BEHAVIORAL AND BRAIN SCIENCES (2000) **23,** 793–1121
Printed in the United States of America

A review of mentation in REM and NREM sleep: "Covert" REM sleep as a possible reconciliation of two opposing models

Tore A. Nielsen
Sleep Research Center, Hôpital du Sacré-Coeur de Montréal, Montréal, Québec, Canada and Psychiatry Department, Université de Montréal, Québec H4J 1C5, Canada
t-nielsen@crhsc.umontreal.ca

Abstract: Numerous studies have replicated the finding of mentation in both rapid eye movement (REM) and nonrapid eye movement (NREM) sleep. However, two different theoretical models have been proposed to account for this finding: (1) a one-generator model, in which mentation is generated by a single set of processes regardless of physiological differences between REM and NREM sleep; and (2) a two-generator model, in which qualitatively different generators produce cognitive activity in the two states. First, research is reviewed demonstrating conclusively that mentation can occur in NREM sleep; global estimates show an average mentation recall rate of about 50% from NREM sleep – a value that has increased substantially over the years. Second, nine different types of research on REM and NREM cognitive activity are examined for evidence supporting or refuting the two models. The evidence largely, but not completely, favors the two-generator model. Finally, in a preliminary attempt to reconcile the two models, an alternative model is proposed that assumes the existence of *covert* REM sleep processes during NREM sleep. Such covert activity may be responsible for much of the dream-like cognitive activity occurring in NREM sleep.

Keywords: cognition in sleep; dreaming; NREM sleep; REM sleep; sleep mentation

1. Introduction

1.1. The discovery of REM and NREM mentation

Initial reports of an association between REM sleep and vivid dreaming (Aserinsky & Kleitman 1953; Dement 1955; Dement & Kleitman 1957a; 1957b) inspired studies designed to clarify relationships between sleep physiology and dream imagery. A perspective emerged – referred to by many as the "REM sleep = dreaming" perspective (see Berger 1994; Foulkes 1993b; Lavie 1994; Nielsen & Montplaisir 1994; Rechtschaffen 1994 for overview) – from which dreaming was viewed as a characteristic *exclusive* to REM sleep. Mentation reported from NREM sleep was attributed to purportedly confounding factors, for example, recall of mentation from previous REM episodes or subjects' waking confabulations. Many subsequent studies cast doubt on the "REM sleep = dreaming" perspective (Foulkes 1962; 1966) primarily by demonstrating elevated levels of mentation recalled from NREM sleep stages. Although the REM sleep = dreaming belief did not disappear entirely, a debate over whether the *quality* of NREM and REM sleep mentation reports differ largely overshadowed it. Initially, qualitative differences in REM and NREM reports suggested that a different – possibly degraded – form of mentation occurs in NREM sleep. From these developments, two relatively distinct points of view concerning REM/NREM mentation emerged and continue to influence the field. These points of view differ as to whether they consider NREM sleep mentation to stem from imagery processes that are fundamentally the same as or different from those that produce REM sleep mentation. I refer to these as the 1-gen (one-generator) and 2-gen (two-generator) models (reviewed in Nielsen 1999a); research supporting and/or refuting each model is reviewed in the following sections. The review concludes with the presentation of a third model, *the covert REM sleep processes*

TORE NIELSEN is Assistant Professor of Psychiatry at the Université de Montréal where he has been Director of the Dream & Nightmare Laboratory at the Hôpital Sacré-Couer de Montréal since 1991. He is a past Research Scholar of the Canadian Institutes of Health Research and presently a Senior Research Scholar of the *Fonds de la recherche en santé du Québec* (FRSQ or *Quebec Health Research Agency*). His main research interests are reflected in his many published papers on mechanisms of sleep mentation production, psychophysiological and EEG correlates of dreaming, hypnagogic imagery processes, disturbances of dreaming (including idiopathic and post-traumatic nightmares), and pain in dreams.

 0140-525X/00 $12.50

model, which combines aspects of both the 1-gen and 2-gen models in a way that may help to reconcile the two opposing points of view.

1.1.1. The 1-gen and 2-gen models. The 1-gen model stipulates that a single set of imagery processes produces sleep mentation regardless of the sleep stage in which it occurs. The model was suggested following demonstrations that reports of *cognitive activity* could be elicited from NREM sleep. Foulkes's (1962) application of more liberal criteria for identifying cognitive activity, as opposed to *dreaming activity,* allowed him and others to demonstrate a higher incidence of mentation during NREM sleep than was previously observed. Many others replicated these findings (see sect. 1.2.2.2).

Further support for 1-gen models came with the development of methods for effecting fair comparisons of mentation quality between reports of obviously different lengths. As REM sleep mentation reports were typically longer than their NREM equivalents, their qualitative attributes were thought to be confounded with quantitative attributes. Both Foulkes (Foulkes & Schmidt 1983) and Antrobus (1983) devised methods for removing quantitative differences and thus permitting – presumably – fair tests of residual qualitative differences. Both investigators found that when length of report was statistically controlled, qualitative differences diminished and often disappeared, a finding supporting the notion that all sleep mentation derives from *a common imagery source that is driven by different levels of brain activation.* Several models based upon the 1-gen assumption were subsequently elaborated (Antrobus 1983; Feinberg & March 1995; Foulkes 1985; Solms 1997a).

Foulkes's 1-gen model – the most influential – stipulates that mentation report from REM and NREM sleep arise from the same processes: (1) memory activation, (2) organization, and (3) conscious interpretation. Mentation differences stem primarily from differences in memory activation. When such activation is high and diffuse, during most REM but some NREM sleep, then organization is more intensely stimulated and conscious interpretation more probable and coherent. When memory activation is low and less diffuse, during most NREM but some REM sleep, then organization is less intensely stimulated and conscious interpretation less probable and coherent. It is thus the *diffuseness or availability of diverse memory elements* and not sleep stage physiology that determines the occurrence and form of sleep mentation.

Solms (1997a) adds some support to this model, primarily by refuting the physiological bases of Hobson's 2-gen model. He shows that lesions of the brainstem regions responsible for REM-related activation do *not* lead to loss of dreaming, whereas lesions in the forebrain ("anterior to the frontal horns of the lateral ventricles") or in the inferior parietal regions ("parieto-occipito-temporal junction"), lead to global cessation of dreaming. Mentation may occur in any state if these areas are active, even though it is most likely in REM sleep. Thus Solms, like Foulkes, views dreaming as largely independent of REM sleep-specific physiology. Unlike Foulkes, however, he does see dreaming to be associated with a neurophysiological substrate. The latter consists of a motivational-hallucinatory mechanism that is more akin to the Freudian psychoanalytical model than it is to a cognitive-psychological one (Solms 1995).

From the 2-gen perspective, REM and NREM sleep mentation reports stem from *qualitatively different* imagery generation systems. This difference was suggested by early findings that REM sleep reports are less thoughtlike, more elaborate, more affectively, visually and kinesthetically involving, and more related to waking life than are NREM sleep reports (Foulkes 1962; 1966; Monroe et al. 1965; Rechtschaffen et al. 1963a). The best-known 2-gen model was developed from the earlier activation-synthesis (A-S) hypothesis (Hobson & McCarley 1977) by Hobson's group (Hobson 1992a; Hobson & Stickgold 1994a; 1995; see also Seligman & Yellen 1987). McCarley (McCarley 1994; Steriade & McCarley 1990b) also updated the A-S hypothesis in different directions. A psycholinguistic 2-gen theory has also been proposed (Casagrande et al. 1996a).

Both the A-S hypothesis and its more recent variant (see Hobson et al., this issue) explain sleep mentation by combining (1) descriptions of the presumed physiological substrates of REM and NREM sleep (see Hobson 1988b; Kahn et al. 1997; McCarley & Hobson 1979 for reviews of the physiological findings) and (2) the assumption of formal mind-brain isomorphism. REM and NREM sleep physiological attributes determine the form of mental experiences and are isomorphic with them (Mamelak & Hobson 1989a). Dreaming mentation – characteristic of REM sleep – is distinguished from nondreaming mentation – characteristic of NREM sleep – according to the presence of six defining characteristics (Hobson & Stickgold 1994a): hallucinoid imagery, narrative structure, cognitive bizarreness, hyperemotionality, delusional acceptance, and deficient memory of previous mental content. Some of these features are embodied in newly proposed dream-content measures (e.g., emotional profile, visual continuity, thematic coherence; Baars & Banks 1994).

1.1.2. Summary. Both 1-gen and 2-gen models have had an important impact on sleep research over the last 40 years. That Foulkes's original findings were replicated and his model tested by so many researchers indicates that his cognitive-psychological framework and his 1-gen model have had a widespread influence. Solms's recent work further bolsters some of Foulkes's key assumptions while refuting others.

Until quite recently, the 2-gen model has been highly visible among the neurosciences and the popular press. The A-S hypothesis is today almost synonymous with dreaming. It has, nonetheless, been roundly criticized for various reasons (see below). How the model relates to dream content remains to be studied in greater depth, for example, discriminant validity of the index measures of the six proposed defining features of dreaming and non-dreaming mentation is still unknown.

As the use of cognitive methods has grown increasingly more popular in the brain and psychological sciences, both 1-gen and 2-gen models have continued to stimulate research within distinct subdisciplines. The result has been that the pros and cons of the two models have been scrutinized ever more closely, even though the two are only rarely compared directly one with the other.

1.2. Widespread evidence for cognitive activity in NREM sleep

1.2.1. Distinguishing "dreaming" from "cognitive activity." Distinctions between "dreaming" and "cognitive activity"

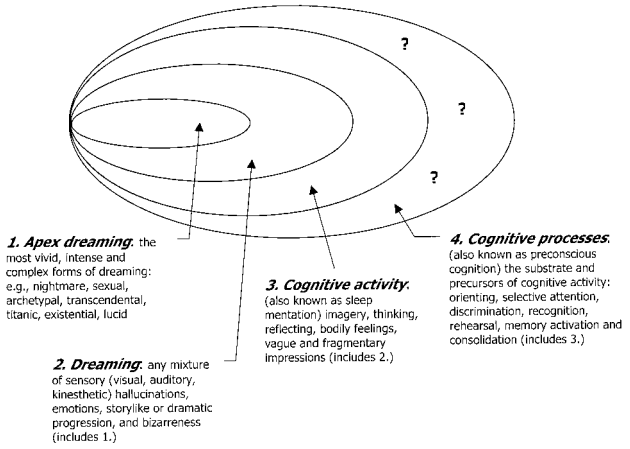

Figure 1. Four levels of specificity in defining sleep mentation. With an increasingly specific definition of sleep mentation, differences between REM and NREM mentation become more apparent. The two most specific levels (1 and 2) tend to occur much more exclusively in REM sleep. Cognitive activity (3) other than dreaming is predominant in NREM sleep. Beyond cognitive activity, there is likely an even more general level of cognitive processes (4) that consists of preconscious precursors to cognitive activity and that may be present in different degrees throughout REM and NREM sleep.

are key to appreciating differences between the 1-gen and 2-gen models. In general, dreaming – which is the object of study of most 2-gen theorists – is more specific than is cognitive activity (see Fig. 1). It is likely to be defined as imagery that consists of sensory hallucinations, emotions, storylike or dramatic progressions, and bizarreness, and that may exclude some types of cognition such as simple thinking, reflecting, bodily feeling, and fragmentary or difficult to describe impressions.

Nonetheless, there is currently no widely accepted or standardized definition of dreaming; definitions vary widely from study to study. There have been attempts to differentiate minimal forms of dreaming from more elaborate, vivid and intense forms, such as "everyday" and "archetypal" (Cann & Donderi 1986; Hunt 1989), "mundane," "transcendental," and "existential" dreaming (Busink & Kuiken 1996), "lucid" and "nonlucid" dreaming (Laberge et al. 1981), and ordinary versus "apex" (Herman et al. 1978) or "titanic" dreaming (Hunt 1989). In Figure 1, the term "apex" dreaming is adopted to refer to a subcategory of dreaming that is distinguished by exceptional vividness, intensity or complexity. Many of the forms mentioned above and other common types (e.g., nightmares, lucid dreams, sex dreams) fall into this category. The fact that such vivid dreaming occurs frequently during REM sleep but rarely during NREM sleep has led many to propose a qualitative difference between REM and NREM mentation, and thus to entertain a 2-gen perspective.

Cognitive activity is a more inclusive term than is dreaming. It is synonymous with the common term "sleep mentation" and refers to the *remembrance of any mental activity having occurred just prior to waking up* (Fig. 1). This may include static visual images, thinking, reflecting, bodily feeling, or vague and fragmentary impressions. However, the precise limits of this inclusiveness have not been clearly established. In a manner analogous to the model presented by Farthing for waking state consciousness (Farthing 1992), cognitive activity during sleep could be viewed as a subset of an even more inclusive category (cognitive processes) that includes preconscious or "nonconscious" information processes (Fig. 1). Processes that are acknowledged building blocks of waking cognition, such as orienting, selective attention, sensory discrimination, recognition, rehearsal, memory activation, and consolidation, have also been shown to be active during sleep (see sect. 2.2) and are more or less accessible to consciousness. For example, most theorists presume that processes of memory retrieval are central to dream generation. In principle, such processes may be active whether or not they possess phenomenological correlates (e.g., sensory imagery) that can be recalled. However, many such processes can in principle become accessible to awareness if subjects are properly trained in self-observation and reporting (see Nielsen 1992; 1995 for examples). The fact that relaxation training (Schredl & Doll 1997) and probe-based interview techniques (Smith 1984) can enhance the amount and quality of recalled mentation illustrates this point. More research bearing on this question is needed.

Differences in definitions of "cognitive activity" and/or "dreaming" presumably account for much of the variability in levels of mentation recall from REM and NREM sleep that has been observed in previous studies. To illustrate, three different studies of NREM sleep mentation used three different definitions of content: a report of (1) "coherent, fairly detailed description of dream content" (Dement & Kleitman 1957b); (2) "a dream recalled in some detail" (Goodenough et al. 1959), and (3) "at least one item of specific content" (Foulkes & Rechtschaffen 1964). The different levels of stringency varied inversely with the number of awakenings with recalled NREM mentation, that is, 7, 35, and 62% respectively.

1.2.2. Evidence for dreaming and cognitive activity in NREM sleep. Numerous studies demonstrate cognitive activity during NREM sleep. How much of this activity qualifies as *dreaming* (or as *apex dreaming*) has been less clearly shown. Some of the strongest evidence for NREM mentation is the association of specific NREM contents with preawakening stimuli (Pivik 1991), for example, sleep talking (Arkin et al. 1970; Rechtschaffen et al. 1962) and experimental auditory and somatic stimuli (Foulkes & Rechtschaffen 1964; Lasaga & Lasaga 1973; Rechtschaffen et al. 1963b) that are concordant with NREM mentation. Similarly, presleep hypnotic suggestions often appear in mentation from all stages of sleep (Stoyva 1961).

An illustration of such incorporative "tagging" in NREM mentation is a report (Rechtschaffen et al. 1963a) of a subject who was stimulated during stage 2 sleep with a 500 Hz tone (7 sec) followed by a pause (27 sec), a second tone (7 sec), and then awakened 32 sec later:

> a little whistling tone was going on . . . and then it went off. And (the other person) said 'Oh, you had better get things over with quickly, because you may have to wake up soon' . . . I just said 'Oh!' to this, and I think I heard the whistling noise again. Then the same scene was there for some time, and I was just walking around trying to think of what was going on. (p. 412)

Some NREM parasomnias also demonstrate vivid mental experiences outside of REM sleep (Fisher et al. 1970; Kahn et al. 1991); sleep terrors arising from stage 3 and 4 sleep often result in reports of dramatic and frightening content. For some awakenings the content may be due to

the arousal itself (Broughton 1968), for others there is some sign of a progression seeming to lead up to, and possibly to induce, the awakening. Fisher et al. also found stage 2 nightmares qualitatively similar to those from REM sleep.

1.2.2.1. Sleep Onset (SO). Perhaps the most vivid NREM mentation reports have been collected from SO stages. These include images from the Rechtschaffen and Kales stages 1 and 2 of sleep (Cicogna et al. 1991; Foulkes & Vogel 1965; Foulkes et al. 1966; Lehmann et al. 1995; Vogel 1991) as well as from the stages of a more detailed SO scoring grid (Hori et al. 1994; Nielsen et al. 1995). SO mentation is remarkable because it can equal or surpass in frequency and length mentation from REM sleep (Foulkes 1982b; Foulkes & Vogel 1965; Foulkes et al. 1966; Vogel 1978b; Vogel et al. 1966). Moreover, much SO mentation (from 31–76% depending upon EEG features) is clearly hallucinatory dreaming as opposed to isolated scenes, flashes or nonhallucinated images (Vogel 1978b).

1.2.2.2. NREM sleep. Many more studies of sleep mentation have concentrated on NREM stages of sleep other than those of SO. Although in many studies stages 2, 3, and 4 are indiscriminately combined, stage 2 sleep is by far the most frequently examined stage.

To summarize this literature, studies of REM and NREM mentation published since 1953 were consulted. Of these, 35 studies[1] were retained for the calculation of global estimates of mentation recall (Fig. 2). Excluded were studies of patients for whom an illness (e.g., depression, anorexia) may have affected mentation recall. To equally weight findings from all studies, only one estimate of recall from each study was included in the global average. If a study contained values for different subgroups (e.g., young vs. old, male vs. female), an average of the groups was taken. Estimates were also calculated separately for studies prior to Foulkes's (1962) work, which was the first to highlight the distinction between *dreaming* and *cognitive activity* (Table 1).

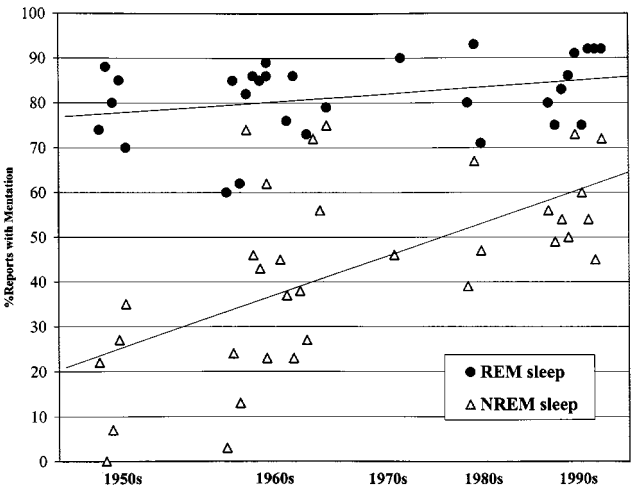

Figure 2. Summary of 35 studies of mentation recall from REM and NREM sleep over five decades. The percent of verbal reports that yielded some form of cognitive content after awakenings from NREM sleep increased from the 1950s to the 1990s, whereas the comparable percentage from REM sleep awakenings remained relatively constant. This difference is likely due to the widespread implementation in the 1960s of more liberal criteria for accepting reports as containing "cognitive activity" as opposed to simply "dreaming."

The overall difference in mean recall from REM (81.9 ± 9.0%) and NREM sleep (43.0 ± 20.8%) is close to 39%. However, this difference is much larger for the pre-1962 studies (i.e., 57.6%) than it is for the post-1962 studies (33.2%). Differences in median recall parallel those for the mean; total: 40%, pre-1962: 59%, post-1962: 37%. The present estimated NREM recall mean of 43.0% is very similar to that of 45.9% (± 15.8%) calculated from nine previous studies (Foulkes 1967). The present REM recall estimate of 81.9% also compares favorably with both (1) an estimate of 83.3% from over 200 subjects and 2,000 REM sleep awakenings (Dement 1965) and (2) an average of 81.7 ± 15.0% from 12 prior studies (Herman et al. 1978).

1.2.2.3. Stages 3 and 4 sleep. Some studies have found cognitive activity in stages 3 and 4 sleep (Armitage 1980; Armitage et al. 1992; Cavallero et al. 1992; Goodenough et al. 1965a; Herman et al. 1978; Pivik & Foulkes 1968). On average, recall from these stages is equal to that of stage 2 sleep; a tally of eight studies (Cavallero et al. 1992; Fein et al. 1985; Foulkes 1966; Lloyd & Cartwright 1995; Moffitt et al. 1982; Pivik 1971; Pivik & Foulkes 1968; Rotenberg 1993b) revealed an average recall rate of 52.5 ± 18.6%. The average stage REM recall rate in these studies was 82.2 ± 8.1%. The values for stages 3 and 4 are consistent with the finding that stage 2 and 4 mentation differences disappear for awakenings conducted at similar times of the night (Tracy & Tracy 1973). Three studies (Moffitt et al. 1982; Pivik 1971; Pivik & Foulkes 1968) found average recall rates to be higher in stage 3 (M = 56%) than in stage 4 sleep (M = 38%), a finding also true of children 9–11 years (42% vs. 26%) and 11–13 years (42 vs. 25%) (Foulkes 1982b). However, Pivik (1971) found nearly identical levels of recall of cognitive activity in stages 3 (41–56%) and 4 (38–58%).

Some subjects appear to have little or no recall of stage 3 and 4 sleep mentation. Ten of 60 subjects (17%) in one study (Cavallero et al. 1992) reported *no* mentation whatsoever after several nights of one awakening/night from stages 3 or 4 sleep; an additional 20 subjects (33%) required from one to five additional nights before recalling at least one instance of cognitive activity. These discrepancies have never been explained satisfactorily.

1.3. Summary

Numerous studies have replicated the finding of mentation outside of REM sleep as the latter is traditionally defined. All NREM sleep stages can produce some form of mentation. However, in accordance with the distinction between *dreaming* and *cognitive activity* discussed earlier, the more recent (post-1962) studies together indicate that about half of all NREM awakenings result in *no recall* of cognitive activity whatsoever. Further, about 50% of subjects appear to have noticeably degraded recall of mentation from NREM sleep, some (e.g., 17% of subjects in the Cavallero et al. 1992 study) have *no* recall after repeated awakenings. Further, because dreaming is a subset of cognitive activity, less than 50% of NREM awakenings produce dreaming. One liberal estimate is that only 25–50% of NREM reports bearing cognitive activity fulfill a minimal definition of dreaming (Foulkes 1962). Thus, at most 25%, but possibly

Table 1. *Summary of 35 studies of mentation recall from REM and NREM sleep (pre-1962 vs. post-1962)*

	N studies	Mean ± SD%	Median%	Range%
REM SLEEP RECALL				
<1962	8	76.0 ± 11.5	77	60–92
≥1962	21	84.1 ± 6.7	86	71–93
TOTAL	29	81.9 ± 9.0	85	60–93
NREM SLEEP RECALL				
<1962	8	18.4 ± 15.4	18	0–43
≥1962	25	50.9 ± 15.5	49	23–75
TOTAL	33	43.0 ± 20.8	45	0–75
REM/NREM SLEEP RECALL DIFFERENCES				
<1962	8	57.6	59	60–49
≥1962	21	33.2	37	48–18
TOTAL	29	38.9	40	60–18

Recall of mentation from REM sleep has been consistently high in studies conducted from the 1950s to the present, whereas recall from NREM sleep has increased on average. This increase reflects liberalization (first operationalized by Foulkes in 1962) of the criteria for accepting a mentation report as a valid object of study: this marked the shift from studing the more delimited category of "dreaming" to studying the wider category of "cognitive activity."

as little as 12% of NREM awakenings in *susceptible* subjects will produce reports of dreaming. The more elaborate forms of ("apex") dreaming are even less prevalent. It has been suggested (Herman et al. 1978) that vivid dreaming may occupy only 7% of recalled NREM mentation.

2. Experimental results bearing on the models

Resolving whether REM and NREM sleep mentation differ qualitatively is complicated by the thorny issue of whether the evaluation of sleep mentation conforms to commonly accepted psychometric principles of hypothetical construct validation, especially as these principles apply to psychophysiological studies. The validation of a hypothetical construct requires *several* criterion measures:

> It is ordinarily necessary to evaluate construct validity by integrating evidence from many different sources. The problem . . . becomes especially acute in the clinical field since for many of the constructs dealt with it is not a question of finding an imperfect criterion but of finding any criterion at all. (Cronbach & Meehl 1955, p. 285)

Further, the criterion measures under consideration should be as methodologically distinct from one another as possible to avoid "method artifact," that is, artifactual correlations among measures due to similarities in method (Strube 1990). Thus, solving the problem of qualitative differences in REM and NREM sleep mentation may require a construct validation approach sensitive to a wide range of methodologically diverse measures with probable or possible associations to sleep mentation. This is the principal justification for examining a variety of research methods in the following review.

How should a variable's "probable or possible associations" to sleep mentation be decided? Clearly, one's theoretical model is a determinant. Hobson's 2-gen model stipulates psychophysiological isomorphism; thus, the fact that REM and NREM sleep differ physiologically warrants investigation of physiological variables in relation to sleep mentation (Hobson & Stickgold 1995). Some proponents of the 1-gen model, on the other hand (Foulkes 1990), contend that mentation is psychologically driven. Physiological variables should be *excluded* from consideration. This assumption is supported by evidence that relationships between physiological variables and dream content have not been clearly demonstrated (see Pivik 1978; 1994; Rechtschaffen 1978, for reviews). However, as explained below, this assumption may not be completely justified on scientific grounds. To meaningfully compare the 1-gen and 2-gen points of view, a wide array of variables – including physiological variables – should be considered.

Foremost among the reasons for a lack of evidence for brain-mind relationships (Cacioppo & Tassinary 1990) may be the particular form of psychophysiological isomorphism proposed. One-to-one correspondences between a physiological (θ) and a psychological (ψ) variable, such as those proposed by the 2-gen model, are not, in fact, common in the literature; more commonly, multiple θ responses accompany a ψ variable or vice versa (Cacioppo & Tassinary 1990). To illustrate, EMG activity in the smiling muscle zygomaticus is associated with *both* positive dreamed affect and dreamed communication (Gerne & Strauch 1985). This problem can be resolved by evaluating a ψ variable in relation to an appropriate *group* of θ measures ("spatial response profiles") or in relation to a combination of such spatial groups *over time* ("temporal response profiles"). Also grouping ψ variables can give even greater specificity. Such procedures are rarely attempted for sleep mentation studies in part because of a lack of computing tools, but also because of a dearth of theoretical frameworks for such work.

Another criterion for accepting a variable as a "probable or possible" correlate of sleep mentation concerns its existing status as a correlate of a waking state mental process. With much research demonstrating sleep mentation to be *continuous* with waking state experiences (see Schwartz et al. 1978, for review), it is reasonable to expect that physiological indicators of waking state experiences should also be valid during sleep. Such cross-state generalization of a measure's validity is, in fact, implicitly accepted whenever a measure (e.g., P300) that has been validated in one waking state (e.g., attentiveness) is applied during a different waking state (e.g., emotional arousal).

In summary, resolution of the debate about REM and

NREM mentation is partly a problem of construct validation of the object of study. The debate was long ago widened to include *cognitive activity* as well as *dreaming* as dependent variables, and many pre-conscious *cognitive processes* may also belong in this category. It thus seems only fitting that a variety of process measures should be explored as potential markers of these objects of study. These measures should be methodologically diverse and have at least face validity as possible or probable correlates of the dependent measure. Thus, measures of cognitive content as well as accompanying physiological activity should be considered. In the review that follows, the measures considered are, for the most part, methodologically diverse and correlated with waking state cognitive processes. Even so, none involves the complex physiological profiles described earlier. Of the nine types of research examined, three (sects. 2.4, 2.6, 2.8) are closely tied to phenomenological features of sleep mentation. The others concern either physiological measures (sects. 2.3, 2.9), behavioral measures (sects. 2.1, 2.2, 2.5) or individual difference measures (sect. 2.7) that are presumed to index some critical aspect of cognitive activity during sleep mentation generation.

2.1. Memory sources inferred from associations to mentation

A 1-gen model might be expected to predict that REM and NREM reports of equivalent length derive from memory sources of equivalent type. This was supported in a study that used subjects' associations to dreams as a measure of their memory sources (Cavallero et al. 1990). Without controls for length, REM reports more frequently than NREM reports led to identifications of semantic knowledge sources, as opposed to autobiographical episodes or abstract self-references; with such controls – temporal unit weighting in this case – no memory source differences were found.

However, the 1-gen model is more often construed to be consistent with studies that *do* report qualitative differences in memory sources as a function of sleep stage. Comparisons of REM and NREM mentation reports do reveal differences in memory sources (Battaglia et al. 1987; Cavallero 1993; Cavallero et al. 1988; 1990; Cicogna et al. 1986; 1991; Foulkes et al. 1989). Compared with REM sleep mentation, memory sources of stage 2 mentation are more often episodic and less often semantic (see Cavallero 1993, for review) and more evidently connected to dream content (Foulkes et al. 1989). The memory sources of SO (1) are predominantly autobiographical and episodic (rather than an even mix of episodic memories, abstract self-references, and semantic knowledge as in REM sleep; Cavallero et al. 1988; 1990; Cicogna et al. 1986; 1991) and (2) more often have episodic sources referring to day residues than to earlier memories (as for REM sleep; Battaglia et al. 1987). Such results are taken to support the contention that "access to memory material is selective in SO, but probably undifferentiated in REM" (Cavallero & Cicogna 1993, p. 51).

2.1.1. Problems with memory source experiments. There are concerns with the notion that *diffuse mnemonic activation* is a precursor to sleep mentation (see sect. 2.9.1), because there are yet no valid correlates of such activation. Equally important is the question of whether memory activation should be considered to be distinct from the production of sleep mentation. If diffuse activation is dedicated exclusively to the production of sleep mentation and is tightly and reciprocally coupled to this production, then might it not better be conceptualized as an integral, inseparable component of it? If so, qualitative differences in memory sources are in fact qualitative differences in mentation production processes.

Other explanations have been offered for some REM/NREM sleep mentation differences, for example, more frequent episodic memory sources for SO reports because of recency effects or a "carry-over" of episodic processes from immediately preceding wakefulness (Natale & Battaglia 1990). This reasoning is consistent with "carry-over" effects following awakenings from REM and NREM sleep as discussed under post-awakening testing (sect. 2.5); however, most of the latter research demonstrates differences for REM and NREM sleep, that is, supports a 2-gen model.

Qualitative differences in memory sources may be due to differential levels of engagement of the dream generation system, but few empirical findings speak directly to this issue. Some authors (Cavallero & Cicogna 1993) link changes in "levels of engagement" to levels of cortical activation, but cannot easily reconcile this explanation with the qualitative *differences* in physiological activation characterizing REM and NREM sleep. Others (Foulkes 1985) eschew links between psychological and physiological activation altogether.

2.2. Memory consolidation

Memory processes are central to both 1-gen and 2-gen models of mentation production. Of the several paradigms that have been used to investigate learning and memory consolidation during sleep, most have produced results consistent with the notion of different forms of cognitive processing during REM and NREM sleep (see Dujardin et al. 1990; McGrath & Cohen 1978; Smith 1995, for reviews). Although the evidence is not unanimous, most suggests that REM sleep is selectively implicated in learning new information.

Some studies have found discriminative responding during REM but not NREM sleep (Hars & Hennevin 1987; Ikeda & Morotomi 1997) or establishment of a classically conditioned response (e.g., hippocampal activity) selectively during REM sleep (Maho & Bloch 1992). Discriminatory cueing during REM sleep even enhances performance on a previously learned skill, whereas cueing during NREM sleep impairs it (Hars & Hennevin 1987). Smith and Weeden (1990) found that stimulation with 70 dB clicks that were previously paired with a learning task enhances later performance only when similar clicks are administered during REM, but not NREM, sleep. Further, stimulation of reticular formation only during REM sleep improves learning over 6 days (Hennevin et al. 1989); such stimulation enhances awake learning if applied after either training or cueing treatment (see Hennevin et al. 1995b, for review).

On the other hand, a few studies have demonstrated transfer of discriminative responding during NREM sleep (Beh & Barratt 1965; McDonald et al. 1975), for example, a second-order conditioned response can be entrenched during either REM or NREM sleep (Hennevin & Hars 1992).

Several types of perceptual, cognitive, and memory skills have been examined in relation to REM and NREM sleep using different types of procedures: selective REM/NREM deprivation, changes in REM/NREM sleep architecture after learning, retrospective assessment of sleep architecture differences in slow versus fast learners, and perfor-

mance differences after REM and NREM awakenings. Much of this research suggests *qualitative differences in the tasks that are dependent upon the integrity of REM and NREM sleep.* Some illustrative findings:

1. Disruption of REM, but not NREM, sleep diminishes performance on a basic visual discrimination task (Karni et al. 1994).

2. Deprivation of REM, but not NREM, sleep diminishes performance on procedural or implicit memory tasks, that is, Tower of Hanoi, Corsi block tapping, but not declarative or explicit memory tasks, that is, word recognition, paired associates (Smith 1995).

3. Training animals on a new, appetitive or aversive task is followed by an increase in REM, but not NREM, sleep (Hennevin et al. 1995b).

4. Successful intensive language learning is accompanied by increased %REM, but not %NREM (De Koninck et al. 1989).

5. Rearing in an enriched environment produces more dramatic increases in REM than in NREM sleep (Smith 1985).

6. Waking recall of stimuli presented during sleep is superior for stimuli presented just before awakenings from REM, but not NREM, sleep (Shimizu et al. 1977).

NREM sleep is associated with memory tasks only rarely; NREM sleep deprivation disrupts Rotor pursuit (Smith & MacNeill 1994) and the learning of lists of word pairs (Plihal & Born 1997). These findings nevertheless point to skills that are qualitatively different from those typically associated with REM sleep and are thus consistent with a 2-gen model.

2.2.1. Problems with memory consolidation experiments. It remains unknown whether the memory processes essential to generating sleep mentation are the same as those shown to be associated with REM and NREM sleep. Almost invariably subjects in these types of experiments are never awakened to sample mentation in relation to learning. Some exceptions (Conduit & Coleman 1998; De Koninck et al. 1988; Fiss et al. 1977) unfortunately have not examined *both* REM and NREM sleep mentation to compare the two.

2.3. Event-related potentials

Different time-locked components of event-related potentials (ERPs) reflect different steps of perceptual and cognitive processing, steps that may be extrapolated to some extent to the various stages of sleep (see Kutas 1990; Salisbury 1994, for reviews). Short-latency auditory components – occurring within 10 to 15 msec of a stimulus – reflect sensory pathway integrity from receptors through to thalamus, and appear not to change in any sleep stage (Campbell & Bartoli 1986). Middle latency responses – 10 to 100 msec post-stimulation – reflect processes such as threshold detection associated with medial geniculate, polysensory thalamus, and primary cortex. Up to 40 msec, these components are largely unaffected by sleep/wake stage (Salisbury 1994). Beyond 40 msec, most studies show some reduction in amplitude and latency during sleep (Erwin & Buchwald 1986; Linden et al. 1985; Picton et al. 1974) although some show an increase in amplitude of potentials such as N1 and P2 (Nordby et al. 1996). These changes vary little from stage to stage, however. Long-latency components – typically later than 100 msec post-stimulation – are of particular interest because of their putative associations with cognitive processes such as selective attention (N1 or N100), sensory mismatch (N2-P3a), orienting (N2), surprise (P3b), novelty (P3a), and semantic processing (N400) (see Kutas 1990; Salisbury 1994, for reviews). Several studies (Addy et al. 1989; Nakano et al. 1995; Noguchi et al. 1995; Nordby et al. 1996; Roschke et al. 1996; Van Sweden et al. 1994) indicate that long-latency components from NREM sleep (vs. those from wakefulness), are both suppressed in amplitude and slowed in latency – independent of the sensory modality stimulated. Most studies find that these components in REM sleep resemble those of wakefulness to a greater extent than they do the more diminished potentials of NREM sleep.

Research pertinent to the critical question of whether P300, a presumed measure of complex cognitive processing, is differentially active during REM and NREM sleep has produced mixed results. Most studies find P300 in REM sleep and stage 1 NREM sleep but not in other NREM sleep stages (Bastuji et al. 1995; Côté & Campbell 1998; Niiyama et al. 1994; Roschke et al. 1996; Van Sweden et al. 1994) suggesting a distinctive mode of higher-order processing during the two sleep states with the most vivid imagery processes. Others have found either a diminished P300 in both REM and NREM sleep (Wesensten & Badia 1988) or no clear evidence of P300 in sleep (Nordby et al. 1996). These discrepant findings may be due, in part, to the large variability of this late component, a variability exacerbated in NREM sleep by the superimposition of endogenous K-complexes, as well as by the fact that oddball stimuli are often not sufficiently disparate (Salisbury 1994) or intense (Côté & Campbell 1998) to evoke the P300 response.

Both 1-gen and 2-gen models stipulate that the blocking of afferent information during sleep is a precondition for cognitive activity. Thus, early- and middle-latency results seem relatively irrelevant to differentiating the models. To the extent that higher-order cognitive functions are necessary for sleep mentation, long-latency ERP studies demonstrating degradation of these components in NREM, but not REM, sleep support the notion of *different* cognitive processes in the two states.

2.3.1. Problems with ERP studies. It might be argued (from the 1-gen viewpoint) that long-latency ERP differences reflect only differences in degree – not quality – of mentation production processes in REM and NREM sleep. Diminished P300 amplitude in NREM sleep might simply index a reduction in memory diffuseness thought to occur (Foulkes & Schmidt 1983). This argument hinges in part on what transformations of the P300 waveform are ultimately found to be correlated with qualitative (and not simply quantitative) differences in REM and NREM mentation. One might expect that minor changes in amplitude or latency reflect only quantitative differences while more dramatic changes in ERP structure (e.g., absence of the waveform) reflect qualitative differences, but this remains an empirical question.

It might also be argued (from the 1-gen viewpoint) that the cognitive processing revealed by long-latency components does not reflect activity that is germane to mentation production. Such components may reflect processing occurring either so early or so late in production that they have no causal bearing on the outcome. Processes such as sensory mismatch recognition, or orienting/surprise to a

stimulus could be simple affective *reactions* to unusual dreamed events, reactions with no real impact on imagery construction (Foulkes 1982c). Conversely, at least one well-articulated theory describes how orienting responses and related affective reactions *engender* sleep mentation (Kuiken & Sikora 1993). Moreover, many findings link P300 to emotional processes such as mood expectancy during reading (Chung et al. 1996) emotional prosody (Erwin et al. 1991) and emotional deficits (Bungener et al. 1996). On the other hand, the suggestion (Donchin et al. 1984) that P300 reflects processes of *creating, maintaining, and updating an internal model of the immediate environment* suggests that P300 underlies more basic representational processes.

2.4. Stimulation paradigms

The presentation of stimuli prior to sleep affects REM and NREM sleep mentation differentially, for example: (1) six hours of cognitive effort prior to sleep produces REM sleep mentation with less thinking and problem solving, and NREM sleep mentation with increased tension (Hauri 1970); (2) presentation of presleep rebus stimuli (e.g., image of a *pen* with a *knee* → penny association) has no effect on REM sleep mentation, but evokes conceptual references to the stimulus words (e.g., pencil, leg) in stage 2 mentation (Castaldo & Shevrin 1970); (3) auditory cues to picture learning leads to superior processing of higher order stimuli in stage 2 (Tilley 1979). These authors conclude that REM and NREM sleep are associated with different levels of cognitive organization – which squares with the notion that NREM sleep mentation is more conceptual or thoughtlike. However, auditory cues are also less impeded by sensory inhibition during stage 2 sleep than during phasic REM sleep (Price & Kremen 1980). On the other hand, superior processing of verbal materials during REM sleep was suggested in a study of associative learning (Evans 1972); such differences are not easily explained by elevated sensory inhibition during REM sleep.

2.4.1. Problems with stimulation paradigms. Many of these studies suggest sleep stage differences that are *opposite* in nature to those suggested by ERP studies, for example verbal stimulation preferentially influences stage 2 mentation, whereas REM sleep has more evident late ERP components of the type one might expect to index the registration of such verbal stimulation. Such ambiguities could be resolved by examining both sleep mentation and ERPs in the same study design.

2.5. Post-awakening testing

Post-awakening testing taps cognitive abilities immediately after awakening from REM or NREM sleep, and is based on the observation that cognitive and physiological components of a sleep state will "carry-over" and influence waking performance. Post-awakening testing has been used by at least six independent research groups in at least eight different studies (see Reinsel & Antrobus 1992, for review). Most studies concur that REM and NREM sleep awakenings produce different patterns of responding. The first demonstration of a "carry-over effect" (Fiss et al. 1966) was that thematic apperception test (TAT) stories generated following REM sleep awakenings were more "dreamlike" than those following NREM sleep. Subsequently, perceptual illusions, such as spiral after-effect and beta movement, were found to vary with preceding sleep stage (Lavie 1974a; Lavie & Giora 1973; Lavie & Sutter 1975). Superior performance on right hemisphere (RH), primarily spatial tasks after REM sleep and on left hemisphere (LH), primarily verbal tasks after NREM sleep were also reported (Gordon et al. 1982; Lavie & Tzischinsky 1984; Lavie et al. 1984). Other studies (Bertini et al. 1982; 1984; Violani et al. 1983) demonstrated RH superiorities after REM sleep on a tactile matching task. Short-term memory is also better after REM versus NREM awakenings (Stones 1977).

One study (Reinsel & Antrobus 1992) did not replicate the reported stage differences, even though many of the same dependent measures were employed. The authors suggest that the discrepancies may be due to subtle methodological differences, for example, greater memory demands in the original studies (Reinsel & Antrobus 1992). Also, stage-related differences on trail-making and vigilance tasks were not found for REM and NREM awakenings (Koulack & Schultz 1974).

Most of these results support the interpretation that qualitatively different cognitive processes are active following and, by inference, just preceding awakenings from REM and NREM sleep. These include both lower-level (perceptual registration, stimulus matching) and higher-level (short-term memory, story generation) processes.

2.5.1. Problems with post-awakening testing. The replicability of post-awakening effects was questioned by at least one study (Reinsel & Antrobus 1992). There is also some concern about whether waking state measures are valid measures of preceding, sleep-related processes. Findings do support the "carry-over" construct, but the weight of evidence is not overwhelming. It is possible, for example, that post-awakening effects are due to different *changes of state* as opposed to "carry-over" of cognitive processes linked to a particular state.

2.6. Inter-relationships between mentation contents from different reports

The 1-gen model might predict that a single imagery generator would produce a great degree of thematic continuity between proximal REM and NREM reports within a night; the 2-gen model would predict different kinds of unrelated mentation. One study (Cipolli et al. 1988) supporting the 1-gen model found that low-level paradigmatic and lexical relationships (but not high-level syntagmatic and propositional relationships) between pairs of mentation reports were higher within the same night than they were between nights, regardless of whether the reports were REM-NREM pairs or REM-REM pairs. An earlier study (Rechtschaffen et al. 1963b) found that high-level themes were often repeated in REM and NREM reports from the same night.

2.6.1. Problems with report inter-relationships. If thematic similarity is an index of unified mentation production, then thematic difference may be construed as an index of two or more generators. In all likelihood, thematic differences would be more prevalent than similarities in any within-night REM/NREM mentation comparisons. Yet chance levels of thematic similarity in adjacent reports remain unknown. It may also be argued (from a 2-gen perspective) that similar themes nevertheless differ in some qualitative respects, for example, an interpersonal aggres-

sion may be more self-participatory, affectively engaging, and visual in a REM report than in a NREM report (cf Weinstein et al. 1991).

2.7. Subject differences in mentation content

Interactions between subject differences and stage-related cognitive activity may set limiting conditions on the generalizability of the two models, for example, they may suggest that one or the other model *is valid only for some types of subjects and under some circumstances.* Also, some prevalent subject variables linked to sleep mentation (e.g., age, insomnia, dream recall frequency) may determine subject self-selection for sleep studies and thus bias the estimated rates of mentation recall from REM and NREM sleep. Three variables illustrate this complexity.

2.7.1. Light versus heavy sleepers. Zimmerman (1970) first proposed that differences in *activation* may account for REM/NREM mentation differences. He classified subjects as either light or deep sleepers (based on auditory arousal thresholds) and awakened them twice each from REM and NREM sleep. Light sleepers reported *dreaming* after NREM awakenings more often (71%) than did deep sleepers (21%). REM and NREM mentation from these groups also differed qualitatively. For deep sleepers, NREM mentation was less perceptual, controlled, and distorted. For light sleepers, such differences did not obtain. If light-sleeping subjects are more cerebrally *aroused* than are deep-sleeping subjects during NREM sleep, then their NREM content may be much more REM-like. Thus, the 1-gen model may apply to light-sleeping subjects; the 2-gen model to deep-sleeping subjects.

2.7.2. Habitual recall of dream content. Mentation from REM and NREM sleep differs for subjects high and low in habitual dream recall. We (Nielsen et al. 1983; 2001) found that stage REM reports were higher on two measures of story organization (number of story constituents, degree of episodic progression) than were NREM reports, *but only for high frequency recallers.* The 1-gen and 2-gen models appear to describe low- and high-frequency recallers differentially.

2.7.3. Psychopathology. Measures of REM and NREM salience (i.e., recall and length) are correlated differentially with measures of psychopathology. For example, the MMPI L scale correlates with REM mentation recall whereas no scales correlate with NREM mentation recall (Foulkes & Rechtschaffen 1964). The two states are further differentiated by correlations between the MMPI Hy scale and REM word count and between several scales and NREM word count. NREM word count also correlates with Ego Strength and Hostility Control. A 2-gen model is favored by such results.

2.7.4. Other studies of subject variables. Many other subject variables are known to interact with sleep mentation although specific relationships remain to be clarified. Some include (1) the differential association of age with late night activation effects on REM and NREM mentation (Waterman et al. 1993), (2) large differences in recall of REM (but not NREM) related mentation for both insomniac (Rotenberg 1993b) and depressed (Riemann et al. 1990) patients versus normal controls, (3) the effects of introspective style on the salience of REM and NREM content (Weinstein et al. 1991) and elevated incorporation of laboratory characters into REM (but not NREM) mentation for women, but not men (Nielsen et al. 1999). Other such correlates of dream recall have been reviewed (Schredl & Montasser 1997) and appear to be consistent primarily with the 2-gen model.

2.8. Residual differences in stage-related measures of mentation quality

Many authors feel that the fairest test of REM/NREM mentation differences is whether mentation reports differ on qualitative measures after report length has been controlled. However, many studies report qualitative REM-NREM stage differences even with such controls (Antrobus 1983; Antrobus et al. 1995; Cavallero et al. 1990; Cicogna et al. 1991; Foulkes & Schmidt 1983; Hunt et al. 1993; Porte & Hobson 1996; Nielsen et al. 1983). With length controls, REM and NREM mentation samples still differ on self-reflectiveness (Purcell et al. 1986), bizarreness (Casagrande et al. 1996b; Porte & Hobson 1986), visual and verbal imagery (Antrobus et al. 1995; Casagrande et al. 1996b; Waterman et al. 1993), psycholinguistic structure (Casagrande et al. 1996a), and narrative linkage (Nielsen et al. 1983). Strauch and Meier (1996) found fewer characters and lower self-involvement in NREM than in REM mentation, again, regardless of report length. Even Foulkes (Foulkes & Schmidt 1983) found more per-unit self-representation in REM than in SO mentation and more per-unit characterization in REM than in NREM mentation. Differences in characterization and self-representation are not trivial since they are two of the most ubiquitous constituents of dreaming.

Visual imagery is perhaps the most defining quality of dream mentation. Visual imagery word count and total word count both differentiate stage REM from stage 2 mentation reports – and a *significant predominance of visual words in REM over NREM reports remains even after total word count is controlled as a covariate* (Waterman et al. 1993). Antrobus et al. (1995) have replicated this finding, failing to replicate Antrobus's own earlier study (Antrobus 1983), as have Casagrande et al. (1996b).

A recent study (Porte & Hobson 1996) reports stage-related differences in fictive (imagined) movement, but also some support for the 1-gen model. Here, the subgroup of 10 subjects who produced the only motor reports in NREM sleep also had the longest mentation reports from both sleep stages. The authors suggest that some factor may have caused their NREM sleep to be influenced by REM sleep processes, for example, an increase in REM sleep "pressure" by REM deprivation, thus lengthening REM reports and raising the odds that a NREM awakening coincides with a pre-REM or post-REM sleep transitional window (Porte & Hobson 1996). I refer to this window as a type of *covert REM sleep* in a later section (see sect. 3).

The accumulation of findings of residual qualitative differences between REM and NREM sleep mentation after length control challenges the 1-gen argument that such controls cause qualitative differences to disappear (Foulkes & Cavallero 1993). Such differences are diminished by controlling length but they are not eliminated altogether.

2.9. Memory versus physiological "activation"

2.9.1. Are memory activation and cortical activation isomorphic? Foulkes's (1985) 1-gen model identifies memory

activation as the instigating force of sleep mentation but excludes physiological activation as a determinant, even though known relationships between cerebral activation and sleep/wake stages might seem consistent with the model. For example, PET imaging studies of the brain have demonstrated that REM sleep is characterized by elevated and more widespread activation than is NREM sleep; higher levels of cerebral blood flow have been measured in most centrencephalic regions (cerebellum, brainstem, thalamus, basal ganglia, basal forebrain), limbic and paralimbic regions (hippocampus, temporal pole, anterior insula, anterior cingulate), and unimodal sensory areas (visual and auditory association; Braun et al. 1997). Note, however, that Foulkes's exclusion of neurophysiological correlates of brain activation in the development of 1-gen models is not supported by all 1-gen theorists.

Studies of whether cortical activation is indeed correlated with cognitive activation offer limited support for the notion of an association (see Antrobus 1991, for review). With EEG slowing and increased voltage there is an associated *decrease* in mentation recall (Pivik & Foulkes 1968, and there is more EEG slowing in NREM than in REM sleep (e.g., Dumermuth et al. 1983). In one study, both delta and beta amplitude predicted successful dream recall from REM sleep whether subjects were depressed or healthy (Rochlen et al. 1998). In our studies (Germain et al. 1999; Germain & Nielsen 1999) fast- and slow-frequency power was associated with recall of dreams from REM and NREM sleep respectively. If EEG-defined activation (delta) is statistically controlled, stage differences in mentation are still obtained (Waterman et al. 1993). At least one study (Wollman & Antrobus 1987) found *no* relationships between EEG power and word count of either REM sleep reports or waking imagery reports.

It is well known that both the recall (Goodenough 1978; Verdone 1965) and the salience (Cohen 1977a; Foulkes 1967) of sleep mentation increases in later REM episodes; these changes are likely due to activation associated with circadian factors (Antrobus et al. 1995). On the other hand, circadian factors appear to influence REM and NREM mentation equally (Waterman et al. 1993) – a finding that would seem to support the 1-gen model. However, when both stage and diurnal activation effects on variables such as visual clarity are assessed simultaneously, the effect size for time-of-night activation is only about 30% of the effect size for REM-NREM stage activation; this difference is interpreted to support the 2-gen, A-S model (Antrobus et al. 1995).

2.9.2. Partialling out activation: Problems with using report length. Controls for report length are effected in different ways. Most studies estimate activation by total word count (TWC; Antrobus 1983), a tally, usually transformed by $\log_{10}$(TWC+1) to remove positive skew, of all non-redundant, descriptive content words in the report. Length is then partialled out of correlations between variables or in some other way (Antrobus et al. 1995; Levin & Livingston 1991; Waterman et al. 1993; Wood et al. 1989). A procedure conceptually related to TWC is to weight dependent variables with a length estimate that is based upon report structure. Foulkes and Schmidt (1983) parsed reports for events that occurred contiguously, the so-called "temporal unit." Similarly, we (Nielsen et al. 1983; 2001) used the presence of story components (characters, actions, settings) to control for their organization – a REM/NREM difference was found in this study. We also used the proportional measures of the Hall and Van de Castle (1966) system to compare REM and NREM reports qualitatively – few REM/NREM differences were seen (Faucher et al. 1999).

Hunt's (1993) challenge to length-sensitive corrections is that variations in report length are an expected correlate of mentation that is qualitatively remarkable in some way, that is, that "more words are necessary to describe more bizarre experiences" (p. 181). To partial out report length from a given qualitative scale may be to partial out the variable from itself (p. 181) and may even "cripple our ability to study what is most distinctive about dreams by misleadingly diluting a key measure of the dreaming process" (p. 190). Even worse, using word frequencies to weight non-verbal variables (e.g., bizarreness) may arbitrarily transform findings and produce unpredictable and artificial effects (Hunt et al. 1993). Using report lengths and bizarreness ratings, Hunt demonstrated that a bizarre pictorial stimulus does indeed require more words to describe than does a mundane stimulus, and that the partialling out of TWC eliminates significant correlations between bizarreness and other measures. Weighting produced a significant loss of information related to the dependent variable.

2.10. Summary

Most of the research reviewed in the preceding nine categories tends to favor the 2-gen over the 1-gen model. The 2-gen model is supported particularly by evidence of REM/NREM differences in sleep mentation and by physiological measures, such as long-latency ERPs, that are valid correlates of waking cognitive processes. The principal claim of the 1-gen model, that qualitative differences are artifacts of quantitative differences, has been challenged by many studies demonstrating process differences and residual qualitative differences after length control, as well as studies questioning the assumptions underlying quantitative controls. Another argument, that residual qualitative differences are attributable to differences in memory inputs, has merit, but has not been supported by all attempts to quantify these inputs. There are also important questions about whether memory indeed functions in a diffuse manner as proposed, and whether memory source activation is not, in fact, an integral part of the dreaming process itself. Recent neuropsychological evidence favors the 1-gen model but has still not directly addressed the question of REM and NREM sleep mentation differences.

On the other hand, the evidence does not overwhelmingly support the 2-gen model either. Evidence for neurobiological isomorphism as currently defined is still slim, and leaves most of the conclusions of this model extremely speculative (Foulkes 1990; Labruzza 1978). The 2-gen model is also weak in describing the nature of REM and NREM mentation comparatively. As a model driven by physiological antecedents to cognition, it can also be criticized for not accounting for forebrain mechanisms that seem central to complex cognitive operations such as the narrative synthesis of dreaming (Antrobus 1990; Solms 1995; Vogel 1978a).

3. An alternative model: Covert REM sleep processes in NREM sleep

The literature presents an apparent paradox. On one hand, there is strong proof that cognitive activity – some of it

dreaming – can occur in all sleep stages. On the other hand, there is evidence that REM and NREM sleep mentation and an array of their behavioral and physiological correlates differ qualitatively. The former evidence supports a 1-gen model, the latter a 2-gen model. How may this seemingly contradictory evidence be reconciled?

One possible reconciliation is that sleep mentation is, in fact, tightly coupled to REM sleep processes, but that some of these processes under certain circumstances may dissociate from REM sleep and stimulate mentation in NREM sleep in a *covert* fashion. This alternative conceptualization maintains a 1-gen assumption but couples it with an assumption of psychophysiological isomorphism. The same (REM sleep-related) processes are thought to be responsible for sleep mentation regardless of stage, even though in NREM sleep these processes may be activated in a piecemeal fashion and against an atypical neurophysiological background. Some REM sleep processes would thus combine in as yet unspecified ways with NREM sleep processes to produce unique profiles of NREM sleep physiology and intermittent occurrences of REM-like sleep mentation. The origin of these mechanisms in REM sleep events may explain observed similarities in REM and NREM mentation reports, while their dissociated nature may explain apparent qualitative differences. This model is in some respects similar to the 1-gen model in that it assumes commonality of processes for all mentation reports, but it differs in that it extends this commonality to physiological processes. The model is also similar in some respects to the 2-gen model in that it assumes psychophysiological isomorphism between sleep mentation and some features of sleep neurophysiology and in that it explains qualitative differences in REM and NREM mentation as a function of the dissociated quality of covert activation (e.g., piecemeal activation, atypical neurophysiological background).

This view leads to several straightforward and easily testable predictions about mentation in relation to sleep stage: *(1) mentation recalled from NREM sleep will be associated with factors linked to preceding and/or subsequent REM sleep.* For example, recall of mentation should be more likely, more abundant or more salient from NREM episodes that are in close proximity to a REM sleep episode, or from NREM episodes that are in proximity to particularly long or intense REM episodes. The former example is supported by several studies reviewed earlier and is described in more detail in the probabilistic model that follows. The latter example has not been systematically investigated. The covert REM sleep model also predicts that *(2) recall of mentation from NREM sleep will be more probable under conditions likely to stimulate covert REM sleep, for example, sensory stimulation during sleep, sleep deprivation and fragmentation, sleep onset, arousal during sleep, psychiatric and sleep disorders, medications.* Evidence supporting the preceding hypotheses is reviewed in more detail below. Finally, the model's isomorphism assumption leads to some predictions about the neurophysiological characteristics of REM and NREM sleep with and without mentation recall: *(3) the neurophysiological characteristics of NREM sleep* with *recall of mentation will differ from those of NREM sleep* without *recall,* and *(4) the neurophysiological characteristics of NREM sleep with the most vivid mentation will resemble the characteristics of REM sleep with typical mentation.* The former prediction we have supported to some extent with evidence that EEG spectral analysis differentiates between NREM sleep awakenings with and without recall of mentation (Germain & Nielsen 1999). The latter prediction we have supported to some extent with evidence of similarities in the EEG accompanying NREM imagery from sleep onset and that accompanying imagery from REM sleep (Nielsen et al. 1995). However, both predictions require testing with more refined multivariate methods.

Covert REM sleep is defined here to be *any episode of NREM sleep for which some REM sleep processes are present, but for which REM sleep cannot be scored with standard criteria.* This notion encompasses previous ideas that have been raised and expanded upon to varying degrees by different authors, but has never been elaborated into a systematic model. The following is therefore a synthesis and systematization of several existing ideas about covert REM sleep as well as a review of research findings that support these ideas. In brief, evidence is reviewed supporting the notion that covert REM sleep processes can occur in NREM sleep under many different circumstances. An easily testable model is then proposed that addresses two of these conditions: covert REM sleep occurring during NREM/REM transitions and that occurring during SO.

3.1. Covert REM sleep is suggested by "intermediate sleep"

Lairy et al. (1967) were among the first to identify atypical mixtures of REM and NREM sleep in human subjects. Their notion of "intermediate sleep" was of sleep that typically arises between REM and NREM sleep episodes but that consists of elements of both. Intermediate sleep was defined primarily by EEG configurations containing both REM and NREM sleep features, such as spindles or K-complexes separated by episodes of "EEG traces identical to that of REM sleep" (p. 277). Mentation elicited from intermediate sleep was noted to be less hallucinatory and more negative in feeling tone than that elicited from REM sleep. Intermediate sleep could also at times replace an entire REM sleep episode. In normal subjects, it was said to occupy 1–7% of sleep; in psychiatric cases, such as psychosis, from 10 to over 40% (Lairy et al. 1967). More recent clinical evidence (Mahowald & Schenck 1992) confirms that components of different sleep/wake states do indeed dissociate and combine in atypical patterns as a consequence of illness or other unusual circumstances. For instance, the violent dream-related outbursts of REM sleep behavior disorder seems to combine features of wakefulness (motor activity) with background REM sleep (Mahowald & Schenck 1994) whereas the cataplexy attacks of narcolepsy appear to combine aspects of REM sleep (muscle atonia) with background wakefulness.

3.2. Physiological processes anticipate REM sleep onset

Some studies suggest that covert REM sleep processes can occur during normal human sleep. First, the REM sleep-related shift in HR variability from predominantly parasympathetic to predominantly sympathetic can occur up to 15 minutes prior to the EEG-defined onset of REM sleep (Scholz et al. 1997). Second, the progressive suppression of REM-related sweating effector activity – an index of thermoregulation – anticipates REM sleep onset by 6–8 min-

utes (Dewasmes et al. 1997; Henane et al. 1977; Sagot et al. 1987). Fluctuations in this measure have been proposed to be due to occurrences of dreaming (Dewasmes et al. 1997; Ogawa et al. 1967). Third, the REM sleep-associated cortical process of N300 amplitude attenuation occurs several minutes prior to other REM sleep indices such as muscle atonia and eye movements (Niiyama et al. 1998).

3.3. Covert REM sleep during "missing" REM episodes

Covert REM sleep processes may be implicated in the atypical NREM sleep episodes for which the absence of one or more electrophysiological criteria prevents a score of REM sleep from being assigned. To polysomnographers, these episodes commonly, but not exclusively, appear as the troublesome "missed" REM sleep episodes early in the night. Their absence can lead to exceptionally long REM SO latencies being scored. During such episodes, most of the electrophysiological signs of REM sleep are present – for example, cessation of spindling, EEG desynchronization, changes occurring approximately 90 minutes after SO – but sometimes chin muscle tonus may remain high, or rapid eye movements may be slow or indistinct, or a brief waking arousal may occur. Such stages may be scored as stage 1 or 2 even though intuition strongly suggests that REM sleep is somehow present.

Other studies have reported the omission of REM periods at other times of the night. Nocturnal penile tumescence, a relatively robust correlate of REM sleep (e.g., Karacan et al. 1972), often occurs at the 90-minute junctures where REM sleep might be expected but is not scored because of missing criteria (Karacan et al. 1979). In Karacan's study, 12 of 19 erections occurring during NREM sleep were related to expected but incomplete REM sleep episodes; an additional four occurred during NREM sleep immediately after REM sleep awakenings. Their paper contains an illustrative hypnogram of three consecutive nocturnal erections overlying three corresponding covert REM episodes.

3.4. Proximity of NREM sleep awakenings to REM sleep

Recordings of spontaneous REM and NREM sleep awakenings in the home setting reveal that NREM mentation reports are longest if they occur within 15 min of a prior REM sleep episode, whereas REM mentation reports are longest if they occur 30–45 minutes into a REM episode (Stickgold et al. 1994a). In fact, in this study seven of the nine longest NREM reports occurred within 15 minutes of a REM episode. These findings replicate an earlier finding (Gordon et al. 1982) that NREM reports occurring within 5 minutes of previous REMs more often produce cognitive activity (81.8%) than do reports occurring more than 10 minutes post-REMs (3.8%). They also replicate the finding (Antrobus et al. 1991) that NREM reports occurring 5 minutes after a REM sleep episode contain more words per report than do those occurring 15 minutes post-REM. Stickgold et al. interpret these kinds of results as possibly supporting a covert REM sleep influence, that is, that "long NREM reports reflect transitional periods when some aspects of REM physiology continue to exert an influence" (p. 25). They also consider that reports from early in NREM sleep episodes might reflect recall of mentation from the preceding REM episode, a notion that has often been suggested as an explanation for dreaming during NREM sleep (Kales et al. 1966; McCarley 1994; Wolpert & Trosman 1958; and see Porte & Hobson 1996 for discussion). It should be noted that at least one study (Kamiya 1962) has found that NREM awakenings conducted prior to the first REM sleep episode of the night, when presumably no prior REM sleep influences could have occurred, nevertheless produced recall of cognitive activity (43%). Similarly, a study (Foulkes 1967) in which awakenings 30 minutes post-REM targeted the *middle* of NREM episodes – also found a sizable recall rate of 64.6%. These recall rates either equal or exceed the mean recall rate estimate for NREM sleep presented earlier. Both studies argue against the possibility of covert REM sleep processes. However, the reconsideration of SO as a possible source of covert REM sleep to some extent counters the first of these arguments (see sect. 3.5), whereas the substantial uncertainty associated with identifying the precise middle of NREM episodes responds somewhat to the latter (see sect. 4.1 below). These arguments are now considered in more detail.

3.5. Covert REM sleep during sleep onset (SO)?

Covert REM sleep processes may manifest during SO episodes. These brief wake-sleep transitions display many of the electrophysiological signs of REM sleep, for example, transient EMG suppressions and phasic muscle twitches, as well as extremely vivid sleep mentation. We have shown that the topographic distributions of fast-frequency EEG power for SO images and REM sleep are similar (Nielsen et al. 1995). REMs are less conspicuous at SO, but they are nevertheless observed (Vogel 1978b). However, the slow eye movements so characteristic of SO also occur frequently in REM sleep, suggesting that they may constitute an unrecognized marker of REM sleep (Porte 1997). It is thus possible that the vivid dreaming of SO derives from a brief, usually undetected passage through REM into descending stage 2 sleep. The sleep onset REM (SOREM) episodes observed frequently in both sleep disordered and normal individuals (Bishop et al. 1996) may be instances of covert REM sleep transitions that have been "unmasked" and thus *do* manifest all of the inclusion criteria for REM sleep. Such unmasking might be influenced by the build-up of REM pressure. For example, we found that SOREM episodes on the MSLT were twice as frequent in sleepy patients (with severe sleep apnea or idiopathic hypersomnia) than they were in non-sleepy patients (with mild sleep apnea or periodic leg movements without hypersomnia) (T.A. Nielsen, J. Montplaisir & A. Gosselin, unpublished results). The fact that reports of dreaming during MSLT naps are *not* good predictors of the presence of classical REM sleep (Benbadis et al. 1995) may reflect the difficulty of differentiating covert REM sleep from REM sleep as it is classically defined. Further evidence for covert REM sleep processes at SO is the variety of sleep starts commonly observed at SO among healthy subjects. Such starts consist of abrupt motor jerks and sudden flashes of visual, auditory, and some esthetic imagery; it has been suggested that they are intrusions of isolated REM sleep events into NREM sleep (Mahowald & Rosen 1990).

3.6. Covert REM sleep: A disorder of arousal?

Mentation is often reported after sleep terror awakenings, which occur in NREM sleep stages 3 or 4 (Fisher et al.

1973). Much of this mentation appears to be induced by the arousal itself, judging by the themes such as death anxiety associated with tachycardia and choking anxiety associated with respiratory difficulty. Other instances appear to be ongoing before the terror erupts although they too appear to be heavily influenced by stimuli from the laboratory (Fisher et al. 1973). In fact, it is possible to induce terrors by external stimulation, such as sounding a buzzer. Thus, it is possible that sleep terror mentation is also a type of brief covert REM sleep event induced by stimulation that arises either internally (autonomic arousal) or from the laboratory environment (electrodes, noise, etc.) during arousals from sleep (see also sect. 3.11 below).

Early studies that examined method of arousal as a determinant of mentation content reported that, relative to abrupt awakenings, prolonged awakenings increase the frequency of thoughtlike mentation reports from both REM and NREM sleep (Goodenough et al. 1965a; Shapiro et al. 1963; 1965). This may mean that the prolonged awakenings induced a type of covert REM sleep state regardless of whether the ongoing state was REM or NREM sleep; the thoughtlike mentation accompanying this sleep state parallels that of what is most commonly reported after NREM awakenings. Physiological evidence that prolonged awakenings produce covert REM sleep is scanty although "stage-1" sleep with rapid eye movements during arousals from NREM sleep have been observed in individual subjects (Goodenough et al. 1965a; Roffwarg et al. 1962). Further, Goodenough et al. report many occasions on which gradual awakenings from NREM sleep are accompanied by a REM sleep-like EEG profile but no rapid eye movements.

3.7. Covert REM sleep underlies the REM sleep "efficiency" concept

Polysomnographers applying the Rechtschaffen and Kales criteria have always accepted a certain degree of ambiguity in their scoring of REM sleep, especially in the notion of REM sleep "efficiency." Within the limits of a given REM sleep episode there can occur transitions into other stages – typically stage 2 or wakefulness – which reduce the efficiency of the REM episode. If this alternate activity does not exceed 15 minutes in length, then the stage is considered a temporary deviation of an otherwise continuous REM sleep episode. If it exceeds 15 minutes, it denotes the start of a new REM/NREM cycle, with a periodicity far short of 90 minutes, that is no longer factored into the efficiency score. Thus, the 15-minute criterion for REM sleep efficiency implies that the underlying physiological state of REM sleep is not *completely* suspended during intrusions by another stage for $<$15 minutes. Some factor continues to exert a "propensity" to express REM sleep, a factor that seemingly remains latent. In view of research reviewed here (see sect. 3.2), the choice of 15 minutes for calculation of REM sleep efficiency seems entirely appropriate.

3.8. Covert REM sleep "pressure" is augmented by REM sleep deprivation

Selective REM sleep deprivation is known to increase "pressure" to express REM sleep. This is measurable as an increased number of "attempts" to enter REM during NREM sleep (Endo et al. 1998), as well as an increased REM density, decreased REM sleep latency (Ellman et al. 1991) and REM sleep rebound on recovery nights. EEG changes on recovery have been observed, even up to three nights post-deprivation (Endo et al. 1998; Toussaint et al. 1997). The probability of covert REM sleep occurrences is thus likely to be increased during or after REM deprivation. This is in fact supported by three kinds of findings. First, REM deprivation produces an increase of ponto-geniculo occipital (PGO) activity during NREM sleep in animal subjects (Dusan-Peyrethon et al. 1967; Ferguson & Dement 1969). Second, REM deprivation destabilizes recovery sleep in some human subjects, producing mixtures of REM and NREM sleep events ("ambiguous" sleep; Cartwright et al. 1967). Third, REM deprivation increases the sensory vividness, reality quality, and dreamlikeness of NREM mentation reports (Weinstein et al. 1991). In fact, REM sleep-deprived subjects in Cartwright's study (Cartwright et al. 1967) were found to have high percentages of dream reports from pre-REM transitional sleep. For one sub-group of subjects in this study (the "substitutors"), the degree of REM rebound after deprivation was negatively correlated with dreamlike content from NREM sleep awakenings. These subjects appeared to "cope with the changed sleep cycle by substituting a pseudo-cycle in which a good deal of REM content comes into awareness during the preREM sleep" (p. 302). Porte and Hobson (1996) have also proposed that increased REM pressure may account for very dreamlike NREM sleep reports in laboratory studies.

3.9. Evidence of covert REM sleep from animal studies

Early animal studies (Gottesmann 1964; Weiss & Adey 1965) detected signs of covert REM sleep even before the observation of intermediate sleep in human subjects. Sleep characterized by combinations of high amplitude anterior spindles (a sign of NREM sleep) and low frequency, dorsal hippocampal theta (a sign of REM sleep) was observed in rats and cats. Jouvet (1967) described PGO activity during transitions from NREM to REM sleep and throughout the REM sleep period and thought that these reflected inputs relevant to the visual images of dreaming. Steriade et al. (1989) also described PGO-related discharges of lateral geniculate neurons during pre-REM sleep states in cats, finding their signal-to-noise ratios to far exceed those found during REM sleep. Steriade's findings suggest that "vivid imagery may appear well before classical signs of REM sleep, during a period of apparent EEG-synchronized sleep" (Steriade et al. 1989, p. 2228). McCarley (1994) further advanced this hypothesis in describing brainstem neuronal membrane changes associated with REM sleep that may begin well before either EEG or PGO signs of REM. The transition at the membranal level is "gradual, continuous, and of long duration" (p. 375); it may also continue after the offset of a REM episode (see also Kayama et al. 1992). McCarley, too, speculates that NREM dreaming takes place during such REM-active transitions. Recent work (reviewed by Gottesmann 1996) has described additional physiological characteristics of intermediate states, including a seeming deactivation of forebrain centers and an apparent link to the processes that generate REM sleep.

3.10. Drug-induced covert REM sleep

Many drugs have been found to influence covert REM sleep, primarily by increasing PGO activity during NREM

sleep. Ketamine (Susic 1976), PCPA (Delorme et al. 1966), reserpine (Brooks & Gershon 1972; Delorme et al. 1965) and LSD (Stern et al. 1972) have all been found to augment the density of PGO spiking in NREM sleep in animal subjects. Other drugs have been found to affect intermediate sleep, such as the barbiturates and benzodiazepines, which prolong intermediate sleep at the expense of REM sleep (Gottesmann 1996), and nerve growth factor, which produces intermediate sleep ("dissociated" sleep) in addition to dramatically increasing REM sleep time (Yamuy et al. 1995).

3.11. Covert REM sleep induced by sensory stimulation

In addition to the many examples of spontaneously-occurring and drug-induced instances of covert REM sleep there are studies in which REM sleep-related processes have been experimentally activated during NREM sleep by simple sensory stimuli. In animal subjects, auditory stimuli reliably elicit PGO waves in all NREM sleep stages (Bowker & Morrison 1976; Hunt et al. 1998; Sanford et al. 1992b). Auditory stimuli also evoke phasic pauses in diaphragm activity during NREM sleep, another response typically associated with REM sleep (Hunt et al. 1998). There is a general tendency for PGO waves elicited in NREM sleep to have lower amplitudes than those from REM sleep (Ball et al. 1991b) although some studies fail to confirm this difference (Sanford et al. 1992a). In human subjects, combined auditory/visual stimulation during NREM sleep produces an *increase* in the amount of reported dream content (Conduit et al. 1997), a finding that prompted Conduit et al. to propose that the increase may be brought about by activation of REM sleep PGO activity during NREM sleep. Stimulation-induced covert REM sleep may even be exacerbated by REM deprivation because the latter reduces or eliminates inhibitory reactions to auditory stimulation during sleep (Mallick et al. 1991). Studies such as these indicate how easily covert REM sleep processes might be inadvertently triggered in (noisy) laboratory or home situations, and thereby produce elevated levels of sleep mentation reporting from NREM sleep. They may even help to explain instances of stimulus "tagging" in NREM sleep (see sect. 1.2.2) or instances of mentation recalled during sleep terror awakenings (see sect. 3.6).

3.12. Genetic factors

Studies of sleep in reptiles, birds, and rare mammals such as the echidna provide examples of apparent mixtures of REM and NREM sleep characteristics (Mukhametov 1987; Siegel 1998; Siegel et al. 1996). Echidna sleep, for example, consists of high brainstem neuron discharge variability (similar to REM sleep) and high-voltage EEG (similar to NREM sleep) (Siegel et al. 1996). Similarities between such patterns and the sleep of neonates have been noted (Siegel 1998).

4. Summary

Evidence from human and animal studies suggests at least nine factors that might induce covert REM sleep to be activated during NREM sleep. These include (1) low-level transitional processes anticipating and following normal REM sleep, (2) sleep onset REM processes during NREM sleep, (3) arousal processes, (4) "omission" of expected REM sleep episodes, (5) sensory stimulation during NREM sleep, (6) REM sleep deprivation, (7) drug effects, (8) mental illness, and (9) genetic factors. Each of these factors and their many possible interactions can be assessed empirically with appropriate experimental designs. In the following section we examine a probabilistic model as it is applied to primarily the first two factors in the preceding list. However, similar probabilistic models could evidently be used to examine any of the factors.

4.1. Evaluation of a probabilistic model

Factors 1 and 2 in the preceding section provide the clearest basis upon which the probability of recalling sleep mentation from NREM awakenings can be modeled. If covert REM sleep is indeed linked to (1) NREM sleep immediately preceding and following REM sleep episodes, and (2) NREM sleep following sleep onset, then probabilities of recalling mentation may be calculated from normative architectural measures. To demonstrate this, I employ an average sleep episode calculated from a sample of 127 nights of sleep recorded from 111 healthy, medication-free subjects (55M; 56F; M_{age} = 36.4 ± 14.5 years) in the Sleep Clinic of the Hôpital du Sacré-Coeur de Montréal. The ideal episode combines recordings from 25 first-night recordings and 102 second- or third-night recordings. Nights for which REM sleep onset latencies were greater than 150 minutes were excluded due to the possibility that these implicated "missing" REM sleep periods (see sect. 3.3). Subjects for whom any measure of REM or NREM time exceeded three

Table 2. *Descriptive statistics for six consecutive NREM and REM sleep episodes for 111 healthy non-medicated subjects (127 nights)*

	NREM				REM				BOTH
	Duration	N	SD	%	Duration	N	SD	%	Duration
1	84.4	127	24.8	85.7	14.1	127	7.8	14.3	98.5
2	85.4	127	22.0	78.5	23.4	127	11.4	21.5	108.8
3	84.0	126	20.7	76.6	25.7	124	13.4	23.4	109.7
4	68.4	116	21.8	71.1	27.8	106	14.2	28.9	96.2
5	56.5	67	19.5	68.8	25.6	49	14.8	31.2	82.1
6	52.3	21	21.4	66.3	26.6	7	13.7	33.7	78.9
	71.8	97.3	21.7	74.5	23.9	90.0	12.5	25.5	95.7

Table 3. *Probabilities of observing recall of sleep mentation assuming a 10-min (p-10) or a 15-min (p-15) covert REM sleep "window" around REM episodes (including sleep onset as a REM episode) for six consecutive NREM episodes. Window calculations are provided for mean NREM episode length and for ± 1 SD from this mean*

	MEAN			+ 1 SD			− 1 SD		
	duration	p-10	p-15	duration	p-10	p-15	duration	p-10	p-15
1	84.4	0.24	0.36	109.2	0.18	0.27	59.6	0.34	0.50
2	85.4	0.23	0.35	107.4	0.19	0.28	63.4	0.32	0.47
3	84.0	0.24	0.36	104.7	0.19	0.29	63.3	0.32	0.47
4	68.4	0.29	0.44	90.2	0.22	0.33	46.6	0.43	0.64
5	56.5	0.35	0.53	76.0	0.26	0.39	37.1	0.54	0.81
6	52.3	0.38	0.57	73.7	0.27	0.41	30.9	0.65	0.97
All	71.8	0.29	0.44	93.5	0.22	0.33	50.1	0.43	0.65

standard deviations (SDs) of the mean were also excluded. The duration of six consecutive REM and NREM sleep episodes were calculated and averaged over the 127 nights. No differences between men and women were noted so the two groups were combined. Descriptive statistics for these results appear in Table 2.

Probabilities of obtaining covert REM sleep (i.e., of recalling sleep mentation) in NREM sleep were calculated for a 10-min and a 15-min covert REM sleep window surrounding each REM sleep episode (Table 3). These two values were suggested by the literature reviewed above on the time course of covert REM sleep processes. They account for 20 and 30 min of each NREM episode respectively or a total of 120 and 180 min of total NREM sleep over the night. These numbers lead rather straightforwardly to probability estimates of finding covert REM in NREM sleep (Fig. 3). For the six NREM episodes, estimates ranging from 23–38% (mean: 29%) were found for the 10-min window and from 35–57% (mean: 43.5%) for the 15-min window. These percentages may be understood as probabilities of recalling sleep mentation with random awakenings from NREM sleep assuming either a 10- or a 15-min covert sleep window. Note that the 15-min window mean probability is strikingly similar to the average proportion of recall of mentation of 43.0% calculated from the 35 studies in Figure 2 (see also Table 1).

Calculations were repeated for the mean NREM episode length plus and minus 1 SD of this mean (Table 3). For longer NREM episodes (+1 SD), the 10- and 15-min window estimates dropped to 18–27% (mean: 22%) and 27–41% (mean: 33%) respectively. For shorter NREM episodes (1 SD), the two estimates climbed to 34–65% (mean: 43%) and 50–97% (mean: 65%) respectively. Thus, according to this model, with normal variations in NREM sleep episode length we might expect to observe large variations in the recall of sleep mentation – sometimes even exceeding the typical recall rate for REM sleep. This is, in fact, what we observed in the review of 35 studies. Across studies conducted after 1962, in particular, the recall of mentation from NREM sleep had a SD (15.5) that is over twice as large as that from REM sleep (6.7).

The prior calculations would suggest that the covert REM sleep window in human subjects is, on average, close to 15 min in duration. This may be an overly large estimate, given what is known about the time course of many processes preceding REM sleep. However, the value is based upon the assumption that mentation sampling takes place at random from any point in the entire NREM sleep episode. In practice (and in the 35 studies reviewed), researchers sample primarily stage 2 sleep, which tends to immediately precede and follow REM sleep. Calculated only for stage 2 NREM sleep, the probability of finding sleep mentation would be higher and the estimated REM sleep window would be correspondingly lower. In the present normative data set, 72.7% of NREM sleep was stage 2; weighting the 15-minute window by this proportion (.727) produces the more conservative estimate of 11 minutes.

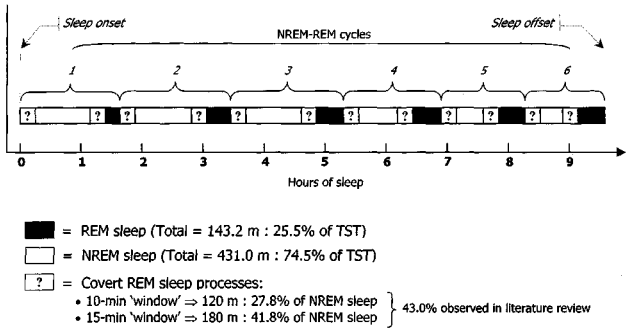

Figure 3. Probability model of covert REM sleep processes over six NREM-REM cycles: Normative results for 111 healthy nonmedicated subjects (127 nights). Illustration (to scale) of the normative sleep results listed in Table 2. The probability of obtaining covert REM sleep processes after a random awakening from NREM sleep may be calculated on a prototypical sleep episode with known architecture, here, a 9.5-hour night with six NREM-REM cycles. It is assumed in the model that covert processes (1) follow sleep onset and (2) precede and follow REM sleep episodes for a fixed duration or "window." The literature suggests a window of 10 to 15 min is possible. For a window of 10 min in length covert REM sleep accounts for 29.0% of NREM sleep. For a 15-min window, the value is 43.5% of NREM sleep. Random sampling of mentation during NREM sleep would thus fall upon covert REM sleep (where dreaming presumably occurs) 43.5% of the time for a 15-min window. Our literature review of mentation recall studies (see Fig. 2 and Table 1) revealed that overall 43.0% of NREM sleep awakenings are accompanied by mentation, a value similar to the postulated 15-min window. When weighted by the proportion of stage 2 sleep in the normative sample (.727), that is, by the stage most often sampled for mentation recall by researchers, the estimated window size can be adjusted to 11 min.

Taken alone, the probabilistic model described here might seem too simplistic to account for the numerous observations of mentation in NREM sleep. Evidence of mentation in stages 3 and 4 sleep is particularly difficult for this model to explain. Nevertheless, the large variability in NREM sleep episode length in the present normative sample illustrates the difficulty inherent in attempting to target the "middle" of NREM episodes to avoid possible covert REM sleep effects. One cannot be certain that covert processes anticipating the *next* REM sleep episode are not already active. Such attempts are clearly more likely to succeed from awakenings performed early in the night, but it is precisely at this time that less dreamlike mentation is observed.

In addition, this model does not bear on all factors thought to be associated with covert REM sleep processes, factors that might even trigger such processes unexpectedly in between the REM sleep windows. Studies reviewed earlier suggest that factors such as the intensity of prior REM episodes, extent of REM sleep deprivation, medication use and, especially, sensory stimulation during NREM sleep might evoke covert REM sleep processes. The laboratory itself influences many of these factors – as evidenced by the "first-night" (Browman & Cartwright 1980) and "second-night" (Toussaint et al. 1997) effects – and it may be an important determinant of the timing of covert REM sleep and, thus, of the chance of recalling mentation from NREM sleep. Research by Lehmann and Koukkou (1984) indicates that salient stimuli presented during all sleep stages may induce short-lasting brain states in the range of minutes, seconds or fractions of a second that are associated with discrete changes in cognitive process and EEG field potentials. They speculate that such "meaning-induced" changes in brain micro-state, whether evoked by internal or external stimuli, produce the typical characteristics of sleep mentation. Indeed, it is possible that closer attention to the phasic microstructure of EEG and other physiological variables may reveal measures by which covert REM sleep processes during NREM sleep can be quantified.

In conclusion, it is hoped that this exercise demonstrates how a new view of sleep stages as fluid and interactive, rather than as discrete and independent, may help reconcile a long-standing problem about one versus two imagery generators in sleep. As various phenomena of state overlap and intrusion among normal and sleep-disordered subjects are documented with increasing precision, their consequences for understanding sleep mentation will undoubtedly come into clearer focus. Obviously, not all recall of mentation from NREM sleep can be explained by the present probabilistic model. However, with further refinements, models of this type could account for a substantial portion of the variance in mentation recall. Several other factors, singly and in combination, remain to be more clearly defined, operationalized, and examined in systematic studies.

ACKNOWLEDGMENTS

This work was supported by the Canadian Institutes of Health Research and the Fonds de la Recherche en Santé du Québec. Dominique Petit, Tyna Paquett, Sonia Frenette and Anne Germain were exceptionally helpful in correcting and commenting on the manuscript. I thank Jacques Montplaisir for permission to publish normative data from the Sacré-Coeur Sleep Center database.

NOTES

1. Antrobus et al. 1995; Aserinsky & Kleitman 1953; Casagrande et al. 1996a; Castaldo & Holzman 1969; Conduit et al. 1997; Dement 1955; Dement & Kleitman 1957b; Fein et al. 1985; Foulkes 1962; Foulkes & Pope 1973; Foulkes & Schmidt 1983; Foulkes & Rechtschaffen 1964; Goodenough et al. 1959; 1965b; Hobson et al. 1965; Jouvet et al. 1960; Kales et al. 1967; Kamiya 1962; Kremen 1961; Lloyd & Cartwright 1995; Moffitt et al. 1982; Molinari & Foulkes 1969; Nielsen et al. 1998; Orlinsky 1962; Pivik & Foulkes 1968; Porte & Hobson 1996; Rechtschaffen et al. 1963a; Rotenberg 1993b; Slover et al. 1987; Snyder 1965; Stoyva 1965; Waterman 1992; Wolpert 1960; Wolpert & Trosman 1958; Zimmerman 1970.

I would like to thank my colleagues most sincerely for the careful attention they have given to evaluating my findings and hypotheses concerning the neuropsychology of dreaming. It appears that we truly are in the midst of a paradigm shift in sleep and dream science, and I consider myself fortunate to be part of it.

NOTES

1. I am referring to comments such as this: "There is a real danger in proceeding as if REM and NREM mentation are the same, which Solms seems to argue" (**Moorcroft**, para. 4).

2. Ogilvie et al. appear to think that this happens only in pathological cases.

3. This issue is obviously relevant to **Conduit et al.**'s question: If spontaneous arousal during sleep does not arise from the brainstem, where is its origin? Cf. **Moorcroft**'s implicit answer: "it is possible that while these forebrain areas are preferentially activated by pontine influences during REM they may also be activated by non-pontine sources" (para. 7).

4. Likewise, when **Portas** draws attention to the apparent discrepancy between my observation that anterior cingulate *lesions* are associated with increased frequency and vivacity of dreaming and the functional imaging data which show that this region is highly *activated* during "dreaming sleep" (REM sleep), she neglects the possibility that the observed activation is inhibitory.

5. Braun (1999) also summarized numerous "viable links" (of the kind requested by **Morgane & Mokler**) between the cholinergic REM-on mechanism and the putatively dopaminergic dream-on mechanism.

6. Cf. **Feinberg**'s pregnant remark: "We reasoned that, since brain physiology is qualitatively different in NREM and REM, but the conscious experience of [apex] dreaming in the two states is not qualitatively different, 'the striking NREM/REM differences in neuronal firing must *not* involve the neural systems that can affect the quality of conscious experience'" (emphasis added).

7. Here is a critical test of the *obligatory* involvement of DA in apex dreaming: cases with suitably located, complete lesions of the ventromesial frontal dopamine pathways and *preserved* apex dreaming would disconfirm my hypothesis. Incidentally, **Morgane & Mokler** seem to be unaware of the "unlikely" fact that all reported cases of cessation of dreaming with pure ventromesial frontal lesions did indeed sustain *bilateral* damage (Solms 1997a).

8. Occhionero & Esposito ask for specific examples of NREM triggers of dreaming. Complex partial seizures (which are not "stage specific" but usually occur during NREM sleep) provide an excellent example. Normal equivalents may be inferred. Incidentally, I do not see a basis for the distinction that **Gottesmann** makes in this connection between "dreams" and "hallucinations." Are apex dreams not hallucinations?

9. For example, **Doricchi & Violani** point to the weak statistical correlation between cessation of dreaming and adynamia in a small group of deep ventromesial bifrontal cases reported in my (1997a) study, but make no mention of the ubiquity of this symptom in the vast psychosurgical literature. (Cf. **Morgane & Mokler**'s questions concerning the putative link between dreaming and motivational mechanisms.)

10. I have responded elsewhere to his detailed criticisms of Freudian dream theory in relation to recent neuroscientific findings (cf. Hobson 1999c; Solms 1999c; 2000) and therefore will not address them again.

Covert REM sleep effects on REM mentation: Further methodological considerations and supporting evidence

Tore A. Nielsen

Sleep Research Center, Hôpital du Sacré-Coeur de Montréal, Montréal, Québec,Canada and Psychiatry Department, Université de Montréal, Montréal, Québec H4J 1C5 Canada, **t-nielsen@crhsc.umontreal.ca**

Abstract: Whereas many researchers see a heuristic potential in the covert REM sleep model for explaining NREM sleep mentation and associated phenomena, many others are unconvinced of its value. At present, there is much circumstantial support for the model, but validation is lacking on many points. Supportive findings from several additional studies are summarized with results from two new studies showing (1) NREM mentation is correlated with duration of prior REM sleep, and (2) REM sleep signs (eye movements, phasic EMG) occur frequently in NREM sleep. The covert REM sleep model represents one class of explanatory models that combines the two assumptions of mind-body isomorphism and a 1-gen mentation generator; its future development will depend largely upon a more detailed understanding of sleep state interactions and their contribution to mind-body isomorphisms.

NR0. Introduction

Reactions to my target article varied from the extremely skeptical to the highly supportive with as many commentators favoring it as doubting its conclusions. Eight principal themes addressed by various authors are listed in Table NR1; these are dealt with in turn in the sections that follow.

NR1. The definition of dreaming is inadequate

Some authors (**Antrobus; Clancey; Kahan; Pagel; Revonsuo**) expressed dissatisfaction with the definition of sleep mentation adopted in my target article. This dissatisfaction is justified to the extent that the classification scheme proposed in Figure 1 illustrates only in very broad strokes distinctions existing in the REM- NREM mentation literature that are central to my review, rather providing a detailed classification system *per se.* However, as the covert REM sleep model has evolved, I have found it increasingly imperative to develop criteria to discriminate among very brief and minimal forms of mentation. To contribute to this goal, I have revised my previous Figure 1 to incorporate several concerns raised in the commentaries (see Fig. NR1).

I agree that a more theoretically neutral definition of dreaming is desirable (**Revonsuo; Kahan**), that is, that a definition of dreaming should be based as much as possible upon the *contents* of subjective experience.[1] At the very least, such a definition would allow investigators of different theoretical orientations to study the same phenomenal objects in a convergent fashion. A chronic lack of agreement on the definition of dreaming has contributed much to the current confusion in the 1-gen versus 2-gen debate (cf. **Pagel**). Revonsuo is therefore justified in questioning my inclusion of "cognitive processes" in the classification of sleep mentation. Cognitive processes are, indeed, a theory-laden descriptor whose superordinate position in relation to other categories in Figure NR1 is based upon the hypothetical notion (e.g., Dixon 1981; Freud 1900) that most activity supporting subjective awareness occurs outside of

Table NR1. *Commentaries on Nielsen target article: Main themes*

Theme	Commentaries
1. The definition of dreaming is inadequate	**Antrobus, Clancey, Kahan, Pagel, Revonsuo**
2. Authors add new information that supports the model	**Borbély & Wittmann, Born & Gais, Cartwright, Feinberg, Gottesmann, Greenberg, Lehmann & Koukkou, Pace-Schott, Rotenberg, Salzarulo, Steriade**
3. Waking state processes need further consideration	**Greenberg, Hartmann, Ogilvie et al., Schredl**
4. Dreaming occurs during stages 3 and 4 sleep	**Blagrove, Bosinelli & Cicogna, Cavallero, Feinberg, Moorcroft, Ogilvie & Koukkou, Stickgold**
5. The model links dreaming exclusively to brainstem activation in REM sleep	**Bosinelli & Cicogna, Domhoff, Porte, Solms, Salin-Pascual et al.**
6. Evidence for isomorphism is lacking	**Hunt, Kramer, Morrison & Sanford, Panksepp, Solms, Vogel**
7. Elimination of REM sleep does not eliminate dreaming	**Panksepp, Solms, Shevrin**
8. The model needs validation	**Blagrove, Coenen, Conduit et al., Franzini, Gottesmann**

that awareness. Although I signaled the tentativeness of this category with question marks in my original Figure 1, its predominance in the diagram cannot be justified on observation alone. I therefore clarify in Figure NR1 that these processes (unobservable cognitive activity) are not necessarily associated with the other categories of mentation. I also describe a second type of cognitive activity that is normally unobservable but accessible through introspective effort. Justification for the category is given below.

Revonsuo proposes an alternative definition of dreaming. "Complex, temporally progressing content" is suggested to be a relatively theory-free feature that distinguishes dreaming from other types of cognitive activity during sleep. **Clancey** also proposes an alternative classificatory system that includes the sequencing or progression of perceptual categories. Temporal progression corresponds to the well-known criterion of "dramatic" quality that Freud (1900) borrowed from Spitta (1882) to define dreams, that is, dreams construct a *situation* out of hallucinatory images (Freud 1900, p. 114). While temporal progression may indeed be a common feature of much dreaming, and especially the dreaming common to most REM sleep, it is not likely a defining feature of all dreaming. For example, the criterion of temporal progression would exclude many of the uni-

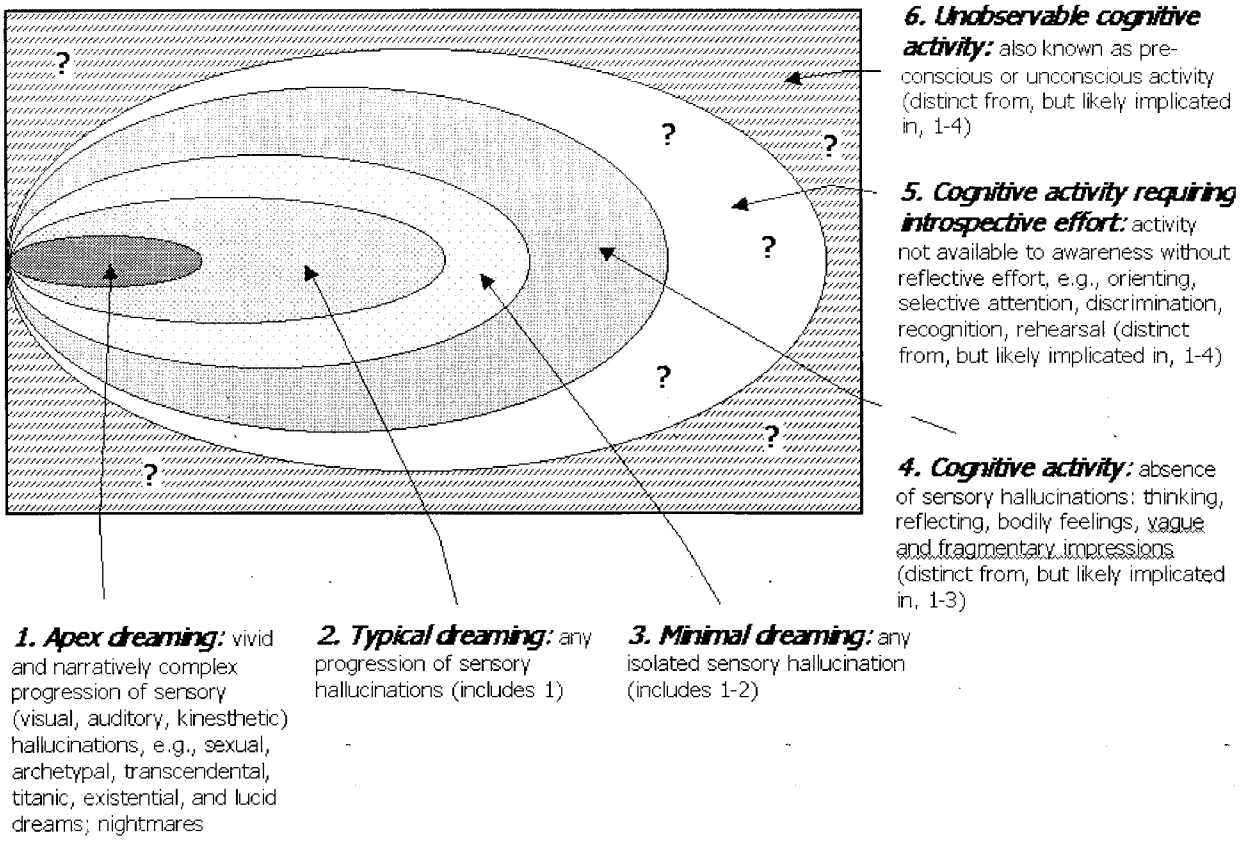

Figure NR1. Levels of specificity in defining sleep mentation – revised version of Figure 1 from target article. See text for details.

modal, static hallucinatory images typically reported in our studies of sleep onset mentation (Germain & Nielsen 1997; Nielsen et al. 1995), and this on a seemingly arbitrary basis. Arbitrary because the studies, including my self-observational studies of brief hypnagogic images (Nielsen 1992; 1995) (http://www.crhsc.umontreal.ca/dreams/TNmodeling.htm), suggest that such static images are often endowed with a hallucinatory quality that renders them quite dreamlike. The hallucinatory quality is unmistakable, even for "fleeting" images and "sleepiness" sensations that occur prior to the more fully formed hypnagogic images themselves. Hallucinatory quality is associated with the seeming *sensory* nature of the imagery and appears to involve a degree of *apparent orientation* to ("self-participation" in) the imagery (e.g., Bosinelli et al. 1974; **Herman**). Apparent orientation here refers to illusory sensations of a spatial distribution of objects, including, and sometimes consisting only of, the self, the apparent vertical, apparent depth, and/or apparent motion. Hallucinatory quality was to Freud as important a defining attribute as was dramatic quality, the purported "transformation of ideas into hallucinations" (Freud 1900, p. 114). In Figure NR1, hallucinatory quality defines a minimal dream, whereas temporal progression distinguishes minimal dreaming from more complex and typical forms of dreaming.

This revision in Figure NR1 also responds somewhat to **Shevrin & Eiser**'s comment that Freudian theory is ignored by the covert REM approach. It may also respond to **Antrobus**'s point that an unidimensional measure of mentation recall/non-recall is inferior to a multidimensional approach in making fair comparisons of REM and NREM mentation. The criterion of "hallucinatory quality" might be applied equally well to mentation in all sensory dimensions, and possibly also to emotion, pain, and other organic sensations. If so, fair *uni*dimensional comparisons of "minimal dreaming" could still be made across sleep states using this criterion.

More generally, I believe that the continued disagreement over defining dreaming is based upon at least two methodological shortcomings. First, there is not only disagreement over how best to accomplish an accurate phenomenology of subjective experience (e.g., Dennett 1991), but all too often available phenomenological methods (e.g., Businк & Kuiken 1996; Husserl 1965) are disregarded in research. The result is that definitions are proposed without much reference to methods of deriving them (cf. **Pagel**), and no standardization is possible. Second, subjects in sleep mentation experiments, on whose responses definitions of subjective experience are often based, are typically naïve to the exigencies of introspective reflection. This issue goes beyond the concerns voiced in commentaries by **Antrobus** and **Schredl** that mentation reports have uncertain validity. Rather, the point is that introspectively untrained subjects simply cannot accurately report upon all microstructural constituents of hallucinatory quality that might be crucial in identifying a subjective experience as a dream. Conversely, there is today very little support for introspective approaches that involve training subjects and/or investigators to access these microstructural levels of subjective experience precisely and reliably. To reflect this concern, Figure NR1 distinguishes a type of cognitive activity that is available to awareness only with some degree of introspective effort.

In sum, although I agree that definitions of dreaming should be theory-free, I doubt that such approaches can be developed without a more concerted emphasis on introspective and self-observational methods of study that involve the training of both subjects and experimenters. Therefore, in lieu of importing definitions from consciousness research or elsewhere, the most reasonable course of action in the short-term may simply be to refine terminology that has evolved over the years *within* the discipline of dream research and whose connotations and nuances are thus understood more or less consensually by a large number of researchers active in the area. However, a long-term strategy for addressing this basic issue is clearly needed.

NR2. Authors add new information that supports the model

At least 12 commentaries (**Borbély & Wittmann; Born & Gais; Cartwright; Feinberg; Gottesmann; Greenberg; Lehmann & Koukkou; Pace-Schott; Porte; Rotenberg; Salzarulo; Steriade**) described research and/or theory consistent with or supportive of the covert REM sleep model. An important paper by Toth (1971), which was suggested by Rotenberg (1982) and another by Schwartz (1968), which was mentioned by Gottesmann**,** were not referred to in my target article but contain evidence fairly directly supporting the covert REM sleep model. I will briefly summarize both. Toth (1971) devised miniature electrodes which, when glued to the eyelids overlying the cornea, more than doubled the sensitivity of standard EEG recordings. This innovation allowed him to quantify very small amplitude eye movements occurring in NREM sleep (cited in Rotenberg 1982). Although this study urgently needs replication, the report suggests both a straightforward method for measuring covert REM sleep processes in NREM sleep and, if confirmed, that such processes may be more present in NREM sleep than has been appreciated.

Schwartz (1968) observed "indeterminate sleep" in both hypersomnolent patients and control subjects shortly after sleep onset during afternoon naps. Distinguishing among *very slow* eye movements, *medium fast* eye movements, and *rapid* eye movements he found that medium fast eye movements could be observed in all patients and controls at each sleep onset and that they were more common than very slow eye movements. Medium fast movements were recorded consistently in stage 1B and especially in stage 2, and then decreased in quantity and amplitude as slow waves predominated. They were rare in stage 3, but nevertheless often accompanied K-complexes. He noted that the voltage of these eye movements varied with electrode distance and individual differences in anatomy, thus standard EOG recordings may be insufficient to identify them under routine recording conditions. He also identified phasic EMG activity occurring immediately after the onset of EEG-defined sleep stage 1Band. These consisted of small movements or twitches of the face, hands, feet, head, shoulders, and even the abdomen, and were indistinguishable from the phasic movements of REM sleep. Schwartz noted that medium fast eye movements occur also in REM sleep, especially just before the onset of rapid eye movement bursts. Finally, he found dream recall after spontaneous awakenings from stages 1B and 2 sleep that had been accompanied by medium fast eye movements. He also cites a study by Kuhlo and Lehmann (1964) in which eye movements sim-

ilar to his medium fast eye movements were studied in conjunction with hypnagogic imagery. We also report these types of events in preliminary study 2 reported in section NR8.2 (see Figs. NR3–8). Although Schwartz's study also requires replication with a larger sample of healthy control subjects, his findings concerning REM sleep-like eye movements, phasic EMG activity, and dreaming at sleep onset are strongly supportive of the covert REM sleep model. Together, our results, the findings of both Toth and Schwartz, and the neurophysiological observations concerning sleep onset eye movements contributed in the **Porte** commentary, all bolster two points I make in the target article: (1) rapid eye movements may not be particular only to REM sleep and (2) slow eye movements may also be a correlate of REM sleep. If so, sleep onset may be considered to be a kind of short-lived or fragmentary episode of (convert) REM sleep, and sleep onset imagery a type of brief (convert) REM dream.

Other commentators discuss findings from sleep deprivation research that are consistent with the covert model. **Born & Gais** and **Cartwright** both emphasize that REM sleep propensity is heightened after REM sleep deprivation. This covert propensity may be a critical factor in studies of deprivation effects on memory because of continued effects of covert REM sleep processes on memory consolidation, despite the apparent absence of the REM sleep state itself (Born & Gais). The improvement in mood and increased drive behaviors produced by sleep deprivation in depressed subjects may also be due to covert REM sleep (Cartwright). We have observed that healthy subjects undergoing sleep deprivation sometimes manifest REM sleep signs in their NREM sleep polysomnograms during recovery sleep (Nielsen & Carrier 2000, unpublished). To illustrate, Figure NR2 shows the sleep onset tracing and hypnogram of a 31-year-old healthy female following 40 h of sleep deprivation. The tracing contains distinct rapid, medium fast, and slow eye movements in conjunction with a background of stage 1 sleep.

Cartwright also suggests that the covert REM sleep model is supported by studies demonstrating a coupling of REM sleep and dreaming under dissociated circumstances such as the NREM dream reports of light sleepers who are in high arousal throughout sleep, and in other sleepers for whom there is a low arousal threshold following sleep deprivation or acute stress. Violent sleepwalking episodes also occur following periods of extended sleep loss and stress. Finally, sleep state dissociation is seen in subjects with REM sleep behavior disorder in which there are REM sleep signs but lapses of muscle atonia. There is a wide range of phenomena that involve dreamlike mentation in NREM sleep (see review in Nielsen & Zadra 2000) whose closer study could shed light on whether dissociated REM sleep processes are implicated in the mentation. Dissociation of REM sleep processes is discussed in greater depth in section NR5.

Several commentators suggested ways that EEG or other brain imaging methods might be harnessed to quantify covert REM processes. A figure in the **Feinberg** commentary illustrates very nicely how delta EEG power could serve as such an index. Delta power normally drops sharply at the onset of REM sleep episodes and then rises again with the start of the following NREM episode and repeats this variation across the night. The Feinberg figure illustrates three types of commonly observed events that are consistent with the covert model (see also Dijk et al. 1995; Landolt et al. 1996):

1. *Sleep onset REM processes:* Not only is delta power low during REM episodes, but it is similarly low at sleep onset, when dissociated REM sleep processes are hypothesized to occur.

2. *"Skipped" first REM episodes:* Delta power estimates during the first 90 min of subjects 1 and 3 recovery nights (RN) drop sharply even though the expected REM sleep episodes are not scored. **Feinberg** indicates that these episodes are often not scored during RN while they are scored during baseline nights (BN). Such findings support the existence of covert REM processes during "skipped" REM episodes as discussed in the target article and further suggest that they may be more likely during recovery from sleep deprivation. Delta power analyses reveal that such tendencies toward skipped REM episodes are more striking in children and young adolescents than young or middle-aged adults (Gaudreau et al., in press) and confirm that the exceptionally long REM onset latencies (up to 3–4 h) seen in young children are often likely due to such skipped REM episodes (Benoit 1981; Bes et al. 1991; Dement & Fisher 1964; Palm et al. 1989; Roffwarg et al. 1966; 1979). Palm et al. (1989), for example, found in a sample of 8–12-year-olds that on 67% of nights the first sleep cycle lacked REM sleep as traditionally scored; in 88% of these, "an abortive EEG sleep pattern was found with traits specific to REM as well as to non-REM" (p. 306). The main anomaly observed in their study was a *lack* of rapid eye movements during the anomalous REM episode. Other research (e.g., Carskadon et al. 1987) has suggested that long REM latencies (i.e., skipped REM sleep episodes) may interact with both the "first-night effect" (with REM latencies higher on the first night) and gender (with REM latencies decreasing over laboratory nights 1 to 3 for girls and nights 1 and 2 for boys). Skipped first REM periods also appear in adults who are under conditions of sleep loss (Berger & Oswald 1962).

3. *Pre- and post-REM covert effects:* The gradient with which delta power decreases and increases before and after REM sleep varies from subject to subject, within nights, and over experimental conditions. Subject 1's BN plot shows that power increased moderately after the first REM episode but remained very low after the second and third. Such profiles correspond to a predominance of stage 2 sleep in the subject record. Are covert REM sleep processes more likely to manifest during these lulls in delta power? Possibly. Waterman (1992) found delta power, but not other frequency bands, to be negatively correlated with dream recall (word count) and to account for a significant portion of the REM-NREM and time of night differences in dream recall. Furthermore, these findings held for young, but not older, subjects. **Salzarulo** also emphasized an inverse relationship between delta power (slow-wave activity or SWA) and cognitive processing in sleep – in this case, the number of statements that comprise each dreamed "story event." Salzarulo goes further, however, to suggest that SWA reduction across the night reflects diminution of the more general process S, and that this reduction serves as a physiological condition for cognitive experience irrespective of sleep stage. Such a 1-gen notion is, in fact, consistent with studies demonstrating increases in dream intensity later in the night (e.g., Antrobus et al. 1995), but the effect appears to be much smaller than the REM-NREM sleep difference in dream intensity (**Antrobus** et al. 1995).

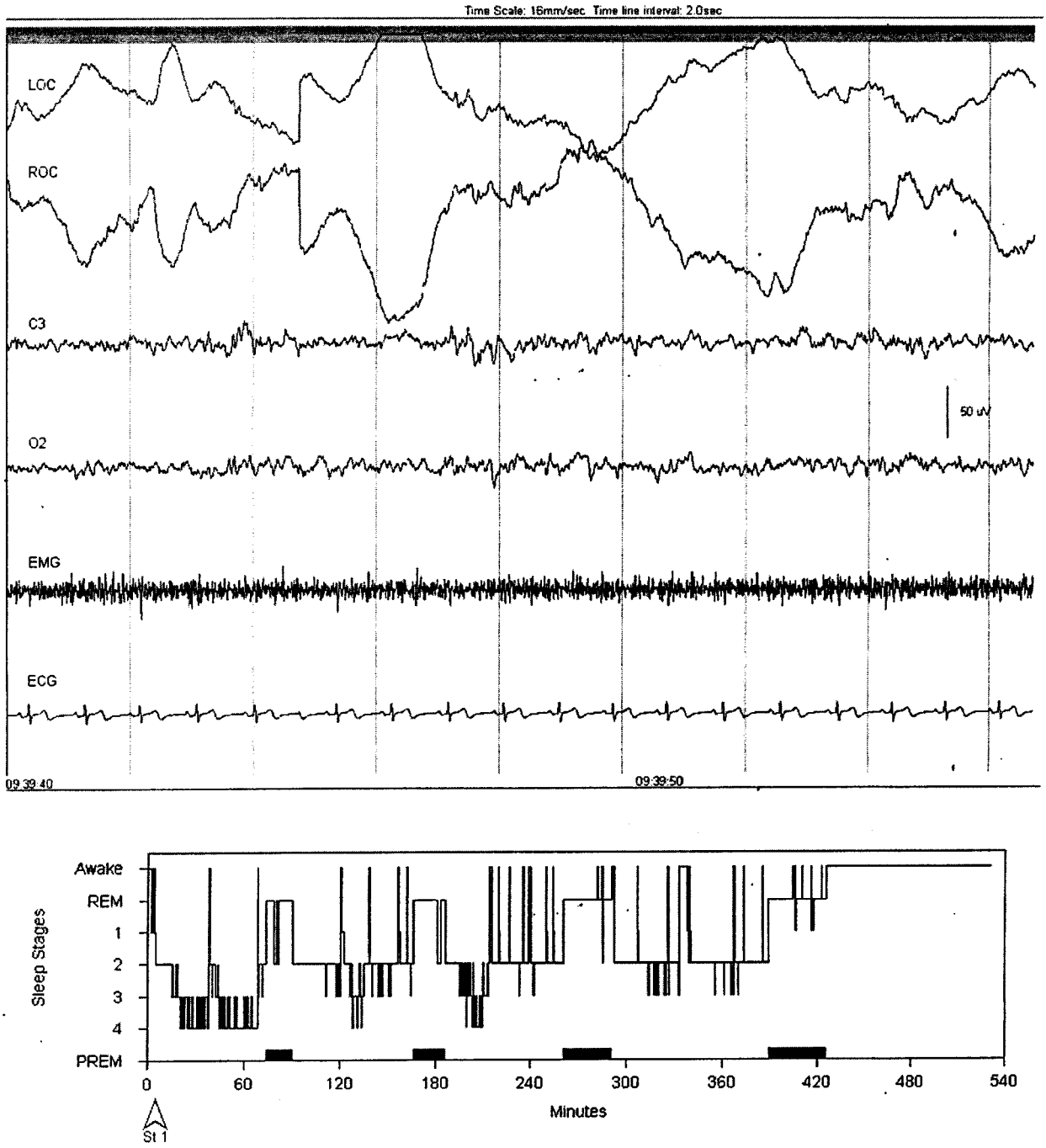

Figure NR2. Hypnogram and polysomnographic (PSG) tracing from a healthy 31-year-old female subject on her first recovery (daytime) sleep after enduring a 40-hour constant routine. Rapid, medium fast, and slow eye movements are clearly visible against a background of stage 1 EEG and EMG.

The commentators considered various other brain imaging measures in relation to hypotheses about covert REM sleep and dream production. The suggestion that episodes of covert REM sleep are equivalent to lapses of attentional control during the waking state (**Greenberg**) is conceptually similar to the hypothesis that a basic dream production mechanism depends upon activation of attentional mechanisms (**Morrison & Sanford; Conduit et al.**), e.g., the PGO wave, and that such mechanisms may be activated sporadically in NREM sleep. Such processes may be indexed by more detailed measures of spontaneous EEG during REM and NREM sleep or by various evoked potential techniques. The dissociation of REM sleep processes into other sleep states also corresponds well with **Lehmann & Koukkou**'s (1984) notion of *momentary brain states,* that is, very brief (in the order of seconds or less) changes in brain state within a sleep stage. Their work suggests that evidence of such momentary state changes my be "hidden" in rapidly changing EEG parameters, but that their decodification may be forthcoming with more sophisticated methods of quantifying the EEG. Alternatively, covert REM sleep processes may parallel rises and falls in mechanisms of brain *synchrony* (**Pace-**

Schott), presumably a measure derivable from EEG coherence. We have found that some features of dream content are associated with generalized cortical coherence in REM sleep (Nielsen & Chénier 1999) but we have yet to examine NREM mentation for the same correspondences.

Steriade points to work he published over a decade ago that supports the covert REM sleep model in suggesting that increases in the signal-to-noise ratio of PGO-related spike bursts in visual thalamus is high during pre-REM sleep transitional periods, a change that might underlie the generation of vivid mental experiences. Other brain indicators of covert REM sleep processes may be tied to deactivation of heteromodal association areas, as indicated by recent brain imaging studies (**Borbély & Wittmann**). Such studies implicate structures and mechanisms in covert REM events that may be beyond the capacity of present-day EEG methods to quantify.

Porte points to the need for further investigation of EEG spindle characteristics in relation to REM sleep signs and describes how the neurophysiological structure of NREM stage 2 sleep could, in fact, be compatible with the intermittent appearance of such signs. Specifically, covert REM processes may be more likely to occur between distantly spaced sleep spindles because of an inhibitory influence during the interspindle wave refractory period. This notion is consistent with our own observations in study 2 (see sect. NR8) of medium fast and rapid eye movements occurring between spindles in stage 2 sleep. However, in our study some eye movements were also observed to occur in close proximity to, if not simultaneous with, sleep spindles (see Fig. NR7), suggesting that any inhibitory influence of the spindle generator on intermittent REM sleep events may be variable and transitory. It must also be noted that non-cortical REM sleep processes such as muscle twitches, penile tumescence, heart rate variability, and other autonomic fluctuations that may manifest in NREM sleep are not likely to be affected by the spindle wave refractory period.

Of course, the development of new forms of sleep monitoring need not be restricted to the EEG. To illustrate, REM and NREM sleep are distinguished by autonomic changes, most notably an increase in sympathetic activation during REM sleep (Berlad et al. 1993). The description of such changes has until recently been severely restricted by a lack of appropriate recording methods. It is therefore noteworthy that a recently developed plethysmographic method for quantifying peripheral vasoconstriction during sleep has found that vasoconstriction is highly characteristic of REM sleep, and that its increase can be detected at least 30 minutes before the beginning of REM sleep as it is traditionally scored (Lavie et al. 2000). This finding is entirely consistent with the covert REM sleep model and suggests that the "window" around the REM sleep state during which covert processes might influence NREM sleep mentation could be larger than the 10–20 min window discussed in the **NIELSEN** target article.

In sum, by directing attention to both micro- and macrostructural dissociations of REM sleep processes into NREM sleep, the covert REM sleep model highlights potentially fruitful directions in which biosignal imaging and interpretation methods may be developed. These methods may lead to more precise definitions of sleep stages and their relationships.

NR3. Consideration of waking processes in the model

Some commentators (**Greenberg; Hartmann; Ogilvie et al.; Schredl**) expressed dissatisfaction that the covert REM sleep model does not deal with potential incorporations of waking state processes into sleep. They viewed this as either a weakness in the model or as a potential avenue for its further elaboration. On the one hand, some authors pointed to the immediate post-awakening state as a factor that could potentially influence REM/NREM mentation differences. For instance, Greenberg emphasized that gradual awakenings from NREM sleep can lead to reporting of more dream content (Goodenough et al. 1965a). Goodenough believed that this accounted for some but not all instances of NREM mentation. However, it remains an open question whether such "gradual awakenings" involve the intermingling of waking state processes with NREM sleep mentation or the brief activation of REM sleep processes during transition to full awakening. There may occur a substantial degree of secondary elaboration during awakening as Freud (1900) suggested, or content may be produced as part of the arousal process as in the case of some sleep terrors (Fisher et al. 1973). In the target article I deal at greater length with the possibility that brief or fragmented episodes of REM sleep occur unnoticed in the course of waking up. It is important to emphasize that even a minor elaboration or generation of content at this time would be sufficient for a report of genuine dreaming to be "identified." As studies of both hypnagogic imagery and "disorders of arousal" demonstrate, *even fleeting experiences of hallucinatory content are sufficient to generate* bona fide, *albeit diminutive, reports of dream mentation.* Subject differences even further complicate the picture, because some factors unique to subjects may enhance REM/NREM differences (**Schredl**). Since more elaborate mentation reports may be given by subjects who have a more verbose verbal style, who have superior verbal short-term memory, or whose recall is "enhanced" by training, the degree of elaboration of even brief mentation samples may also be increased.[2] Subjects who are introspectively inclined and verbally confident may well find it a simple task to elaborate a single fleeting image into a coherent, multi-propositional, narrative episode.

A study by **Herman** et al. (1978) illustrates the subtlety of the problem. This work demonstrates clearly that mentation reports from NREM (but not REM) sleep are rendered more "dreamlike" (as measured by Foulkes's *dreamlike fantasy* scale) when experimenters or subjects themselves are systematically biased to believe that this is the expected result. Herman et al. even suggested that "a possible major source of variance in NREM recall studies is the predisposition of the investigator" (p. 91). Factors such as experimenter influence are methodological obstacles to conducting fair and unbiased comparisons between REM and NREM mentation. The covert REM sleep model helps to bring many of these methodological issues into focus and it suggests novel means for controlling them. It is, in one sense, a methodologically driven model whose stance in the face of acknowledged shortcomings in the definitions of REM and NREM sleep is to advocate that these definitions be more precise and their presumed cognitive correlates be more thoroughly studied.

Other authors consider waking state processes as a means

of extending the covert REM sleep model. For example, **Ogilvie et al.** take issue with the notion of covert REM mechanisms underlying sleep onset mentation in the first NREM-REM cycle, this based upon the presumably circadian nature of sleep onset REM periods (Sasaki et al. 2000). It is argued that waking state processes are more likely to be incorporated into sleep onset mentation that are REM sleep processes. This suggestion is feasible and consistent with some work on sleep onset mentation (e.g., Cicogna 1994) and some results from study 2 reported in section NR8.2. However, the covert REM sleep explanation cannot be ruled out in light of several studies previously described. For example, the study by Schwartz (1968) and our own preliminary findings (sect. NR8) are consistent with the assertion that REM sleep events occur at sleep onset. I agree that REM sleep processes are influenced by circadian factors, but such factors do not necessarily *preclude* the occurrence of extremely brief, if not fragmented, REM sleep processes at sleep onset and elsewhere. In fact, if a REM sleep potential does exist early in sleep, a very weak circadian pressure might be *expected* to fragment, dissociate, or diminish it rather than simply to impede its expression in an all-or-none fashion.

Hartmann suggests that dreaming mentation should be seen as part of a continuum with daydreaming and other varieties of waking mentation, and that the components of this continuum are not different enough to warrant considering them products of different mentation generators. It is true that some comparative studies of waking and sleep mentation find evidence of structural similarity (Kahan et al. 1997; Kahan & Laberge 1996) but there are in my view too few comparative studies of such features and their physiological correlates to elaborate a definitive model. *The evidence in support of a REM-NREM sleep mentation continuum is controversial enough!* Nevertheless, Hartmann does take some constructive steps toward specifying a global structure for one possible wake-sleep mentation continuum and of proposing factors that might describe how dreaming and waking vary on this continuum.

NR4. Demonstrations of dreaming during stages 3 and 4 sleep and their implication for the existence of mentation unique to NREM sleep

Several authors suggest that the covert REM sleep model cannot explain reports of dreamlike mentation in stages 3 and 4 sleep (or slow-wave sleep; SWS). Supporters of this notion point to, among other evidence, a study by **Cavallero** et al. (1992) that involves direct sampling of SWS mentation. There is much evidence reviewed in the target article and in the present reply that provides a basis for at least questioning the definitiveness of this and other such studies of SWS cognition (Cicogna et al. 2000). In general, I question how many of the mentation reports collected from SWS occurred under conditions which, according to the covert model, were demonstrably free from the potential influence of covert REM sleep? These include variables such as time from preceding REM sleep periods, time prior to next REM sleep periods (which, with today's instruments, may be impossible to calculate with any certainty), partial sleep deprivation (producing increased REM sleep pressure), sources of sensory stimulation during sleep (which are potentially numerous in a laboratory), the effects of drugs or alcohol and/or withdrawal from these, and so forth. This might seem like an exorbitant list of criteria to exclude but the approach is not unlike how a clinician proceeds in excluding possible alternative diagnoses of a sleep problem. In fact, a partial remedy to the caveats posed by the covert REM sleep model may be to routinely evaluate (and publish) pertinent details of subjects' sleep states along with the usual reporting of sleep mentation characteristics. For example, analyses of NREM sleep hypnograms or sleep tracings from the preawakening interval could exclude the presence of sleep fragmentation, eye movements, motor activation, and other possible REM sleep signs. Further, quantified measures of sleep state transitions, sleep efficiency, and so forth could provide valuable information about how "dissociable" a subject's sleep is. Subjects could also be screened for frequency of nightmares and other parasomnias, especially because such subjects may be particularly inclined to participate in studies of sleep mentation. Our findings from study 2 (see sect. NR8) suggest that covert REM processes might be more prevalent or more active among nightmare sufferers. One post-traumatic nightmare patient from our sample who demonstrated a very high number of REM sleep signs in NREM sleep also had an extremely variable hypnogram on both recording nights and reported dreaming vividly throughout the night (see Figs. NR6 and NR7).

In addition to these concerns, the **Cavallero** et al. study and others like it should be interpreted with caution for at least two methodological reasons. First, several subjects (17%) in the Cavallero et al. study recalled *no mentation from SWS whatsoever* and were excluded from the study sample. Other subjects required more than one night in the laboratory to achieve a recall of mentation from SWS. Had such observations been made for awakenings from REM sleep, they would likely have caused a significant stir and provoked further investigation to determine their clinical implications. However, for NREM sleep such a finding raises no eyebrows, is readily dismissed, yet remains completely inexplicable to a model that proposes regular SWS dreaming. Second, it is not stated whether the experimenters in this study were naive to the nature of the hypotheses. Subjects could have been pressured inadvertently by experimenters to produce mental content, as **Herman** et al. (1978) so clearly demonstrate. As noted in the previous section, the amount of mental activity during SWS that is stimulated either by covert REM sleep or wakefulness processes could be quite small while still seeming to produce a somewhat elaborate mentation report from SWS. Cavallero et al.'s work on SWS dreaming has made an important contribution to research in the area but it is not without its methodological limitations.

Some commentaries (**Bosinelli & Cicogna; Cavallero**) reiterated the argument that studies of REM/NREM mentation that have controlled for the length of the mentation report (with, for example, total word count as a covariate) have found that apparent REM/NREM stage differences are diminished or disappear altogether. The finding of residual differences that are discussed in the target article are thus seen to be artifactual, for example, the result of differences in the spreading of mnemonic activation in the two sleep states. Such research findings are interpreted as supporting the view that dreaming occurs in both REM and NREM sleep but not because of any link to possible covert REM sleep processes. Although more studies would seem to be called for, two points should be reiterated: (1) The

widespread use of report-length correction methods over the last decade may well be in doubt (see discussions in **NIELSEN** and **HOBSON ET AL.** target articles). Thus, the seeming diminution of stage differences with length-control may be a dramatically over-stated phenomenon. (2) My review of the literature on REM/NREM mentation comparisons in the target article resulted in no less than a dozen studies that report residual differences, *despite* the implementation of such report-length controls. In fact, in this literature I have found little evidence that stage differences are ever entirely eliminated with length controls.

Blagrove adds to this debate the observation that purportedly qualitative residual differences are nevertheless quantitative in nature (e.g., number of characters, visual imagery word count); there are thus no qualitative differences *per se* between REM and NREM reports, and a 1-gen hypothesis is supported. This observation points out an important problem: measurements are quantitative (usually), whereas features themselves are qualitative (usually). So a seemingly quantitative difference between groups could belie what is, in fact, an important qualitative difference. For example, it would be foolish to suggest that a group of subjects each bearing three eyes was only *quantitatively* different from a group of normal two-eyed subjects. Yet an eye-count measure would lead to just such a conclusion. Such comparisons must be informed by the normative context of the measurements. One solution to this type of methodological problem is discussed in the **HOBSON ET AL.** target article (disallow length controls). Another is discussed by **Antrobus** (compare mentation reports on a multidimensional measure). Alternatively, if the use of report-length controls is justifiable, then a fair approach would seem to be to evaluate all quantitative measures in the same units as the weighting factor, for example, word count of all bizarreness text weighted by total word count (cf. **Hunt** et al. 1993). Such an approach could also lend itself to multidimensional comparisons because all measures would be based upon the same metric. This approach is similar to one employed by Antrobus et al. (1995).

NR5. The model links dreaming exclusively to brainstem activation in REM sleep

Several commentators (**Bosinelli & Cicogna; Domhoff; Porte; Solms; Salin-Pascual et al.**) suggest that the covert model implies a particular view of REM sleep as governed exclusively by brain stem sources of activation. This "bottom-up" interpretation of the model derives from the early reciprocal interaction model of REM sleep (McCarley & Hobson 1979) that places control of REM sleep in pontine "REM-on" neurons. The **Solms** commentary provides a clear definition of this view of REM sleep state and thus allows useful comparisons with the covert REM sleep model. Solms defines REM sleep to be synonymous with an executive mechanism that recruits various physiological events (e.g., EEG desynchronization, muscle atonia, rapid eye movements) and coordinates them into "a distinctive configuration." He identifies the brainstem as this executive mechanism and he disputes whether it can, in fact, be responsible for the generation of dreaming. The **SOLMS** target article further addresses this claim. This view, the separation of REM sleep into a specific control mechanism and its coupled components, can be compared with the covert REM model by posing the following three key questions about the definitional concepts.

NR5.1. Are all aspects of REM sleep control located in the brainstem?

There is still disagreement as to the extent of involvement and, ultimately, of the importance to REM sleep generation of pontine brainstem regions. **Salin-Pascual et al.** review several studies that challenge the notion and that implicate a major role for the hypothalamus. **Morrison & Sanford** and **Feinberg** also qualify this notion with reference to forebrain structures, such as the hypothalamus, which may influence brainstem activity. **Jones** calls into doubt brainstem control by referring to Jouvet's critical experiments that eliminated REM sleep by eliminating corticofugal influences on brainstem. **Nofzinger** describes new brain imaging findings that support forebrain involvement and that cast doubt on the specificity of brainstem involvement. **Lydic & Baghdoyan**, on the other hand, support the notion of brainstem control quite vigorously. This small sampling of diverse opinions reveals the wide disagreement about whether pontine brainstem should be accorded the status of a *unique* control mechanism for REM sleep. It also underlines the importance of distinguishing among types of executive control; for example, between mechanisms that *trigger* REM sleep onset and those that *maintain* REM state integrity over time. Pontine brainstem may well be a primary determinant of REM sleep onset (although this notion is still contested) while forebrain may affect REM sleep intensity, consolidation, or duration. Consistent with this possibility, there is evidence (Montplaisir et al. 1995) that among patients with Alzheimer's disease, which affects basal forebrain but not pontine brainstem, REM sleep timing is normal, but REM sleep episodes are shorter than normal in duration. To reiterate the preceding, there is disagreement as to whether brainstem is the only, or even the most important, controller of REM sleep; this is largely because there are so many features of REM sleep that must be controlled.

NR5.2. Do isomorphic correlates of dreaming exist only at the level of REM sleep executive control?

Notwithstanding the previous problem, it may be premature to conclude that REM sleep control and dreaming control are isomorphic. This is because little if any research has studied the isomorphism question at these corresponding levels of complexity. In fact, most studies seeking to find isomorphic relationships in sleep have concentrated exclusively on what **Solms** refers to as the individual "components" of the REM sleep state. As I argue in the next section, there is in fact evidence that isomorphic relationships exist between isolated physiological variables and specific attributes of dream content. On the other hand, there are no studies that have yet managed to directly assess whether the pontine "REM-on" neurons and their presumed executive control structure are associated with dreaming.

In contrast to **Solms**'s view, I think it is feasible that some essential processes of dream organization occurring at a *microstructural* level may be found to be associated with components of the REM sleep state. By microstructural organization I mean processes governing the ordered and coherent presentation to awareness of a sequential flow of inter-connected multisensory images. To achieve this, it seems likely that the dream production system depends upon a great degree of autonomy in the *local* organization

of image elements such that the integrity of every part of the (arguably complex) imagery sequence does not hinge upon the fidelity of a single, central control mechanism. Image elements may have mechanisms of attraction and repulsion that allow them to dissociate and regroup into larger units much as basic physical elements combine to create more complex molecules and substances. Elsewhere (NIELSEN 1995) (www.crhsc.umontreal.ca/dreams/TNmodeling.htm), I describe a mechanism referred to as *transformative priming* that could fulfill such a local control function over information contained in a wide variety of modalities. Transformative priming involves one image or image element activating a conceptually related image or element (priming) and then combining with it into a completely novel form (transformation). The process, which unfolds over a time span of milliseconds, could account for the local coherence of minimal dreaming and of more complex forms of dreaming as well.

NR5.3. Can REM sleep events dissociate from the REM sleep configuration?

According to **Solms**'s commentary, even individual physiological events that may be correlated with dreaming should not be identified with the REM sleep state if they occur outside of that state because they are not part of the presumed brainstem control mechanism; their source is "indeterminate." On the other hand, the notion of the covert REM sleep model is that REM sleep events that occur outside of REM sleep are somehow *dissociated* from the state and can continue to exert an influence; their source is somehow still "linked" to REM sleep. In fact, to the extent that the frontal and parietal structures identified by Solms are typically implicated in dreaming and are also typically associated with REM sleep, I would view his findings as completely consistent with, if not splendidly supportive of, my own model. The action of these structures Solms considers to be *independent* of REM sleep; the covert model would describe them as a *dissociation* of REM sleep processes into another sleep state. The solution to this discrepancy may lie in whether state dissociation can be proven to be a valid construct.

A substantial body of literature in fact supports the concept of sleep state dissociation (Mahowald & Schenck 1991) and thus also supports the related notion of dissociated or covert REM processes. State dissociation purportedly explains a variety of bizarre clinical phenomena involving mentation, such as the symptoms of narcolepsy, REM sleep behavior disorder, disorders of arousal (e.g., sleep terrors, sleepwalking, sleep drunkenness), automatic behavior, and "out-of-body" experiences. In most of the cases discussed by Mahowald and Schenck, however, the state *into* which intrusions occur is of more importance in defining the phenomenon than is the state *from* which the isolated intrusions originate. For example, in the case of REM sleep behavior disorder, there is very little doubt that the REM sleep state is involved, whereas the precise origin of the isolated, intruding event (absence of muscle atonia) is of less importance to the definition of the syndrome. It may be a waking-state intrusion or some unspecified type of motor activation. In the case of covert REM sleep, identification of the state *from* which intruding events arise is of primary importance. Thus, the REM sleep processes that may intrude upon other states vary in complexity from, on the one extreme, the *absence* of a single defining component (as in the absence of eye movements during "skipped" first REM periods) to, on the other extreme, the *presence* of a single component in a NREM sleep state (as in the presence of eye movements during stage 2 sleep). It is validation of the latter type of event, involving the intrusion of single components, that is most at issue in **Solms**'s commentary; instances of the former type are more obviously variations of a known state. The problem of validating many such isolated physiological events as bona fide REM sleep dissociations will require more detailed scrutiny of the events' characteristics. To illustrate, Lavie (1990) describes episodes of penile tumescence without REM sleep in a patient with shrapnel fragments lodged in his left cerebellar hemisphere and prepontine cistern. Over five recording nights, this patient had a total lack of REM sleep on three nights, and only a single REM episode on each of the two others (REM% = 0.6 and 5.9%, respectively). The episodes of tumescence might thus seem to be "indeterminate," that is, completely unrelated to REM sleep. Nevertheless, closer scrutiny reveals that episodes of penile tumescence were recorded (1) that followed the expected temporal REM sleep rhythmicity of about 90 min (e.g., erections were spaced 82, 150, and 101 min apart on three recording nights), (2) that occupied portions of total sleep time that were similar to typical REM sleep times (35.5, 22.9, and 26.2% on the three nights), and (3) that were coincident with REM sleep on the two occasions that REM sleep was, in fact, detected. Lavie even concluded that "in spite of the drastic reduction of REM sleep, there was an indication of a 'REM-like' cyclicity in penile erections" (p. 278). To Lavie, the finding "supports the notion that nocturnal penile erections can be dissociated from REM sleep" (p. 278), a notion proposed earlier by Karacan and colleagues (Karacan 1982; Karacan et al. 1976).

To extend this notion even further, the *dissociability* of physiological processes during REM sleep may be speculated to be a basic feature of the state. **Antrobus** points out that the imaging results of Braun et al. (1998) reveal a high degree of dissociation among normally associated brain structures in REM sleep. The same is true of a wide variety of autonomic systems (Parmegianni 1994). Much cognitive literature (e.g., Hecker & Mapperson 1997; Livingstone & Hubel 1987) demonstrates how components of perception and memory can be experimentally dissociated, revealing that such information is processed in parallel along anatomically separate channels in the CNS. Dissociation of information may just be a necessary condition of dreaming which, as Foulkes (1985) proposes, must draw upon a diffuse pool of "dissociated elements of memory and knowledge" (p. 27). If REM sleep is at least partly about the dissociation of normally coupled systems in the service of reorganizing them for dream formation, then perhaps we should not be surprised to see such dissociations also occurring outside of the state.

Arguments about organization and isomorphism aside, differences between **Solms**'s model and my own may only constitute a difference in *interpretation* of findings. If a given process is reliably associated with a given sleep state, say with a concordance of 85–100%, and if that relationship is highly specific to that sleep state, then it would seem appropriate to consider the attribute as a biological marker of

the sleep state. But if the relationship is not specific to the sleep state, then its role as a marker is cast in doubt. It is the *degree* of specificity of the process to the state that will determine whether it is trusted to be a valid marker of the state. The covert model is an attempt to more precisely identify that degree of specificity for REM sleep.

To summarize, until isolated REM sleep signs occurring in NREM sleep can be confidently *excluded* as (1) being "linked" to REM sleep initiation or maintenance or (2) bearing some isomorphic relationship to sleep mentation variables, I am comfortable in viewing them as "dissociated" rather than "indeterminate" events. The interpretation of these signs depends heavily upon how the REM sleep state is conceptualized as well as upon what specific and/or general features of REM sleep prove to be isomorphic with sleep mentation.

NR6. Lack of evidence for isomorphism

At least six commentators (**Hunt; Kramer; Morrison & Sanford; Panksepp; Solms; Vogel**) referred to the lack of evidence for isomorphic relationships between physiological variables and sleep mentation, evidence that is critical in evaluating the covert REM sleep model. Although authoritative reviews of psychophysiological isomorphism such as those by Pivik (1991) are often taken as evidence that strongly refutes isomorphism, such reviews in fact offer ample evidence supporting some types of isomorphic relationships, and even some evidence supporting the covert REM sleep model. First, whereas there is inconsistency in many findings that bear on different classes of physiological variables in relation to mentation, some classes (e.g., autonomic) appear particularly strongly associated with sleep mentation variables. Variability in respiration rate has been observed to correlate with both quantitative (Shapiro et al. 1964) and qualitative (Hobson et al. 1965; Kamiya & Fong 1962; Van de Castle & Hauri 1970) aspects of sleep mentation. Hobson et al. (1965) even observed such relationships in both REM and NREM sleep. Other autonomic indicators, such as sudden penile erections, have also been found to be associated with increased recall (Karacan 1966) and erotic content (Fisher 1966). In NREM sleep, including stages 2, 3, and 4, both the recall and hallucinatory quality of mentation has been found to be higher on awakenings that follow brief phasic inhibitions of the H-reflex (Pivik 1971). Sleep onset has also yielded associations between EEG theta bursts on the one hand and visual imagery and discontinuity on the other (Pope 1973). The physiological measures in NREM sleep (respiration variability, H-reflex inhibition, theta bursts), by virtue of their similarity to REM sleep phenomena, are good candidates for indicators of covert REM sleep processes. Note that this holds true for both stage 2 sleep and SWS. As I specified in the target article, one reason that isomorphic relationships between physiological and sleep mentation variables have not been more robust may be because methods for analyzing combinations of such variables in coherent groupings have not been available. Studies that are able to simultaneously consider variations in respiration, penile tumescence, EMG inhibition, and other autonomic indicators may well prove to demonstrate more reliable isomorphic relationships with sleep mentation at different levels of complexity.

NR7. Elimination of REM sleep does not eliminate dreaming

Two commentators (**Bosinelli & Cicogna; Panksepp**) and a target article (**SOLMS**) suggest that the covert REM sleep model is inconsistent with the demonstration (Solms 1999b) that elimination of REM sleep does not necessarily eliminate dreaming. This contention depends crucially on whether REM sleep can, in fact, be eliminated as claimed. **HOBSON ET AL.** suggest in their target article that it cannot. They suggest, on the basis of proven difficulties in experimentally suppressing REM sleep with pontine lesions in animals, that any lesion capable of destroying the pontine REM sleep generator in humans would have to be so widespread so as to eliminate consciousness altogether. Solms (1999b) himself conceded this point at a recent symposium on the neurophysiology of sleep.

Repeated polysomnograpy over many nights would be crucial to determining the presence or absence of REM sleep or, more precisely perhaps, the degree of presence of REM sleep. This was amply demonstrated by the case of purportedly suppressed REM sleep described in section NR5 (Lavie 1990). The subject of this case study had severely reduced REM sleep, but it was found to be totally absent on only three out of five recording nights. Experimental awakenings from sleep in subjects like this, who suffer from brainstem lesions and reduced REM sleep, could serve as a critical test of the covert REM sleep model. Subjects' sleep records could be examined for evidence of residual REM sleep events, even in the absence of stage REM sleep as traditionally scored. As Lavie's paper demonstrated, REM sleep signs can be detected in the absence of the full-blown REM sleep state.

NR8. The model needs validation

I agree wholeheartedly with commentators (**Blagrove; Conduit et al.; Franzini; Gottesmann**) calling for validation of the covert REM sleep model. I think that the **NIELSEN** target article, many of the excellent points raised in the commentaries, and this reply to the commentaries together suggest straightforward ways in which such validation could proceed:

1. Replication of early unreplicated findings demonstrating state overlap in NREM sleep (Schwartz 1968) and at sleep onset (Toth 1971).

2. Extension of previous studies that have examined percent and type of NREM mentation recall as a function of preceding REM sleep characteristics. Time since previous REM sleep has been evaluated in several studies, but time in previous REM sleep, intensity of previous REM sleep, propensity for previous REM sleep, and so on, have not (although see results of Study 1 in sect. NR8.1).

3. Assessment of clinical phenomena in which vivid NREM dreaming occurs (e.g., stage 2 nightmares) for evidence of covert REM processes.

4. Replication of recent findings concerning the effects of during-sleep stimulation on dreaming, for example, Conduit et al.'s (1997) finding that stimulation in NREM sleep increases recall of mentation.

5. Examination of EEG parameters for evidence of brief state shifts (Lehmann & Koukou 1984) and REM sleep-like

intrusions, for example, brief EEG desynchronizations in NREM sleep.

6. Use of topographic mapping to determine simultaneous activation of NREM and REM signs in NREM sleep (e.g., central vs. frontal leads).

7. Examination of continuous delta power profiles for evidence of reduced delta and/or rapid delta fluctuations during the covert REM sleep of "missing" first REM periods (cf. **Feinberg**).

8. Exploration of covert REM sleep signs during REM sleep deprivation (cf. **Cartwright**).

9. Effects of measurements taken at home versus in the laboratory on NREM mentation; does the laboratory environment induce covert REM sleep processes?

10. Architectural assessment of covert REM signs (e.g., penile tumescence, eye movements, EMG bursts) in relation to mentation recall: do they conform to a 90-min ultradian rhythm? Is their duration from 20–25% of TST? Are they in close proximity to an overt REM sleep episode? Are they concordant with other REM signs (eye movements, phasic muscle activity, heart rate or respiratory variability, etc.)?

11. Assessment of REM-NREM content differences in subjects highly trained in introspection.

12. Effects of experimenter bias, subject verbosity, speed of awakening, and so on, on frequency and complexity of NREM mentation reports.

I undertook preliminary validation of the model in two studies that address the first three of these considerations. One study was designed to assess correlations between the amount of mentation recalled following awakenings from stage 2 sleep and the simple duration of immediately preceding REM and NREM sleep stages. The second study was an exploratory assessment of a sample of sleep records from both normal and sleep-disordered subjects for evidence of signs of covert REM sleep in NREM sleep. I briefly describe these studies below.

NR8.1. Study 1: Is stage 2 mentation associated with prior duration of REM and NREM sleep?

To test whether the amount of mentation recalled from stage 2 sleep would be associated with longer durations of prior REM and/or NREM sleep, we drew upon a sample of 26 healthy control subjects (20W, 6M; Mean age = 25.7 ± 6.5 years, range: 18–42) who in a previous study (Faucher et al. 1999) had been awakened from REM and stage 2 sleep to report mentation. We identified all stage 2 awakenings for which there had also occurred a preceding, uninterrupted REM sleep episode (N = 74). A trained polysomnographer scored the sleep records for two variables: (1) time in prior REM sleep, and (2) time in prior stage 2 sleep (stage 2 onset to point of awakening), according to the standard criteria (Rechtschaffen & Kales 1968). Another judge counted the number of relevant, nonredundant words in each mentation report from which total word count (TWC) and log (TWC + 1) were calculated. Correlations were calculated for the entire sample of 74 (N = 26 subjects) and for a reduced sample of 34 reports (N = 18 subjects) that excluded all TWC scores that were equal to zero.

TWC and log (TWC + 1) scores gave similar patterns of results (Table NR2). Correlations only partly supported the hypothesis that proximity to a prior REM episode ("prior stage 2 duration") would be associated with lengthier stage 2 mentation reports. Duration of prior stage 2 sleep correlated negatively with TWC $r = -.315$, $p = .069$) and log (TWC + 1) ($r = -.420$, $p = .014$) when zero-recall reports were excluded, but not when they were included (both p = NS). Further, duration of the prior REM sleep episode was positively correlated with report length whether zero-recall reports were included ($r = .380$; $p = .0008$) or not ($r = .373$, $p = .030$). This did not seem to be due to a circadian phase effect (i.e., longer REM episodes occurring later at night) because correlations between the clock time of REM episode onset and TWC were negligible ($r = .097$ and .118) for the two samples (both p = NS).

Table NR2. *Correlations between total word count (TWC) and duration of prior REM and NREM sleep episodes*

	TWC r (p)	Log_{10} (TWC+1) r (p)
Reports with WC≥0 (N=74)		
Prior REM duration	+0.380 (*.001*)	+0.335 (*.004*)
Prior stage 2 duration	−0.138 (*.243*)	−0.033 (*.789*)
Reports with WC>0 (N=34)		
Prior REM duration	+0.373 (*.030*)	+0.255 (*.145*)
Prior stage 2 duration	−0.315 (*.069*)	−0.420 (*.014*)

These analyses thus partly support predictions of the covert REM sleep model replicate the findings of several previous studies demonstrating greater recall with closer proximity to REM sleep (see **NIELSEN** sect. 3.4 "*Proximity of NREM sleep awakenings to REM sleep*"). They are also the first to suggest that parameters of a prior REM sleep episode *other* than its proximity might influence NREM mentation. Whether the REM duration measure in the present study reflects heightened REM pressure (due to awakenings for mentation recall from other REM episodes) or to some other factor has yet to be determined. However, the findings together are consistent with the possibility that the presence and degree of elaboration of stage 2 sleep mentation is affected by interactions between prior REM and stage 2 sleep processes. Specifically, the present results suggest that the *duration* of a prior REM episode may determine whether or not content will appear in a subsequent stage 2 episode, but that the stage 2 episode's *proximity* to this REM episode may determine the degree of elaboration of that content, given that it is present.

NR8.2. Study 2: Do signs of covert REM sleep appear in NREM sleep?

To examine whether REM sleep signs appear at sleep onset and in NREM sleep more generally, a polysomnographer with six years of full-time experience using the Rechtschaffen and Kales (1968) criteria evaluated a series of 35 records from 20 subjects (11W, 9M; mean age = 32 ± 11.6) for evidence of rapid eye movements and other signs in NREM sleep. Eight of these subjects (5W, 3M; mean age = 29 ± 12.5) were healthy controls, seven (3W, 4M; mean age = 27.6 ± 5.4) were patients consulting for idiopathic nightmares (INM), and five (3W, 2M; mean age = 44.6 ± 8.4) were patients consulting for post-traumatic nightmares (PTNM). The polysomnographer used Schwartz's (1968) criteria for scoring slow, medium fast, and rapid eye move-

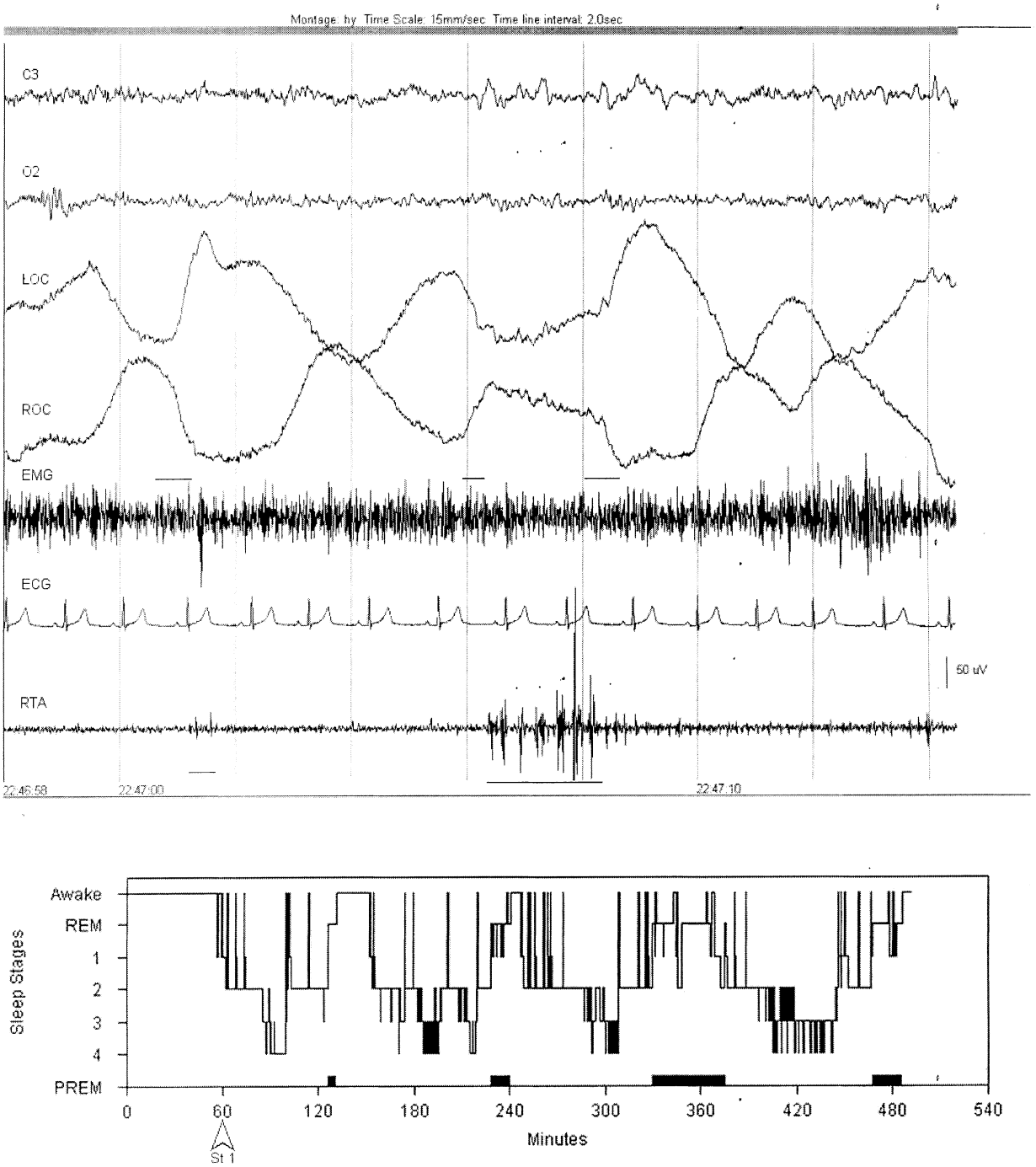

Figure NR3. Hypnogram and polysomnographic (PSG) tracing from a 24-year-old male patient with long-term idiopathic nightmares (INM). Medium fast and rapid eye movements are visible in this sleep onset stage 1 epoch, with phasic tibialis activation occurring between two bursts. C3: C3/linked ears; O2: O2/linked ears; LOC: left ocular; ROC: right ocular; EMG: chin muscle activity; ECG: bipolar cardiac; RTA: right tibialis anterior. Vertical grey lines indicate 2 second intervals.

ments as a guide only, since the latter criteria were not found to be precise enough to apply systematically. For example, the duration criteria for the three types are *slow:* 1.0 to 4.0 sec; *medium fast:* 0.25 to 2.0 sec; and *rapid:* 0.2 to 1.5 sec.

Of the 20 subjects, 12 (60%) showed at least one clear example of covert REM signs either at sleep onset (No. events = 13) or during later stage 2 or 3 sleep (No. events = 16). Examples were noted in 4 of 8 (50.0%) control subjects, 4 of 7 (57.1%) INM patients, and 4 of 5 (80.0%) PTMN patients. They occurred in 6 of 11 (54.5%) women and 6 of 9 (66.7%) men. Events were found more often in stage 2 sleep (17/30 or 56.7%) than in stage 1 sleep (12/30 or 40.0%), stage 3 sleep (1/30 or 3.3%) or stage 4 sleep (0/30 or 0.0%). More events occurred shortly after (23/30 or 76.7%) rather than before (2/30 or 6.7%) an episode of wakefulness, and before (4/30 or 13.3%) rather than after (1/30 or 3.3%) an episode of REM sleep. Some examples of these REM sleep events with their corresponding hypnograms appear in Figures NR3 to NR8 (see also Fig. NR2).

Figures NR3 and NR4 are taken from a 24-year-old male

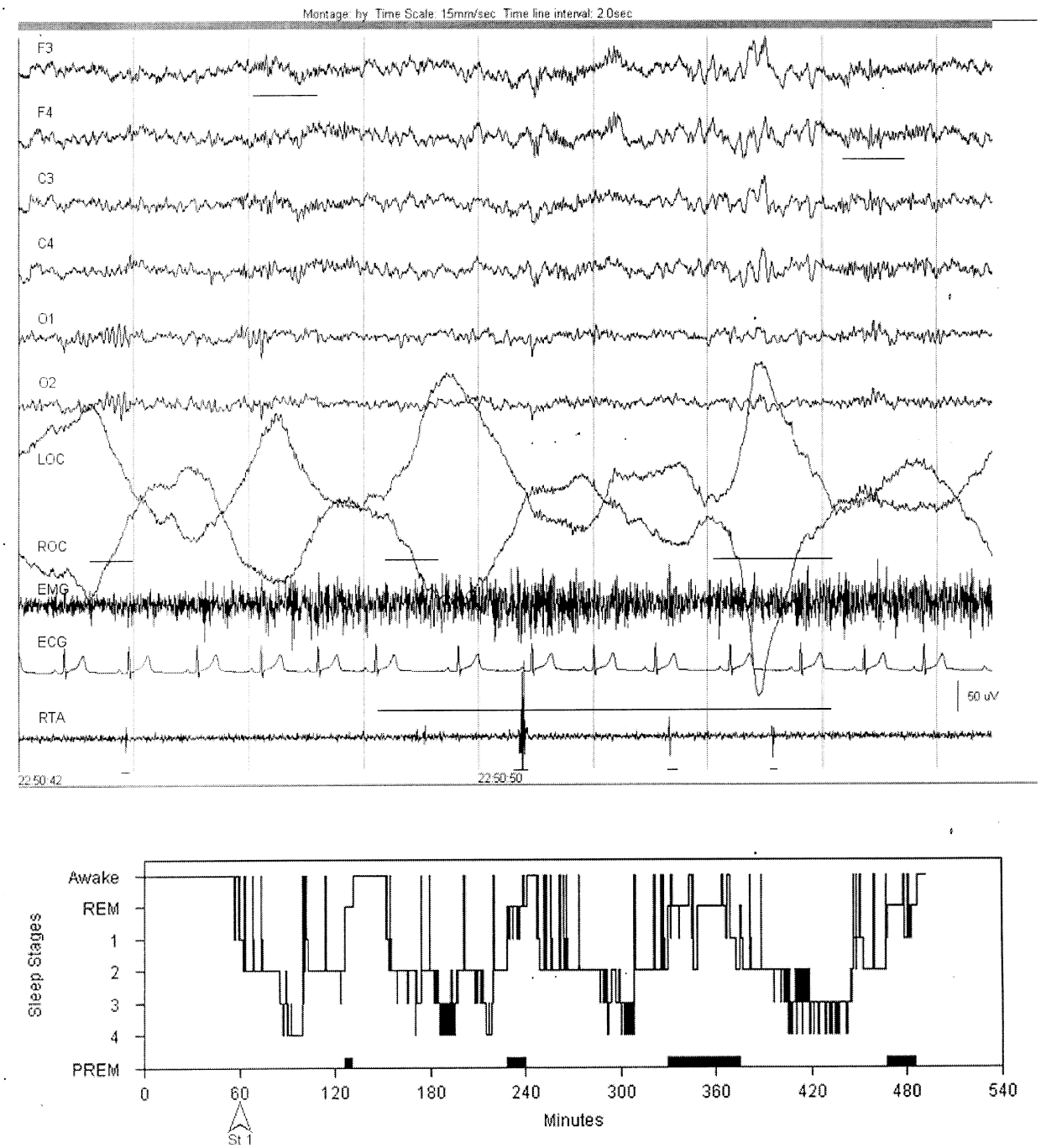

Figure NR4. Hypnogram and PSG tracing from same patient as in Figure NR3. The tracing occurred within 4 min of the previous one. A mixture of slow, medium fast, and rapid eye movements can be seen. Phasic tibialis EMG is also evident as is REM sleep-like cardiac variability on the ECG. Spindles are clear in the EEG. Legend as in Figure NR3 with addition of F3, F4, C4, and O2 all referenced to linked ears.

INM patient. These tracings occurred within 4 min of each other only minutes after initial sleep onset. They illustrate a mixture of slow, medium fast, and rapid eye movements occurring within the same eye movement bursts. A given eye movement may be medium fast or rapid in one direction yet slow in the other. Further, these eye movement bursts are accompanied by REM sleep-like phasic tibialis muscle bursts (both Figures) and abrupt cardiac variability, as well as by spindling in the EEG (Fig. NR4).

Figure NR5 is taken from a 25-year-old female patient with INM. It displays a section of stage 1 sleep shortly after a long episode of wakefulness in the sleep onset period. Rapid and medium fast eye movements again occur in the same eye movement burst. Spindles are also present.

Figure NR6 is a section of late night stage 2 sleep from a 43-year-old male PTNM patient. This patient had the highest number of identified REM sleep signs (3 at sleep onset; 9 in late night NREM) out of the entire sample and had a highly fragmented hypnogram in general. He also reported dreaming vividly throughout the night, every night. A phasic EMG burst of chin muscle activity and a single rapid eye movement occur amidst several stage 2 sleep spindles in the

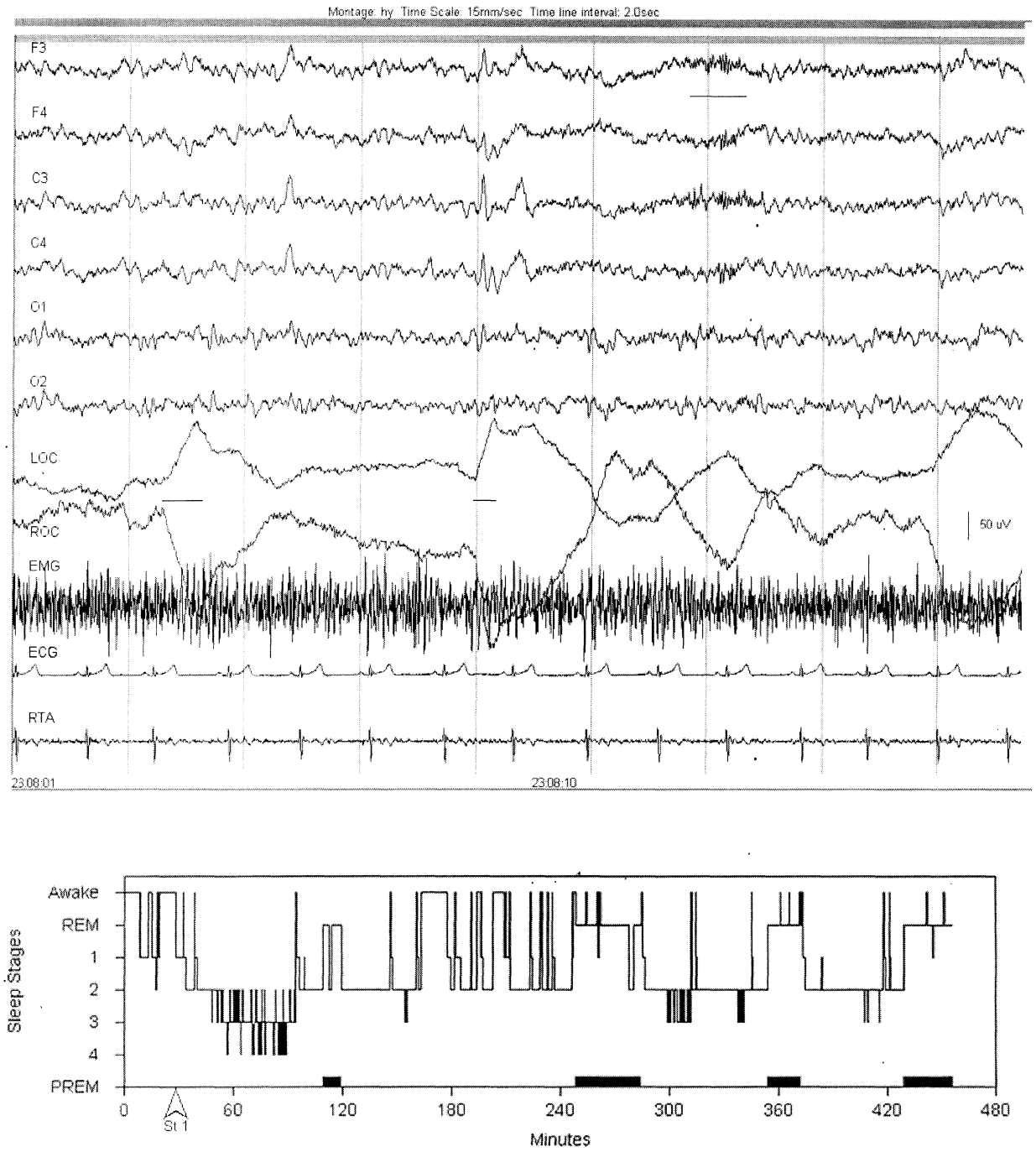

Figure NR5. Hypnogram and PSG tracing from 25-year-old female with INM. A section of stage 1 sleep with spindling at sleep onset contains both medium fast and rapid eye movements in the same eye movement burst. Legend as in Figure NR4.

tracing. This patient displayed a second such event 9 min later, just prior to an apparently aborted REM sleep episode.

Figure NR7 is a section of stage 2 sleep from the same patient as in Figure NR6 but on the following night and transpiring less than 10 min after a lengthy REM sleep episode. The tracing shows medium fast and rapid eye movements, one of which occurs in exact synchrony with a sleep spindle. This type of synchrony suggests that inhibitory influences associated with sleep spindles (see **Porte** commentary) may be less generalized than is thought.

Figure NR8 is taken from a 30-year-old female INM patient. It illustrates a burst of medium fast-to-rapid eye movements coincident with a 5-sec burst of chin muscle activity against a background of relatively quiescent EMG in stage 3 sleep. This event occurred several minutes prior to a brief awakening.

This study was not undertaken to prove that eye movements and other REM sleep signs observed in NREM sleep are frequent enough to account for all the observed sleep mentation reported in this stage, although the correspondence between the fact that 50% of normal subjects had such signs and that recall of NREM sleep mentation is about 50% on average (see target article) should be noted. Rather, it was intended simply to raise doubts in a concrete

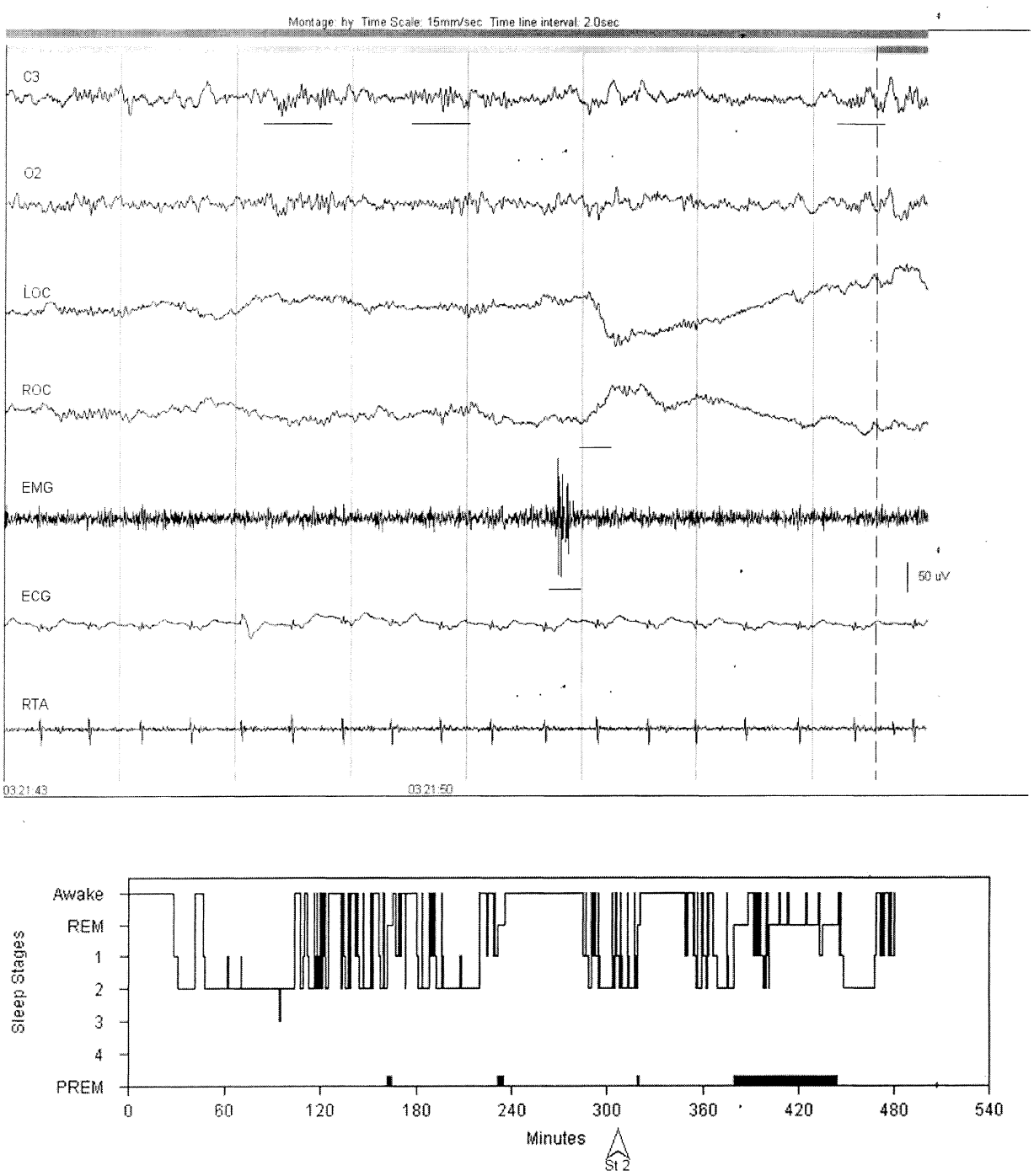

Figure NR6. Hypnogram and PSG tracing of late night stage 2 sleep from 43-year-old male post-traumatic nightmare (PTNM) patient. This patient had the most REM sleep signs of the entire sample and a fragmented sleep hypnogram on both nights (see Fig. NR7). He also reported dreaming vividly throughout the night, every night. A phasic EMG burst of chin muscle activity and a single rapid eye movement occur with stage 2 sleep spindles. A second similar event occurred 9 min later, just prior to an apparently aborted REM sleep episode. Legend as in Figure NR3.

fashion as to whether REM and NREM sleep states are as completely distinct as commonly thought. The findings together do suggest that: (1) REM sleep events are common enough in NREM sleep that they warrant more careful study with more sensitive recording equipment (e.g., higher sensitivity eye movement detectors); (2) sleep onset, in particular, often resembles REM sleep, if only for brief intervals, with some of the standard scoring criteria absent; (3) covert REM signs occur in normal subjects but more frequently in sleep-disordered patients; and (4) covert REM signs are closely linked to prior wakefulness, and to *subsequent* (more so than to *preceding*) REM sleep. The importance of the last point is that subsequent REM sleep episodes are technically very difficult to predict and thus are very likely to affect NREM mentation reports.

If, as this study suggests, readily measurable peripheral signs of REM sleep occur with some regularity in NREM sleep, then there should be even more reason to suspect that *less* easily measurable peripheral and central signs of REM sleep may also be active outside of their normal

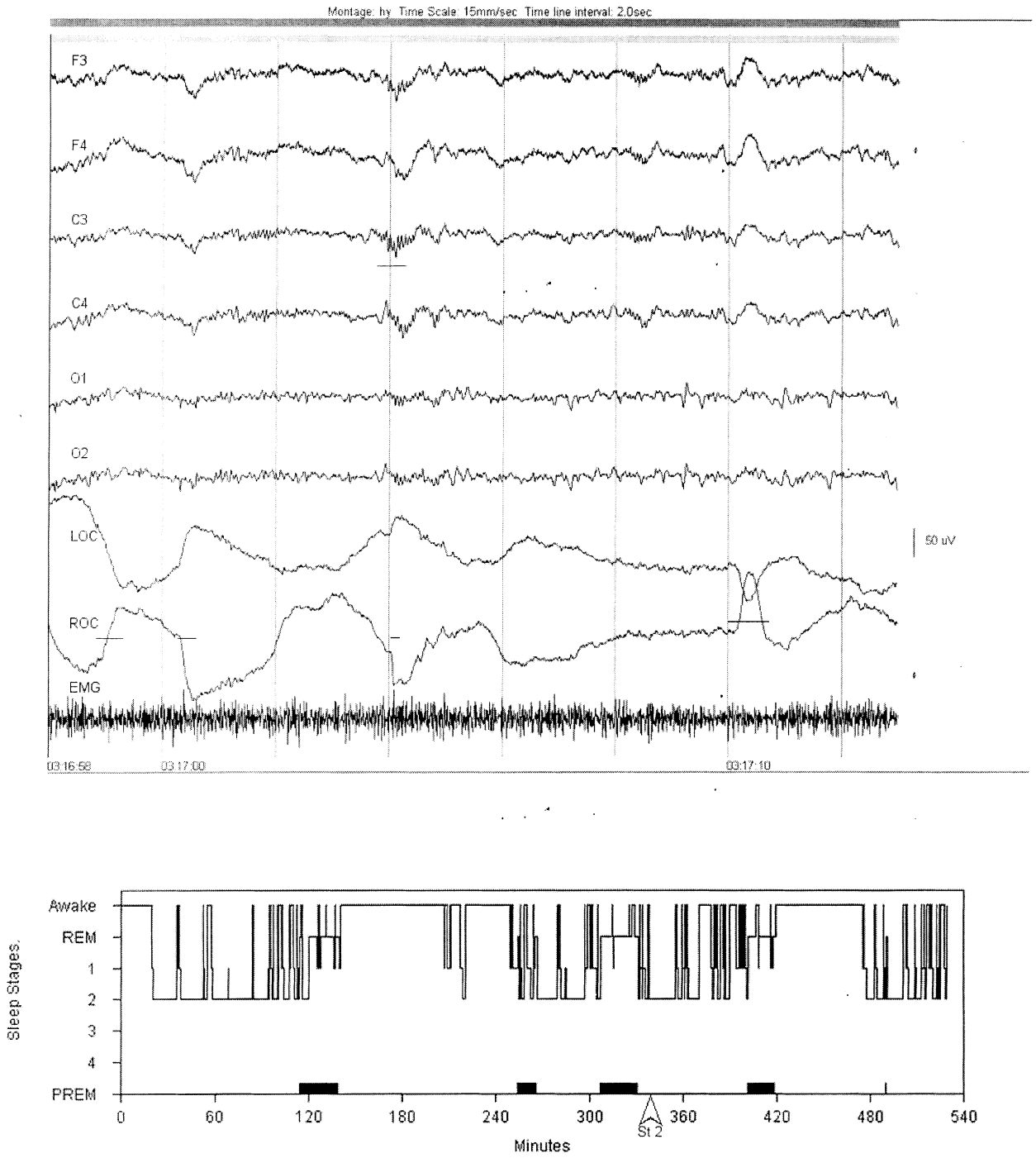

Figure NR7. Hypnogram and PSG tracing of stage 2 sleep from same patient as in Figure NR6 but on the following night. The epoch occurs less than 10 min after a lengthy REM sleep episode. Medium fast and rapid eye movements are visible; one of these occurs in exact synchrony with a sleep spindle. Legend as in Figure NR4 minus RTA.

boundaries. There is a multiplicity of physiological systems participating in the chaos of REM sleep but only a fraction of these are ever monitored. In fact, many such processes may manifest sporadically during NREM sleep even when *none* of the standard criteria for REM sleep are visible. In particular, important changes in a variety of autonomic effector systems in REM sleep (Parmeggiani 1994) are often technically difficult to measure, yet these seem particularly pertinent to assessing emotional features of sleep mentation that might become dissociated from REM sleep (cf. **Panksepp**).

NR9. Conclusion

The covert REM sleep model can be seen to be an instance of one of four alternative viewpoints on the sleep mentation question, each of which makes a different combination of assumptions concerning (1) mind-body isomorphism and (2) the presence of one versus two mentation generators (see Table NR2). Isomorphism with a 1-gen assumption describes the covert REM sleep processes model. Isomorphism with a 2-gen assumption describes the activation-synthesis and AIM models, while non-isomorphism with

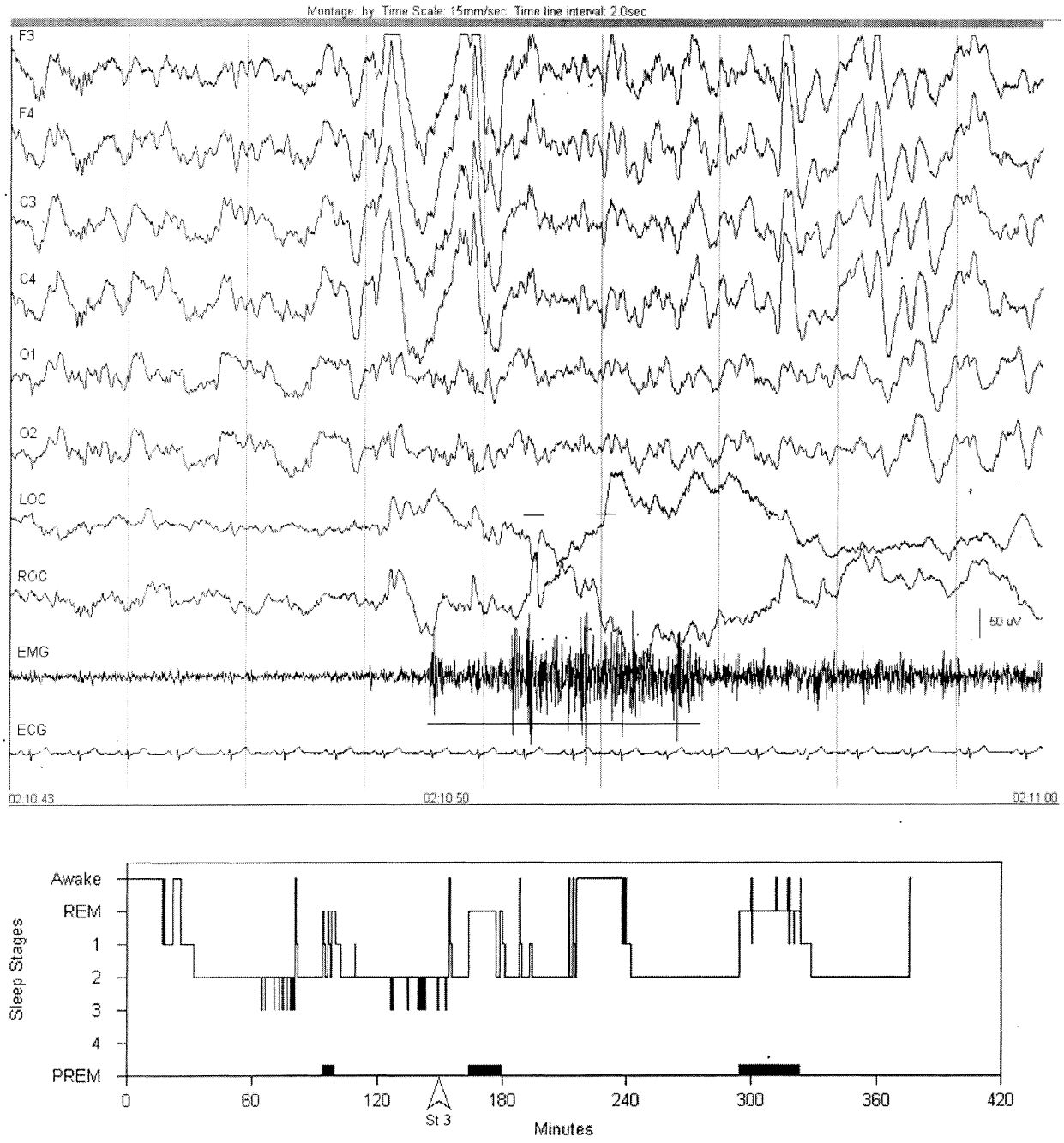

Figure NR8. Hypnogram and PSG tracing of stage 3 sleep from a 30-year-old female INM patient. A burst of medium fast-to-rapid eye movements coincides with a 5-sec burst of chin muscle activity against a background of quiescent EMG. A brief awakening occurred several minutes later. Legend as in Figure NR4 minus RTA.

1-gen and 2-gen assumptions describe Foulkes's model and models such as that proposed by Casagrande, respectively. There is in all likelihood room for models that take intermediate positions on these two basic assumptions. For example, commentators such as **Cavallero, Bosinelli & Cicogna,** and **Feinberg** acknowledge a limited role for cortical activation in initiating sleep mentation, but they do not appear to subscribe to isomorphism beyond this general level. Because so little is known about mind-body isomorphism, it would be premature to exclude consideration of such models.

If *both* strict isomorphism and a 1-generator mechanism are true assumptions, then so also is the covert REM sleep model true *in some form.* By this I mean that some uniform set of physiological isomorphs exists that is reliably correlated with sleep mentation – regardless of sleep state. In fairness to the most adamant critics of the covert model, such physiological variables *need not* be the dissociated REM sleep processes that I propose. They may prove to be much subtler patterns of neural coding that have little to do with the overt measures that we routinely record from surface electrodes. Some examples are discussed in Helekar (1999). They may even be active during much of the waking state. Then again, it may prove to be convenient to adopt a REM sleep-related nomenclature if only because these variables will likely be more typical of REM than of NREM sleep, that is, they will be more prevalent, more fre-

Table NR3. *Models of sleep mentation necessitated by different assumptions about isomorphism and number of mentation generators*

	1-generator true	2-generator true
Isomorphism false	A. One factor mnemonic activation model (Foulkes and others) or equivalent	B. Two-factor psycholinguistic model (Casagrande and others) or equivalent
Isomorphism true	C. Covert REM sleep processes (Nielsen and others) or equivalent	D. Activation-synthesis and AIM models (Hobson, McCarley, and others) or equivalent

quent, and more intensely activated in REM sleep than they will in NREM sleep – or in the waking state for that matter. This fact, the regular association of vivid imagery with REM sleep, still remains as the legacy of last century's neurobiologically driven dream research, regardless of the convincing demonstrations of sleep mentation in NREM sleep. However, a definitive explanation of dreaming awaits a much more detailed understanding of what constitutes REM and NREM sleep, and of precisely how mind and body are inter-related as these states surge, recede, dissociate, and blend together across the sleep/wake cycle.

NOTES

1. I prefer the term "subjective experience" (cf. Helekar 1999) to "conscious experience" and especially to "subjective conscious experience" in the case of sleep mentation because the manner in which dreaming is "conscious" vis-à-vis waking consciousness has not been clearly articulated (although cf. Kahan & Laberge 1996).

2. This kind of explanation is very difficult to evaluate because verbatim mentation reports are only rarely published.

REM sleep is not committed to memory

Robert P. Vertes[a] and Kathleen E. Eastman[b]

[a]*Center for Complex Systems, Florida Atlantic University, Boca Raton, FL 33431;* [b]*Department of Psychology, Northern Arizona University, Flagstaff, AZ 86011.* **vertes@walt.ccs.fau.edu** **k.eastman@nau.edu**

Abstract: We believe that this has been a constructive debate on the topic of memory consolidation and REM sleep. It was a lively and spirited exchange – the essence of science. A number of issues were discussed including: the pedestal technique, stress, and early REMD work in animals; REM windows; the processing of declarative versus procedural memory in REM in humans; a mnemonic function for theta rhythm in waking but not in REM sleep; the lack of cognitive deficits in patients on antidepressant drugs that suppress or eliminate REM sleep; the disposition of conscious (dreams) and nonconscious material of REM sleep; and finally our theory of REM sleep. Although our position was strongly challenged, we still hold that REM sleep serves no role in the processing and consolidation of memory.

VR0. Seeds of our target article

Several years ago I (**VERTES**) carried out a series of studies in behaving rats examining the relationship between the activity of cells of the pontine reticular formation (PRF) and the theta rhythm of the hippocampus. I showed that the discharge of a subset of PRF neurons was highly correlated with theta rhythm of waking and REM and subsequently that these PRF cells are directly involved in the generation of the theta rhythm.

Prior to recording, I deprived rats of REM sleep in order to increase the amount of time spent in REM sleep (i.e., REM rebound) during subsequent recording sessions. Rats were deprived of REM for 24–36 hours using the pedestal technique. Although my sole purpose for using REMD was to boost REM during recording periods, I was surprised to observe that even 24 h of REMD produced severe detrimental effects on the rats. The rats were cold and often still wet from having fallen in the water, physically fatigued from balancing on the small diameter surface of the inverted flower pot, tired from a considerable lack of sleep (mostly REM, but both SWS and REM), and generally debilitated (much like we would be without sleep for 1–2 days). Although rats are reportedly hyperactive following REMD, I found that they were essentially immobile for at least 6 h post REMD. This experience led me to question the validity of experiments examining the effects of REMD on learning and memory; that is, if rats were so severely incapacitated following this procedure how could they adequately perform on behavioral tasks following REMD?

In 1995, Peter Shiromani asked me to participate in a forum on sleep and memory for Sleep Research Society (SRS) Bulletin. I agreed and indicated that I would be taking the "con" position: no relationship between REM sleep and memory. Of eight participants in the forum, I was the only one taking this position. Possibly based on my article in SRS Bulletin, Mike Chase invited me to participate in a debate with Carlyle Smith on this same topic at an international workshop on sleep and cognitive function sponsored by the World Health Organization in Cancun, Mexico, in 1999. The debate was fruitful and further fueled my interest in the issue of memory consolidation and REM sleep. The target article by my colleague and me developed from this background.

VR1. Early REMD studies in animals, the pedestal technique, and stress

As we discussed in our target article, there was an intense interest in the role of REM sleep in memory consolidation in the 1960–1970s, interest waned in the 1980s, and has recently resurfaced. This is now a lively topic in the sleep field. As we previously indicated, our coverage of the early REMD work in animals was not meant to serve as a detailed analysis of this area, but rather to convey a general sense of the net contribution of this work to an understanding of the possible involvement of REM sleep in memory consolida-