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Psychophysiological insomnia: the behavioural model and a neurocognitive perspective

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SUMMARY A number of paradoxes are apparent in the assessment and treatment of psychophysiological insomnia and sleep state misperception. Three of these paradoxes exist as discrepancies between polysomnographic (PSG) measures and the subjective impressions regarding sleep quality and quantity. The remaining incongruity exists largely within the objective domain. In the case of subjective–objective discrepancies, patients with insomnia: (1) frequently identify themselves as having been awake when awakened from PSG defined sleep; (2) tend to overestimate sleep latency and underestimate total sleep time as compared with PSG measures; (3) appear to derive more benefit from pharmacotherapy that can be explained by objective gains. The remaining paradox pertains to the observation that hypnotic medications, by and large, do not normalize sleep architecture or produce a more 'sleep-like' EEG. In this paper, we review possible explanations for these various paradoxes, introduce a new perspective and suggest possible research avenues. The model introduced is based on the observation that beta and/or gamma activity (which have been found to be associated with cognitive processes) is enhanced in insomnia at or around sleep onset. We propose that this kind of high frequency EEG activity may interfere with the normal establishment of sleep onset-related mesograde amnesia. As a result, the patient with insomnia maintains a level of information and/or memory processing that blurs the phenomenological distinction between sleep and wakefulness and influences retrospective judgments about sleep initiation and duration.

KEYWORDS behavioural model, beta activity, gamma activity, insomnia, subjective/objective discrepancies

INTRODUCTION

As many as one in four adults around the world have been reported to have problems with sleep initiation, sleep maintenance and/or non-restorative sleep (Angst *et al.* 1989; Janson *et al.* 1995; Silva *et al.* 1996). In the United States, approximately 26 million people or 10% of the population suffer from insomnia (Hammond 1964; Bixler *et al.* 1979; Institute of Medicine 1979; Karacan *et al.* 1983; Mellinger *et al.* 1985; Ford and Kamerow 1989; Gallup Organization 1991). Each year millions of prescriptions for hypnotic medications are prescribed to ameliorate such symptoms (Insitute of Medicine 1979; Mellinger *et al.* 1985; Shader *et al.* 1991; Pharmacy Times 1996). Despite the magnitude of the problem and the costs of treatment (Stoller 1994), little is known about the pathophysiology of insomnia, the mechanisms of action of hypnotic medication and how both relate to the experience of insomnia.

The complaint of insomnia may be symptomatic of a variety of disorders. Insomnia may be related to other sleep disorders such as nocturnal myoclonus or sleep apnoea, to pain associated with medical conditions, to medical conditions themselves, or to psychiatric disorders such as major depression (American Sleep Disorders Association 1990; Bootzin and Perlis 1992; Buysse and Perlis 1996). When contributing disorders such as

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these are ruled out, the most probable diagnoses are psychophysiological insomnia or sleep state misperception.

In the assessment and treatment of psychophysiological insomnia and sleep state misperception, a number paradoxes are apparent. Three of these paradoxes exist as discrepancies between objective polysomnographic (PSG) measures and subjective impressions regarding sleep quality and quantity. The remaining incongruity exists largely within the objective domain. The objective–subjective discrepancies pertain to (1) perception of PSG sleep, (2) subjective assessment of sleep latency and total sleep time, and (3) patient impressions of improvement when prescribed sedative hypnotics. The objective discrepancy pertains to the paradoxical EEG effects of sedative hypnotics. These incongruities appear to various extents in both psychophysiological insomnia and sleep state misperception insomnia. The two disorders, it may be argued, do not differ categorically (e.g. Salin-Pascual *et al.* 1992). For the purposes of this paper we will focus on the incongruities between the subjective impression of sleep and PSG measurement as they are manifested in psychophysiological insomnia. However, it should be borne in mind that our observations apply to both disorders.

THE FOUR PARADOXES OF INSOMNIA

The perception of PSG sleep

When awakened from polysomnographically-verified sleep, patients with psychophysiological insomnia more frequently report having been awake than good sleeper subjects (Borkevec *et al.* 1981; Coates *et al.* 1983; Mendelson *et al.* 1986; Mendelson *et al.* 1988). On average, the patient with insomnia tends to identify PSG sleep as wakefulness 73% of the time. Good sleepers identify PSG sleep as wakefulness between 45–50% of the time. These kind of findings vary as a function of number of sleep study nights, sleep stage from which the subject is awakened and the manner in which the subject is awakened. However, the discrepancy between PSG sleep and subjective impression is most evident for awakenings that occur shortly after sleep onset(s) (Mendelson 1995).

Subjective vs. PSG measures of sleep latency and total sleep time

Patients with insomnia tend to overestimate how long it takes them to fall asleep and the amount of time that they are awake over the course of the night when compared with objective assessments, e.g. polysomnography (Monroe 1967; Rechtschaffen and Monroe 1969; Bixler *et al.* 1973; Carskadon *et al.* 1976; Frankel *et al.* 1976; Lutz *et al.* 1977; Monroe and Marks 1977; Coates *et al.* 1982; Coates *et al.* 1983; Edinger and Fins 1995). Generally, patients estimate that it takes them 10–45 min longer to fall asleep and that they sleep 30–45 min less than is evident upon polysomnography. Good sleepers tend to estimate correctly how long it takes them to fall asleep, and if a trend is evident, they tend to overestimate the amount of sleep they obtain. Both patients with insomnia and good sleepers, when compared with polysomnographic measures, tend to underestimate number of awakenings or number of nocturnal arousals. This last finding may be an artifact of the manner in which sleep is scored. Most laboratories score in 30-s epochs and thus intervals as brief as 16 s may be categorized as wakefulness. Such extremely brief arousals may not be recalled by either good sleepers or patients with insomnia (Koukkou and Lehmann 1968, 1973).

Subjective vs. PSG measures of treatment efficacy

When treated with hypnotic medication, patients with insomnia tend to report greater benefits than can be explained by objective measures of sleep improvement (Mendelson and Maczaj 1990; Mendelson 1993; Mendelson 1994, 1995). While it is true that medication decreases sleep latency and increases sleep time, the magnitude of these effects are surprisingly moderate. Typically, sleep latency is reduced by about 15 min and total sleep time is increased by about 30 min (Mendelson *et al.* 1982; Mendelson *et al.* 1988; Mendelson and Maczaj 1990; Mendelson 1993; Mendelson 1995). The disproportionate relationship between perceived benefit and objective gains can be observed on the first night of treatment (e.g. Mendelson *et al.* 1982) and thus this paradox cannot be understood as 'improvement over time' (i.e. the additive effects of more sleep over a protracted period of time).

The EEG effects of sedative hypnotics

Despite reported subjective benefits, it is clear that benzodiazepine hypnotic medications do not normalize sleep: sleep architecture percentages remain unchanged and/or abnormal (Mendelson *et al.* 1982; Beary *et al.* 1984; Wittig *et al.* 1985) and the sleep EEG itself is faster than that observed in normal sleep or in the sleep of unmedicated insomniacs (Johnson *et al.* 1975; Johnson *et al.* 1983; Borbely *et al.* 1985; Borbely *et al.* 1991). With respect to the former, most hypnotic agents tend to decrease Stage 1 sleep and increase Stage 2 sleep but do not normalize slow wave or REM sleep percentages. With respect to the latter issue, soporific agent do not produce an EEG that is typical of normal sleep (i.e. more delta and theta activity) but instead introduce a high frequency component into the sleep EEG which occurs largely in the 12–14 Hz (sigma) range (Johnson *et al.* 1975; Johnson *et al.* 1983; Borbely *et al.* 1985; Borbely *et al.* 1991). This activity, referred to as a 'benzodiazepine signature', also appears to varying extents with the use of barbiturates and imidazopyridine (e.g. Lester *et al.* 1968; Johnson *et al.* 1975; Johnson *et al.* 1975; Schwartz *et al.* 1982; Brunner *et al.* 1991).

POSSIBLE EXPLANATIONS FOR THE PARADOXES OF INSOMNIA

Several of the paradoxes observed in insomnia could be, and have been, understood within a strictly psychological framework, i.e. in terms of psychological traits. The tendency

for patients with insomnia to 'overestimate how long it takes them to fall asleep', to 'identify PSG sleep as wakefulness' and, to 'underestimate the amount of time that they are asleep' over the course of the night may be related to a general tendency to overestimate severity of symptoms. Similarly, the insomnia patient's tendency to 'perceive greater improvement than can be justified by treatment effects', may be related to a tendency to be prone to placebo/expectancy effects (Bootzin *et al*. 1976). With respect to symptom exaggeration, there is substantial evidence, on such measures as the MMPI, to suggest that patients with insomnia are more prone to 'neuroticism' and 'hypochondriasis' (Corsey *et al*. 1975; Kales *et al*. 1976; Bixler *et al*. 1977; Monroe and Marks 1977; Kales *et al*. 1984b; Levin *et al*. 1984; Tan *et al*. 1984). With respect to expectancy effects, there is some evidence that patients with insomnia are prone to experimental demand characteristics (Bootzin *et al*. 1976).

While psychological traits, when taken together, may explain several of the paradoxes, stable personality factors cannot account for the EEG paradox of hypnotic treatment (i.e. that hypnotics appear to 'speed up' rather than 'slow down' the sleep EEG) or effects that occur as interactions. With respect to the latter, the general tendency toward neuroticism, hypochondriasis and susceptibility to expectancy effects, cannot explain: (1) how individuals develop insomnia in the first place; (2) why patients with insomnia report/exhibit improved sleep in some circumstances [(watching TV, in a motel, in the sleep laboratory (Hauri 1989)] but not others (in bed at bedtime) and/or; (3) why patients with insomnia do not uniformly identify all stages of sleep as wakefulness but rather do this predominantly around sleep onset (Mendelson 1995).

Since the mid 1980s, the etiology of insomnia has been understood within a behavioural framework. The perspective provides a compelling conceptualization and the treatments that derive from the theory have demonstrated clinical efficacy (e.g. McClusky *et al*. 1991; Engle-Friedman *et al*. 1992; Morin *et al*. 1994). The behavioural perspective also provides a way of understanding at least one of the interaction effects that occur in association with insomnia (i.e. better sleep in novel environments).

THE BEHAVIOURAL PERSPECTIVE ON PSYCHOPHYSIOLOGIC INSOMNIA

The behavioural model, as originally put forth by Spielman and colleagues (1987), posits that insomnia occurs acutely in relation to both trait and precipitating factors. Thus, an individual may be prone to insomnia due to trait characteristics, but experiences actual episodes because of precipitating factors. The acute insomnia becomes sub-chronic when it is reinforced by maladaptive coping strategies. These strategies, in turn, result in conditioned arousal and chronic insomnia (e.g. (Spielman *et al*. 1987; Bootzin and Perlis 1992; Buysse and Perlis 1996).

Trait factors extend across the entire biopsychosocial spectrum. Biological factors include hyperarousal or hyper-reactivity. Psychological factors include worry or the tendency to be excessively ruminative. Social factors, although rarely a focus at the theoretical level, include such things as the bed partner keeping an incompatible sleep schedule or social pressures to sleep according to non-preferred sleep schedule. Precipitating factors, as the name implies, are acute occurrences that constitute life stress events.

When an insomnia episode is initiated there are a variety of maladaptive strategies that individuals adopt in the attempt to get more sleep. Research and treatment have focused on two in particular: *excessive time in bed* and the practice of *staying in bed while awake*. Excessive time in bed refers to the tendency of patients with insomnia to go to bed earlier and/or to get out of bed later. Such changes are enacted in order to increase the opportunity to get more sleep. However, these behaviours lead to decreased sleep efficiency. That is, when the opportunity to sleep exceeds basal ability to generate sleep, the consequence is more frequent and longer awakenings. The practice of staying in bed while awake, as with the prior strategy, is enacted to increase the opportunity to get more sleep. In addition, the practice is often adopted under the rationale that staying in bed is at least 'restful'. While a seemingly reasonable behaviour, staying in bed while awake leads to an association of the bed and bedroom with arousal, not sleepiness and sleep. That is, when confronted with stimuli that are typically associated with sleep, they elicit arousal responses via classical conditioning.

The two maladaptive behaviours are likely to occur concurrently and promote one another. Excessive time in bed increases the likelihood that the individual will be awake while in bed. Being awake while in bed increases the likelihood that the individual will attempt to get more sleep by increasing sleep opportunity. The end result is conditioned arousal during the traditional sleep period and chronic insomnia.

The behavioural paradigm, aside from providing a theory about the etiology of insomnia and specific targets for treatment, provides a way of understanding why patients with insomnia report improved sleep in novel sleep environments. The novel sleep environment, because it is novel, has fewer visual (and perhaps temporal) cues for arousal and thus allows for transient improvements. It should be noted that there is actually very little systematic work on the clinical observation that patients with insomnia report sleeping better outside of the home. This said, Hauri and colleagues (1989) observations regarding the reverse first night effect (i.e. that patients with insomnia sleep better, rather than worse, on the first sleep laboratory study night) suggest that the clinical observation may be true.

Several investigators have attempted to refine the concept of conditioned arousal (e.g. Monroe 1967; Haynes *et al*. 1974; de la Pena 1978; Lichstein and Rosenthal 1980; Freedman and Sattler 1982; Kuo *et al*. 1994). Two constructs in particular have received attention: *somatic arousal* and *cognitive arousal*. The extent to which these different forms of arousal contribute to chronic insomnia, has been, and continues to be, a subject of investigation. Classic work by, for example, Monroe (1967) and Freedman and Sattler (1982) provide evidence that patients with insomnia are physiologically hyperaroused prior to sleep

onset and/or during PSG sleep. Recent work by Bonnet and colleagues (e.g. Bonnet and Arand 1995) has confirmed such findings using a measure of metabolic rate. Classic work by, for example, Lichstein and Rosenthal (1980) and Mitchel (1977) provide evidence that patients with insomnia are prone to intrusive cognitions at or around sleep onset. Recent work by Hall and colleagues (1996) provides evidence that experimentally induced cognitive arousal is associated with sleep initiation and maintenance difficulties.

To date, somatic and cognitive arousal have been considered, conceptually and empirically, separate phenomena. Somatic arousal has fallen with the domain of 'body' and thus has been explored via physiological study. Cognitive arousal, on the other hand, has fallen within the domain of 'mind' and thus has been explored via psychological study. We would like to propose that these domains are not orthogonal and introduce a perspective that may (1) account for the relationhip between these separate domains and (2) provide an alternative way of understanding the various paradoxes that occur in association with insomnia.

A NEUROCOGNITIVE PERSPECTIVE ON CHRONIC INSOMNIA

In keeping with the behavioural model, it is our position that acute insomnia is precipitated by life stress, that persistent insomnia occurs because of the engagement of maladaptive 'coping' strategies and that chronic insomnia occurs as a result of conditioned arousal. However, our model of chronic insomnia focuses on one form of conditioned arousal: cortical arousal. In this model, cortical arousal is a form of somatic arousal to the extent that it is a measure of brain, as opposed to mental, activity. However, it is also the case that cortical arousal is an analogue of 'cognitive arousal'. This is the case because it can be measured as a form of EEG that has been found to correlate with cognitive processes (e.g. Sheer 1976; Spydell *et al* 1984; Makeig 1993; Makeig and Inlow 1993; Jokeit and Makeig 1994; Lutzenberger *et al*. 1994; Pantev 1995; Pulvermuller *et al*. 1995; Jeffreys *et al*. 1996; Makeig and Jung 1996). This form of EEG activity, which occurs in the Beta and Gamma ranges, has been found to be elevated in patients with insomnia (Freedman 1986; Mercia and Gaillard 1991). We are proposing that high frequency EEG activity at or around sleep onset is a primary feature of chronic insomnia and that this form of conditioned arousal allows for a variety of sensory and cognitive phenomena that do not occur (or are at least diminished and/or suppressed) in good sleeper subjects.

In specific, we are proposing that as one develops chronic insomnia (via behavioural contingencies), there is an increase in high frequency EEG activity at or around sleep onset. In transient insomnia such activity may occur in association with stress induced worry and/or rumination. In chronic insomnia, high frequency EEG activity occurs as a result of classical conditioning. That is, high frequency EEG is elicited in response to the visual and/or temporal cues usually associated with sleepiness and sleep (e.g. bedroom, bed, bedtime) and this occurs in the absence of situational stressors. High frequency EEG activity, in turn, allows for increased 'sensory processing', 'information processing' and the 'formation of long term memory'. The latter two phenomenon, it is hypothesized, account for the paradoxes observed in association with insomnia.

In the following section, we will highlight each of the described components and discuss how they are related to the measure and experience of insomnia and the potential mechanisms of actions of sedative hypnotics. In the next section we specifically apply the neurocognitive model to the four paradoxes of insomnia. In the final section of this paper we will highlight research avenues that may be useful for testing the various assumptions and components of our model. For a schematic representation of the model, see Fig. 1.

THE COMPONENTS OF THE NEUROCOGNITIVE MODEL AND RELATED EVIDENCE

Cortical arousal

There has been a substantial amount of work in the last two decades that suggests that high frequency EEG activity in the beta (14–32 Hz) and more probably in the gamma (>32 Hz) range is associated with cognitive function (e.g. Sheer 1976; Spydell *et al*. 1984; Makeig 1993; Makeig and Inlow 1993; Jokeit and Makeig 1994; Lutzenberger *et al*. 1994; Pantev 1995; Pulvermuller *et al*. 1995; Jefferys *et al*. 1996; Makeig and Jung 1996). There is still considerable debate about what cortical regions (e.g. frontal vs. temporal) and what frequency bands (e.g. 40 Hz vs. 80 Hz) are most associated with cognitive behaviour. This notwithstanding, it is now possible to construe 'cognitive arousal' as 'cortical arousal' and to measure this construct in terms of high frequency EEG activity.

High frequency EEG and insomnia

Typically the transition from wakefulness to sleep entails a shift in the frequency spectum of the EEG from high frequency (e.g. 8–80 Hz) to low frequency activity (0.5–7.0 Hz) (Hori 1985; Wright *et al*. 1995). Recent work has demonstrated that subjects with insomnia, when compared with normal control subjects, exhibit increased high frequency EEG activity (>20 Hz) prior to sleep onset and for a period that may be as long as 15 min into the first NREM cycle (Freedman, 1986; Mercia and Gaillard, 1991). This finding, in combination with the finding that high frequency EEG activity may be associated with cognitive activity, suggests that peri-sleep onset cortical arousal may be a feature of chronic psychophysiological insomnia. The presence of such activity also suggests that the patient with insomnia may be sensory processing, information processing and/or forming long term memories during periods when such capacities are normally substantially attenuated (e.g. Portnoff *et al*. 1966; Koukkou and Lehmann 1968; Goodenough *et al*. 1971; Koukkou and Lehman 1973; Lasaga and Lasaga 1973; Lehmann and Koukkou 1974; Guilleminault

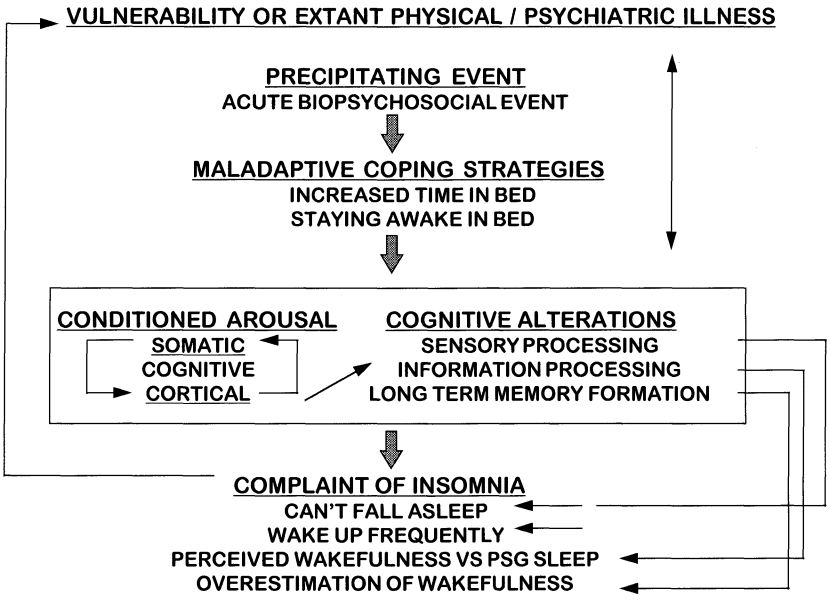

Figure 1. A schematic representation of the behavioural model and the role of neurocognitive processes.

and Dement 1977; Bonnet 1983; Bootzin *et al.* 1991; Wood *et al.* 1992; Anthony *et al.* 1994; Wyatt *et al.* 1994b). Not clear from the prior work, however, is whether high frequency EEG activity occurs in association with all sleep-wake transitions or whether this effect is limited to the initial sleep onset. We hypothesize that, although probably most pronounced at the initial sleep onset, high frequency EEG characterizes all the sleep-wake transitions that occur over the course of the night.

Enhanced sensory processing

Enhanced sensory processing at, or around, sleep onset may be directly associated with sleep initiation and/or sleep maintenance difficulties. That is, if the individual is particularly vulnerable to perturbation by environmental stimuli, they will be more likely to have trouble falling and staying asleep. If enhanced sensory processing is a feature of insomnia, this may help explain one of the benefits of sleeping pills. Hypnotics may directly diminish sensory processing and protect the insomnia patient from perturbing environmental stimuli.

To our knowledge there are no data on the association between beta and gamma activity and measures of sensory processing in insomnia. However, there is evidence that cortical arousal (in the form of alpha sleep) is associated with an increased 'EEG arousability' (Perlis *et al.* 1997). In this study, fibromyalgic patients with high amounts of alpha activity (as compared with fibromyalgic subjects with low amounts of alpha activity) exhibited larger EEG arousals in response to auditory stimuli presented during PSG sleep. This suggests that even nominal levels of high frequency EEG activity may be associated with increased sensory processing. With respect to the effects of hypnotics on sensory processing, there is good evidence that hypnotics, and particularly benzodiazepines, decrease startle reflexes and the amplitude of early and late component ERPs during wakefulness (e.g. Martin *et al.* 1992; van Leeuwen *et al.* 1992; Pang and Fowler 1994; Joordens *et al.* 1996), and increase arousal thresholds durng sleep (e.g. Bonnet *et al.* 1979; Mendelson *et al.* 1988). Thus it seems plausible that hypnotic efficacy may derive, in part, from the diminution of sensory processing. That is, benzodiazepines may protect the insomnia patient from perturbing environmental stimuli.

'*Enhanced information processing*' at around sleep-onset (i.e. the perception of environmental stimuli) may be directly related to the perception of sleep and wakefulness. The ready perception of perturbing environmental stimuli may blur the phenomenological distinction between sleep and wakefulness. That is, one of the cues for 'knowing' that one is asleep or awake may be a lack of awareness for events occurring during sleep. If one is asleep by PSG criteria, and yet still capable of information processing (identifying sensory information and retaining it in short-term memory), then when awakened following such an interval, subjects would be expected to have trouble assessing whether they were asleep or awake prior to the awakening. Enhanced information processing may therefore account for the tendency in insomnia to judge PSG sleep as wakefulness. If enhanced information processing is a feature of insomnia, this may help explain another of the benefits of sleeping pills; hypnotic medications may diminish information processing because they block sensory processing. Thus the patient will be less aware of environmental events and more likely to conclude that he/she was asleep.

To our knowledge, no research has been undertaken on the relationship between high frequency EEG activity, short-term memory function and the perception of sleep. However, there have been two studies that evaluate the association of alpha activity during sleep to short-term memory function and perception of sleep (Engle-Friedman *et al.* 1985; Perlis *et al.*

1997). In a preliminary study by Engle-Friedman *et al*. (1985) alpha sleep was found to be associated with increased short-term memory function and an increased tendency to perceive PSG-defined sleep as wakefulness. In the study by Perlis and colleagues (1997), alpha sleep was not found to be associated with increased short-term memory function or the tendency to perceive PSG sleep as wakefulness. In the latter study, it was concluded that alpha sleep, although higher frequency than normal EEG sleep, may be sufficient for sensory processing but is not sufficient for the formation of short-term memory. With respect to the effects of hypnotics on short-term memory function and the perception of sleep, there have been very few studies evaluating short-term memory function but a variety of studies have found that patients treated with hypnotic medications are less likely to perceive PSG defined sleep as wakefulness (e.g. Mendelson *et al*. 1988; Mendelson and Maczaj 1990; Mendelson 1993; Mendelson 1995). This effect appears to be particularly evident for sleep periods (Mendelson 1995).

'*Enhanced long-term memory function*' at or around sleep onset may interfere with the attribution process that affects morning judgments about ease of initiation and duration of sleep. Normally, subjects cannot recall information from periods 'immediately prior' to sleep (Portnoff *et al*. 1996; Guilleminault and Dement 1977; Wyatt *et al*. 1994a; Anthony *et al*. 1994), 'during sleep' (Koukkou and Lehmann 1968; Lasaga and Lasaga 1973; Lehmann and Koukkou 1974; Bootzin *et al*. 1991; Wood *et al*. 1992;), or 'from brief arousals' which occur during the night (Goodenough *et al*. 1971; Bonnet 1983). If the ability to encode and retrieve information, at least for sleep initiation periods, is intact in insomnia, this would be expected to substantially influence judgments about latency to sleep onset and sleep duration. Thus, it could be argued that enhanced memory function at or around sleep onset may account for the tendency in insomnia to overestimate retrospectively (1) the amount of time it takes to fall asleep and (2) the amount of time spent awake during the night.

Enhanced memory function during sleep may also be helpful in understanding why patients with insomnia tend to perceive dramatic treatment gains with hypnotics despite only modest objective gains in total sleep time. In this instance, the potency of sedative hypnotics may not reside only with their soporific potential but also with their ability to produce anterograde and/or retrograde amnesia (Lister 1985; Curran 1986; O'Boyle 1988; Polster *et al*. 1993). The patient may perceive that sleep is improved because the medication effectively prevents the encoding and/or recollection of the kind of information that promotes a negative assessment of sleep quality and quantity.

To our knowledge there is only one study in the literature that evaluates the association of high frequency EEG activity at sleep onset and the formation of long-term memory. In this study by Wyatt and colleagues (Wyatt *et al*. 1994; Wyatt *et al*. 1997), it was found that increased beta activity is associated with enhanced long-term memory function for auditory information presented prior to sleep onset. This study, while undertaken in normal subjects, strongly suggests that the increased beta activity observed in patients with insomnia may indeed be related to increased long-term memory function. With respect to the effects of hypnotics on long-term memory function, there is no doubt that at least benzodiazepines have potent amnestic properties (Lister 1985; Curran 1986; O'Boyle 1988; Polster *et al*. 1993). There is also substantial evidence that patients report great benefit from hypnotic use. Whether the two phenomena are substantially correlated remains to be determined.

THE NEUROCOGNITIVE PERSPECTIVE AND THE PARADOXES OF INSOMNIA

The perception of PSG sleep

As noted earlier, patients with insomnia are at least 20% more likely to identify PSG defined sleep as wakefulness. As part of the neurocognitive perspective, we hypothesized that sleep state misperception occurs as a result of high-frequency EEG related increases in information processing. That is, the ready perception of perturbing environmental stimuli serves to blur the phenomenological distinction between sleep and wakefulness. Given that high frequency EEG activity appears to be centered at sleep-wake transitions, it follows that such 'misperceptions' should be most likely to occur at sleep onset. This deduction is consistent with the work of Mendelson and colleagues who found that sleep state misperceptions are most evident for awakenings that occur shortly after sleep onset(s) (Mendelson 1995).

Subjective vs. PSG measures of sleep latency and total sleep time

In summarizing the various paradoxes that occur in association with insomnia we noted that patients with insomnia estimate, relative to PSG measures, that they take between 10–45 min longer to fall asleep and acquire between 30–45 min less sleep. With respect to sleep onset, these numbers roughly approximate the amount of time that there is persistent high frequency EEG after sleep onset (Freedman 1986; Mercia and Gaillard 1991). Thus it follows that, if high frequency EEG activity is associated with increased information processing and long-term memory formation, then the patient with insomnia is correctly estimating the amount of time required to disengage from the environment. Moreover, if the average person with insomnia awakens 3–5 times per night, then roughly 10 min offset should cause them to misestimate total sleep time by 30–50 min.

Subjective vs. PSG measures of treatment efficacy

The same reasoning that applies to overestimation of symptom severity also applies to perceived treatment gains. If it is the case that hypnotics diminish the ability to information process and/or promote mesograde amnesia, the patient receiving treatment should overestimate treatment gains relative to polysomnography.

The EEG effects of sedative hypnotics

Of the four paradoxes, the effect of hypnotics on the sleep EEG is the most puzzling and appears to be in strong contradition to the neurocognitive model. *How can an efficacious hypnotic speed up, rather than slow down, the sleep EEG*? Even if it is the case that sensory, information and/or long-term memory processing are altered in insomnia (and indirectly treated with pharmacotherapy), high frequency EEG activity is not likely to be the biologic correlate of these abnormalities. Some investigators have suggested that medication-related increases in sigma frequency activity actually represent increased spindling and an intensification of the processes associated with normal spindling, i.e. increased sensory inhibition occurring at the level of the thalamus (Johnson *et al*. 1975). While there is evidence that hypnotic medications increase auditory arousal thresholds (e.g. Mendelson *et al*. 1988), the benzodiazepine signature lacks the morphological characteristics of sleep spindles (i.e. a waxing and waning appearance that is of short duration and is periodic) and there is no evidence that benzodiazepine-induced sigma activity is similarly topographically distributed or thalamically generated (Steriade 1994).

A resolution to this apparent contradiction may reside in how one conceives of 'high frequency' EEG activity. The benzodiazepine signature (12–14 Hz) is, comparatively speaking, faster than the tonic delta activity that occurs during sleep. However, it is also substantially slower than the beta (14<32 Hz) and gamma (>32 Hz) frequencies that characterize alert wakefulness and/or are associated with cognition (e.g. Sheer 1976; Spydell *et al*. 1984; Makeig 1993; Makeig and Inlow 1993; Jokeit and Makeig 1994; Lutzenberger *et al*. 1994; Pantev 1995; Pulvermuller *et al*. 1995; Jefferys *et al*. 1996; Makeig and Jung 1996). Because beta activity has been observed in insomnia (Freedman 1986; Mercia and Gaillard 1991), it is possible that benzodiazepines produce the anticipated slowing of the EEG (e.g. from 40 Hz to 12 Hz), but that it simply occurs in a range not typically studied during sleep and has not been assessed relative to pharmacological interventions.

POSSIBLE RESEARCH AVENUES

In order to test the proposed model, a variety of protocols could be undertaken. One productive approach would be to present auditory information during brief awakenings in patients with insomnia and good sleep controls and then test for differential recall at the end of the sleep period. Such a protocol could be engaged naturalistically, as part of a forced awakening paradigm (Mendelson and Maczaj 1990) and/or in combination with pharmacological probes (e.g. with various hypnotic and/or amnestic medications). These kind of projects would allow for the replication of Friedman and Sattler (1992), and Mercia and Gaillard's (1991) finding regarding high frequency EEG activity, the assessment of whether information processing and/or memory function are related to high frequency EEG activity and allow for the exploration of how these effects are correlated and/or affected by medical treatment. Moreover, given a large sample, evaluating sex and age interactions might prove illustrative. Given that insomnia complaints are more prevalent in the elderly (e.g. Mellinger *et al*. 1985) and in women (Liljenberg *et al*. 1988; ASDA 1990; Rosekind 1992; Radecki and Brunton 1993), one would expect to find increased amounts of high frequency EEG related information processing and memory formation in these subgroups. We are currently undertaking such a project. Other possibilities include testing one of two assumptions underlying our perspective, or testing one of the assumptions underlying specifically the behavioural perspective.

Our assumption that cortical arousal is a primary feature of insomnia, as opposed to rumination, could be tested by evaluating the speed of arousal responses as they are elicited. Extremely fast response latencies might indicate that rumination cannot be a primary mediator of the arousal response in chronic insomnia. Similar approaches have been used to provide evidence against the primacy of cognition in emotion (Zajonc 1980; Zajonc 1984).

Our assumptions regarding 'levels of processing' could be assessed using ERP technology. For example, an 'odd ball' paradigm (using beeps and words) (Wyatt *et al*. 1997) could be used to explore sensory and information processing differences in patients with insomnia and in good sleeper controls. Such a paradigm could be undertaken naturalistically or in combination with pharmacological probes. Alterations in early ERP components in response to auditory stimuli (larger amplitudes and/or shorter latencies in the insomnia group) would suggest enhanced sensory processing. Alterations in late ERP components in response to auditory stimuli (larger amplitudes and/or shorter latencies in the insomnia group) would suggest enhanced information processing and possibly increased long-term memory formation.

Finally, the behavioural assumption that normal sleep-eliciting stimuli can be conditioned to elicit arousal responses could be tested in good sleeper subjects by repeatedly pairing arousal, mechanically-or-pharmacologically induced, with a sleep related stimulus (e.g. the sleep laboratory bed). If such maladaptive conditioning is possible, and resembles that which occurs in insomnia, it should be found that sleep continuity is negatively affected and that such effects should be persistent after the eliciting circumstances are discontinued.

CONCLUDING REMARKS

To date, most of the factors that have been associated with insomnia (e.g. neuroticism, metabolic hyperarousal, EEG abnormalities, etc.) have been implicated at a correlational level. Few investigators have hypothesized the direction of the correlations, and/or the possible mediating links. The neurocognitive perspective is powerful because it can establish direct links, and causal directions, between several domains including the cognitive (information processing and memory function), the symptom complaint (perceived sleep deficits), objective measures (high frequency EEG activity) and one of

the effects of pharmacotherapy (the amnestic properties of most hypnotic medications). If it is found that memory function around sleep onset is altered in insomnia, that this alteration is associated with increased high frequency EEG activity and that both of these phenomena are altered by medication, a strong case can be made that at least part of the pathophysiology of insomnia resides in a cognitive dysfunction which is mediated by an abnormal neurophysiological process, i.e. altered information and memory processing which stems from elevated cortical arousal. Moreover, what has been traditionally considered a side-effect of hypnotic medications (memory effects) will have been found to be a factor that contributes to treatment efficacy. Both findings will strongly influence how insomnia is conceived of and which psychological and physiological functions should be targeted for treatment.

Finally, it should be noted that our conceptual framework for psychophysiological insomnia, and particularly the subjective experience of insomnia, is that it is inherently a sleep disorder. There are, of course, several alternative points of view. One is that insomnia is a 24 h problem (e.g. Stepanski *et al*. 1988; Regenstein *et al*. 1993) and that to construe it as specifically a sleep disorder may be misbegotten. One example of such a perspective is the concept that patients with insomnia are somatically hyperaroused 24 h a day. During the daytime this may be associated with anxiety and/or affective symptomatology. During the night-time this is manifested as insomnia. Another point of view is that insomnia is not a disorder but rather a symptom. This is certainly true with respect to such maladies as sleep apnoea and periodic leg movements. But even when such 'intrinsic' sleep disorders are ruled out, it is still possible to conceive of the diagnostic entity of psychophysiological insomnia as actually symptomatic of other more systemic diseases. For example, psychophysiological insomnia may be a feature of undiagnosed, or incipient depression or a prodromal symptom of affective disorders. There are several recent studies that support these possibilities (Ford and Kamerow 1989; Dryman and Eaton 1991; Breslau *et al*. 1996; Perlis *et al*. 1997).

In the case of both alternative view points, it is possible that one facet of the parent disorder is a fundamental change in the sleep EEG and in the ability to disengage sensory/cognitive processes at specifically the sleep–wake transition. Thus it may be the case that there is a fundamental problem at sleep onset in patients who present with the complaint of chronic insomnia irrespective of whether the insomnia is associated with other more primary disorders. Alternatively, it is also possible that primary insomnia (psychophysiological insomnia) is unique because it is characterized by increased information processing and long-term memory formation while secondary insomnia is related to only increased sensory processing. These distinctions, along with other possible variations on the neurocognitive model, remain to be explored.

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