorders, for both sleep state misperception syndrome (by definition) and some cases of psychophysiological insomnia (where sleep may be relatively normal in other than the normal bedroom environment) may have no specific abnormality on a PSG obtained in a non-home environment.

## 6.0 WOULD MORE COMPREHENSIVE POLYSOMNOGRAPHY BE OF GREATER HELP IN THE DIFFERENTIAL DIAGNOSIS AND MANAGEMENT OF INSOMNIA?

Polysomnography as conventionally performed is quite limited in the amount of physiological data actually provided, yet the time and cost is substantial. Emerging evidence suggests that a more comprehensive assessment of the insomniac patient's physiological status may help in separating subtypes of insomnia.

Areas of potential interest in this regard are assessment of the circadian system and a more comprehensive assessment of EEG activity.

### 6.1 Assessment of the circadian system

There is evidence for a close relationship between body temperature and sleep (89). Sewitch (90) has postulated that "the down regulation of body temperature at sleep onset is critical for the presence of slow wave sleep". Morris et al. (91) found that the temperature circadian rhythms in 13 sleep onset insomniacs were delayed about 2.5 hours compared to the temperature rhythms of nine normal sleepers when studied in a constant routine paradigm. The usual bedtimes of these sleep onset insomniacs fell within the wake-maintenance body temperature zone, whereas the bedtime body temperature of the good sleepers was well beyond their wake-maintenance zones. Campbell et al. (92) found altered phase relationships between body temperature and sleep in elderly women compared to elderly men, which corresponded to subjective sleep disturbances in the women. Evidence of this type supports the possible usefulness of systematic temperature assessment as part of a sleep onset insomnia work-up, and possibly as part of a polysomnographic evaluation. Findings of this type might suggest the appropriate use of light therapy, which has been found useful in phase shifting the circadian system (93) and as a treatment for delayed sleep phase syndrome (94).

Additionally, Matsumoto and Morita (95) found that older shift workers had evidence of a phase advance in circadian (oral) temperature rhythms, with decreases in amplitude and mesor that appeared to be associated with more difficulty with daytime sleep following night work. In situations such as this, information on circadian temperature measures might prove useful in understanding and treating sleep complaints.

These data suggest that routine recordings of temperature circadian rhythms might prove useful in establishing subtypes of insomnia associated with circadian phase alterations. Operationally, however, such recordings are not widely available, and their routine incorporation into conventional PSG will benefit from improvements in recording ease and reliability, and controlling for the effects of masking by activity or other rhythms.

### 6.2 Computerized EEG analysis

There is far more information present in the EEG than made available by conventional paper record-based sleep staging. A major question is whether that information, if properly extracted, might contribute to our understanding of the insomnia complaint, or help in the isolation and description of specific sleep disorders presenting as insomnia. The advent of digital computer technology has greatly enhanced our capability in this regard, as well as in a variety of analytic strategies, including period analysis, spectral analysis and various combinations.

Period analysis has been used in sleep quantification. Feinberg et al. (96) found that flurazepam decreased the amount of stage 4 sleep, but period analysis indicated this was primarily due to an amplitude decrease and that, in fact, delta-wave activity remained essentially at baseline levels during flurazepam administration. Period analysis has recently been used to quantify EEG sleep measures in normals (97,98) and depressed patients (99).

Freedman (27) compared C3A2 and O1A2 EEG power spectra in 12 insomniacs and 12 normal controls. The insomniac group exhibited greater relative beta activity during wakefulness, stage 1, and stage REM than normals, but no differences in EEG spectra in insomniacs and normals were found during the other NREM stages. Merica and Gaillard (54) examined the sleep onset period (about 3 minutes) in 26 insomniac and 28 control nights. Discriminant analysis based primarily upon beta and delta measures was found to significantly separate the groups, with beta activity contributing most prominently. Such findings suggest EEG spectral analysis may prove to be a useful strategy in studying insomnia, but insufficient data are available to further substantiate its usefulness. A combination of manual sleep staging and EEG spectral analysis was useful in distinguishing control subjects from elderly delusional and depressed patients (100).

Insomnia complaints have been found to be increased prior to nonfatal myocardial infarctions (101–103). In a recent study by van Diest and Appels (104), polysomnographic sleep patterns in a group of nine "exhausted" male adults with insomnia-related sleep complaints, whose complaints were considered typical of those at increased risk for near future myocardial infarction, were compared with the sleep patterns of eight normal controls. Spectral analysis of slow-wave sleep indicated that the "exhausted" group demonstrated significantly lower delta-band power; it was postulated that slow-wave sleep might exert an antiarrhythmic effect, and its decrease was associated with an increased risk for adverse myocardial events.

Spectral analysis facilitates measures of coherence, which offer considerable promise for examination of shared information and/or processing in different central nervous system regions or structures. Early studies by Brazier (105) demonstrated significant coherence in theta and alpha frequency bands between fronto-temporal cortex and dorsomedial thalamus in humans while awake, which disappear during sleep. More recently

scalp EEG coherence measures have been used in alert humans to demonstrate aspects of shared cortical information processing in certain task performance measures (106). Coherence measures during sleep have suggested greater beta-delta coherence in normals compared to depressed subjects (96). Regestein and colleagues have described increased levels of alpha and nonalpha activity, as well as higher amplitude auditory-evoked responses, in chronic insomnia subjects during daytime EEG recordings (107).

Conventional PSG does not quantify the amount of or specify the nature of spindle activity. Spindles appear to be generated in the thalamus and propagated to the cortex. During spindles, thalamocortical neurons may spend 80% of their time in a hyperpolarized state, creating a favorable condition for cortical deafferentation (108). Do the spindles merely represent epiphenomena associated with the prolonged states of hyperpolarization, or might they, as Steriade suggests, prevent the metabolic inertia that would otherwise exist during a period of inhibition lasting for 10 minutes or more? If the latter, would this information be of benefit in examination of phenomena such as the recuperative effects of sleep? Jobert and colleagues used EEG frequency mapping to quantify two distinct spindle types, a 12-Hz type occurring predominantly frontally and a 14-Hz type found parietally, with different responses to administration of benzodiazepine medications (109). Similarly, spectral analysis could contribute materially to quantifying (in topography, frequency and amount) sleep activities such as the so-called "alpha-delta" morphologies, that most of the time is only mentioned in passing in conventional PSG.

Thus, although sophisticated analytic capabilities are now available, sleep EEG analysis has yet to benefit systematically from the availability of this computational capability, and most of the literature continues to report sleep variables by conventional staging criteria. Still to be resolved is the question of how any or all available methodologies are to be used to further our understanding of the composition of the EEG during sleep, and what this information can contribute to our knowledge of the etiology and pathophysiology of sleep disorders.

## 7.0 CONCLUSIONS REGARDING THE USE OF POLYSOMNOGRAPHY IN INSOMNIA

A review of relevant literature suggests polysomnography as conventionally performed has a limited role in the evaluation of patients with insomnia complaints. It cannot, and should not, be substituted for a thorough clinical evaluation by a knowledgeable sleep specialist. It is the opinion of the authors that, unless there is strong presumptive evidence based on the clinical evaluation that the patient has an insomnia complaint related to a sleep-related breathing disorder or to periodic limb movements, little evidence suggests a conventional polysomnogram would contribute significant additional useful information.

Many clinicians believe that if a careful history and physical examination do not suggest the presence of physiological disturbances, such as sleep apnea or periodic limb movements, then it may be appropriate to first treat with nonpharmacologic and/or pharmacologic techniques for insomnia. The recent development of a structural interview for diagnosis of sleep disorders may improve accuracy of clinical diagnosis and probability of treatment response (110). If after a reasonable time the patient does not respond as expected, or develops a more complex clinical picture, a polysomnogram might be considered.

Overall, we believe the existent data would support the following conclusions:

1. The use of polysomnography is not supported for the routine evaluation of either transient or chronic insomnia. In many if not most cases, a therapeutic trial might be profitably undertaken prior to consideration of polysomnography.
2. Polysomnography is indicated if there is strong clinical evidence for the presence of a sleep-related breathing disorder or periodic limb movement disorder as the underlying cause of the insomnia. Polysomnography is more likely to yield important diagnostic information when conducted in older insomniacs.
3. Polysomnography may be indicated for insomnia patients who have failed a comprehensive behavioral and/or pharmacologic treatment program for the management of insomnia.

In summary, although conventional polysomnography may be of limited value in the routine evaluation of chronic insomnia, it is important to continue active research on how more sophisticated polysomnographic evaluation might improve our understanding of, and ability to treat, patients with these sleep disturbances. There are several areas where additional physiologic information not usually included in routine polysomnography, which include measurement of circadian and other biological rhythms, and more sophisticated spatial and temporal analysis of waking and sleep EEG data, may contribute to its value as a diagnostic tool. We must not forget that the insomnia complaint can be a disturbing and debilitating symptom, and increases in our knowledge of effective diagnostic and evaluation strategies is of fundamental clinical importance.

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