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

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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-

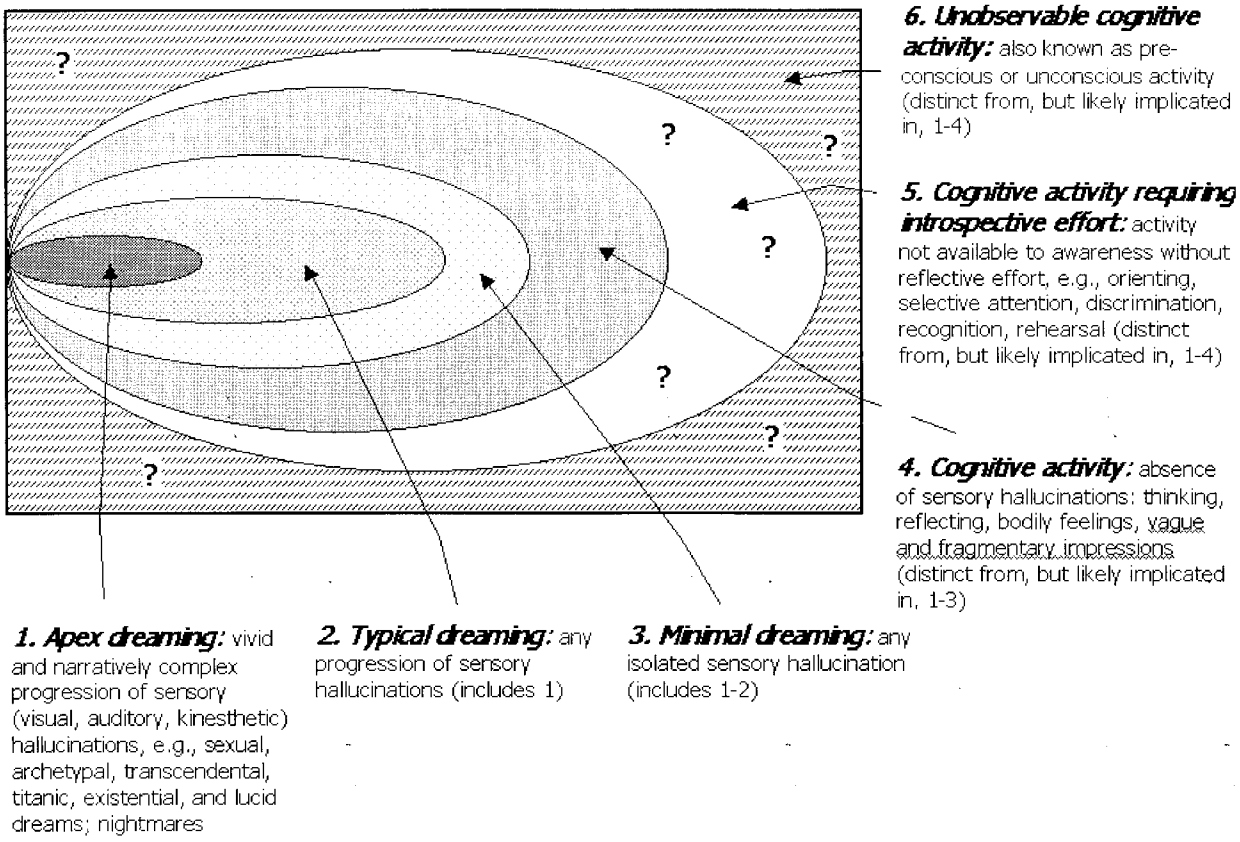

Figure NR1. Levels of specificity in defining sleep mentation – revised version of Figure 1 from target article. See text for details.

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).

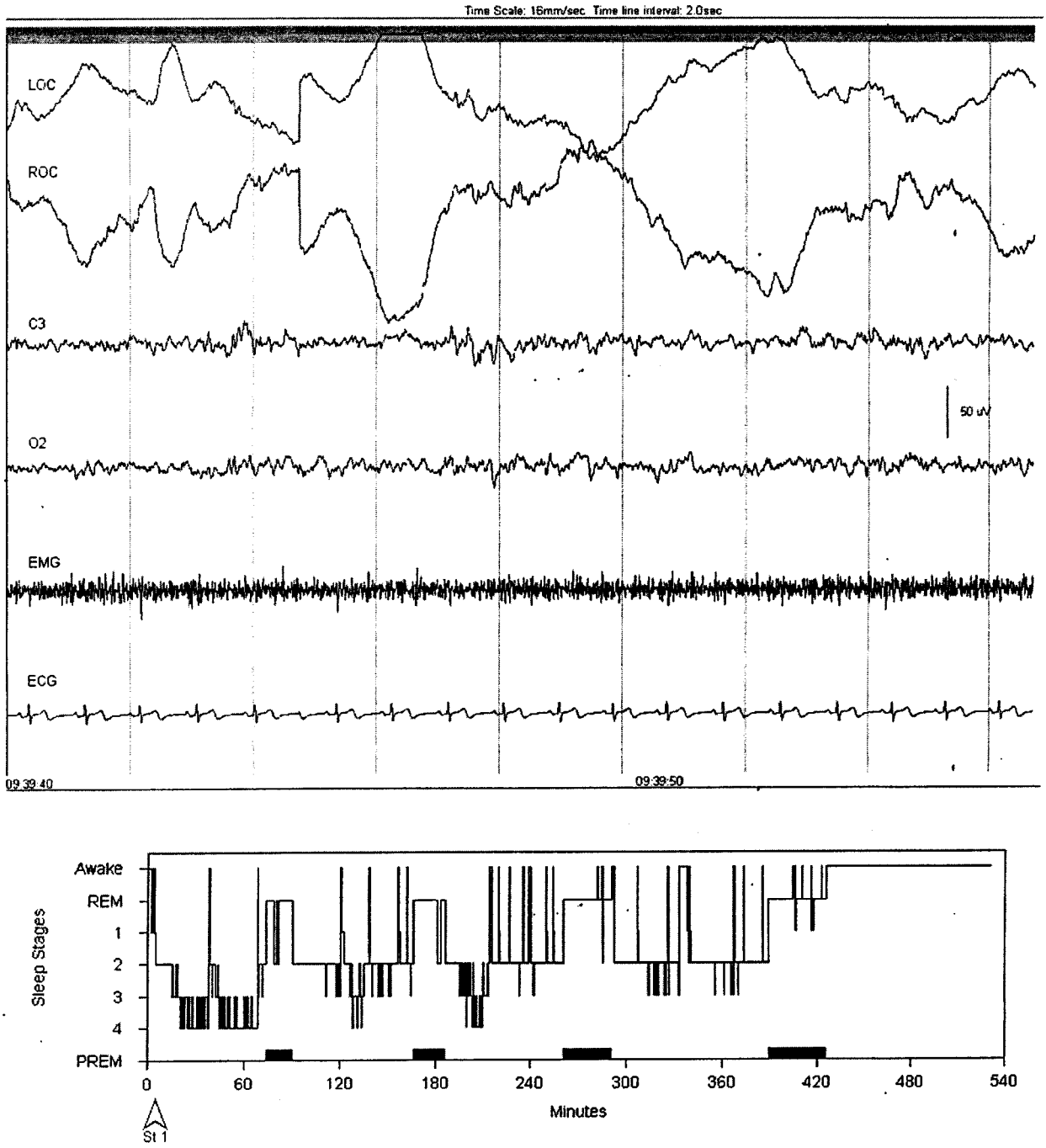

Figure NR2. Hypnogram and polysomnographic (PSG) tracing from a healthy 31-year-old female subject on her first recovery (daytime) sleep after enduring a 40-hour constant routine. Rapid, medium fast, and slow eye movements are clearly visible against a background of stage 1 EEG and EMG.

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-

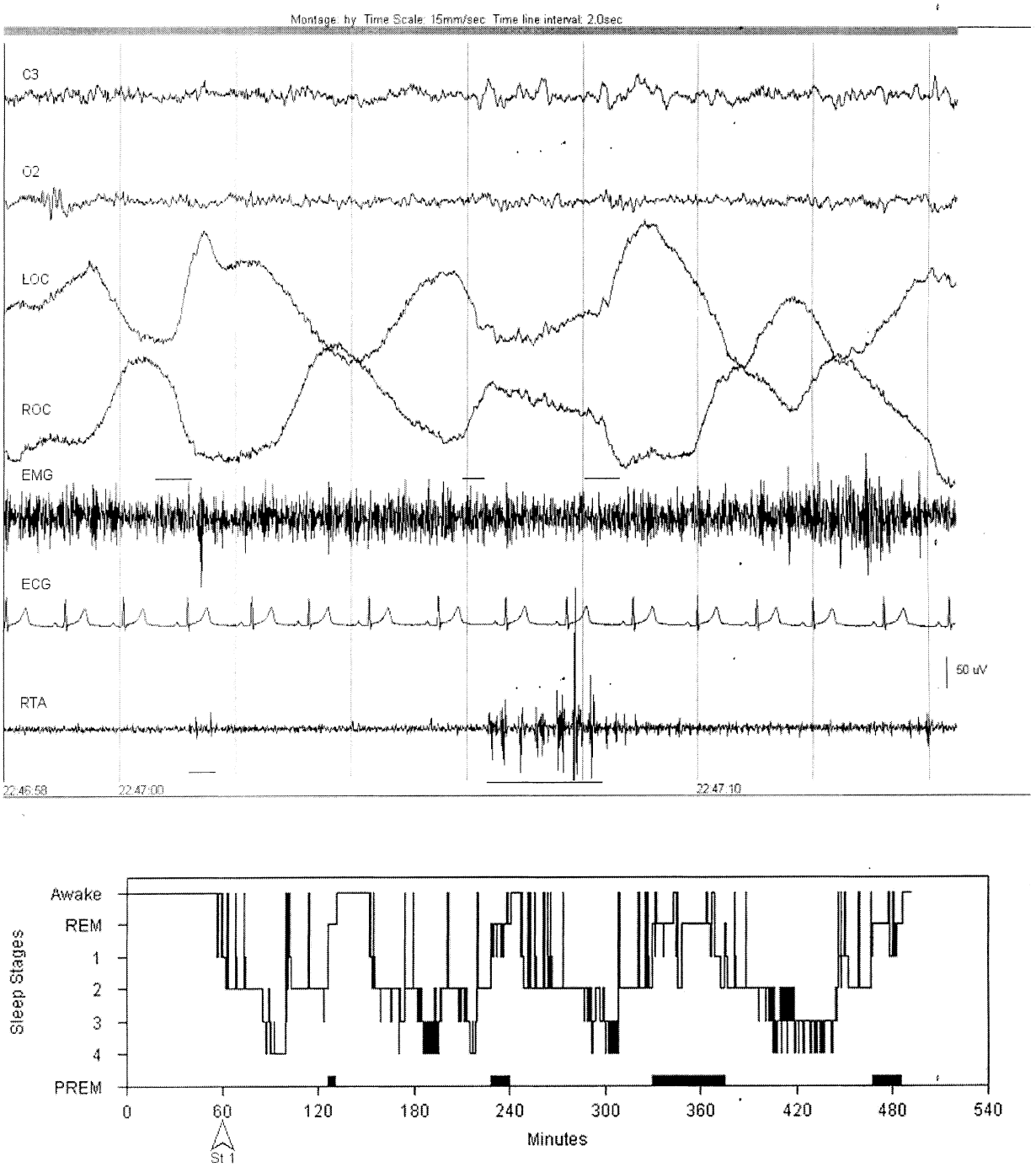

Figure NR3. Hypnogram and polysomnographic (PSG) tracing from a 24-year-old male patient with long-term idiopathic nightmares (INM). Medium fast and rapid eye movements are visible in this sleep onset stage 1 epoch, with phasic tibialis activation occurring between two bursts. C3: C3/linked ears; O2: 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

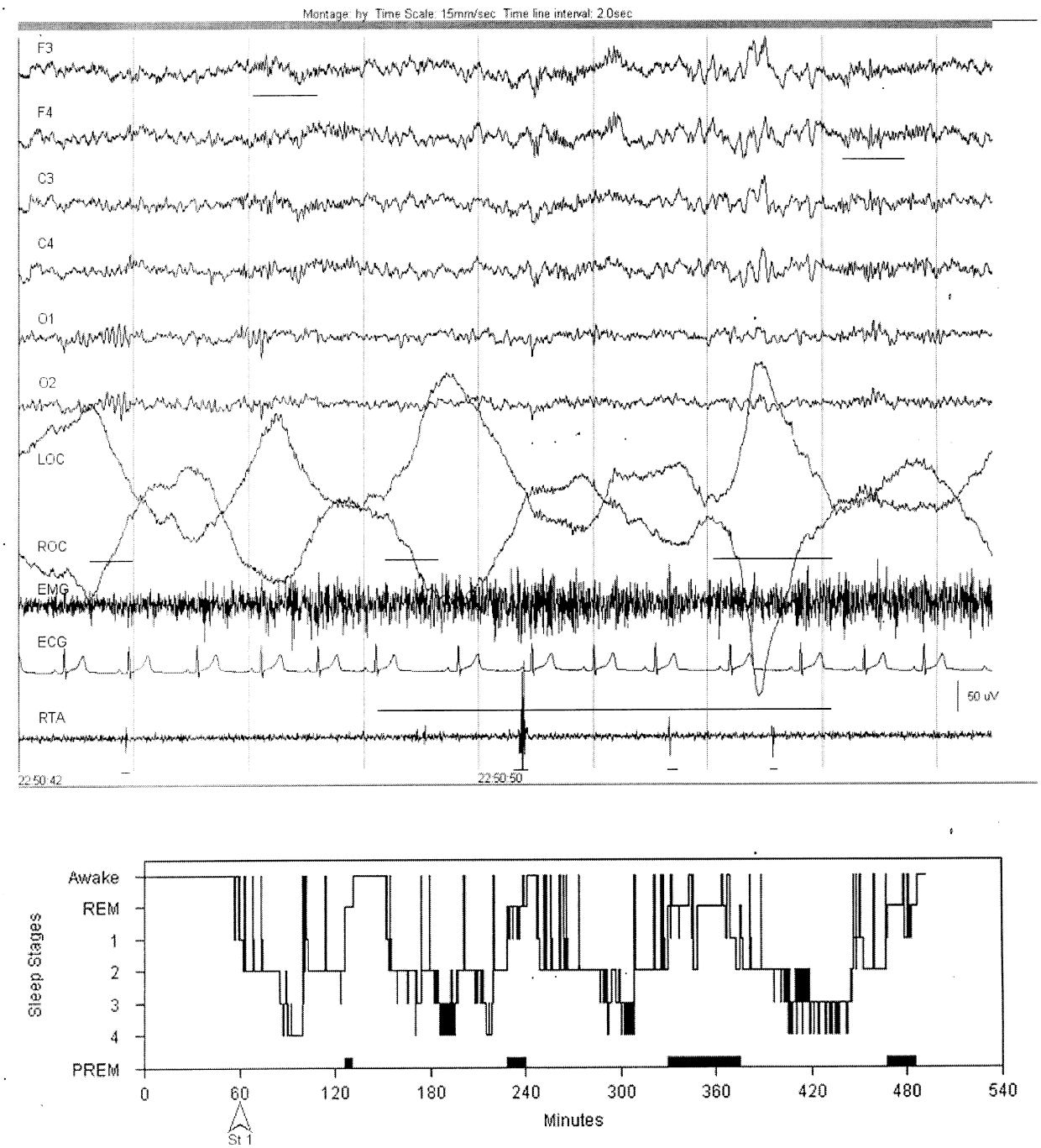

Figure NR4. Hypnogram and PSG tracing from same patient as in Figure NR3. The tracing occurred within 4 min of the previous one. A mixture of slow, medium fast, and rapid eye movements can be seen. Phasic tibialis EMG is also evident as is REM sleep-like cardiac variability on the ECG. Spindles are clear 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

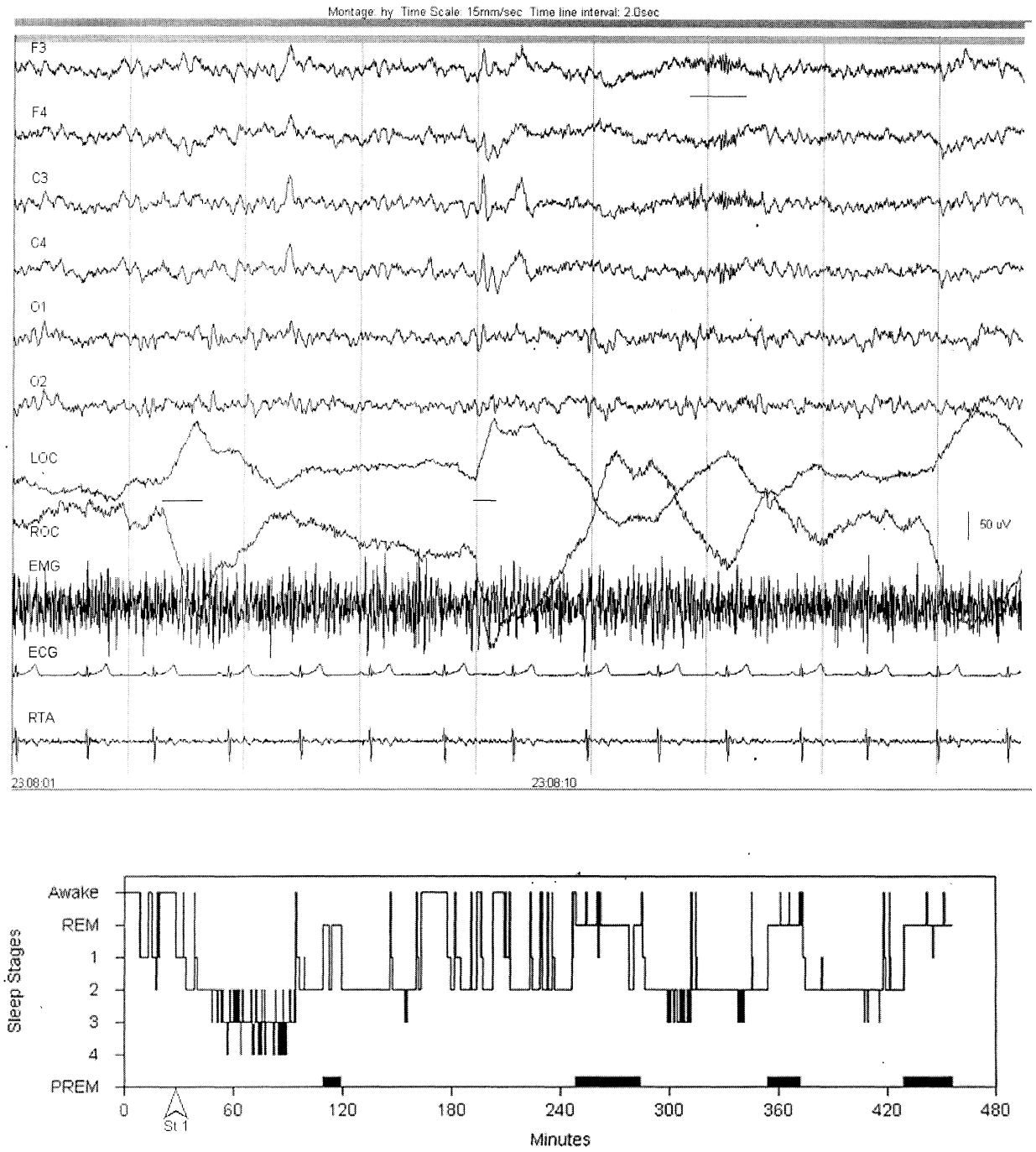

Figure NR5. Hypnogram and PSG tracing from 25-year-old female with INM. A section of stage 1 sleep with spindling at sleep onset contains both medium fast and rapid eye movements in the same eye movement burst. Legend as in Figure NR4.

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

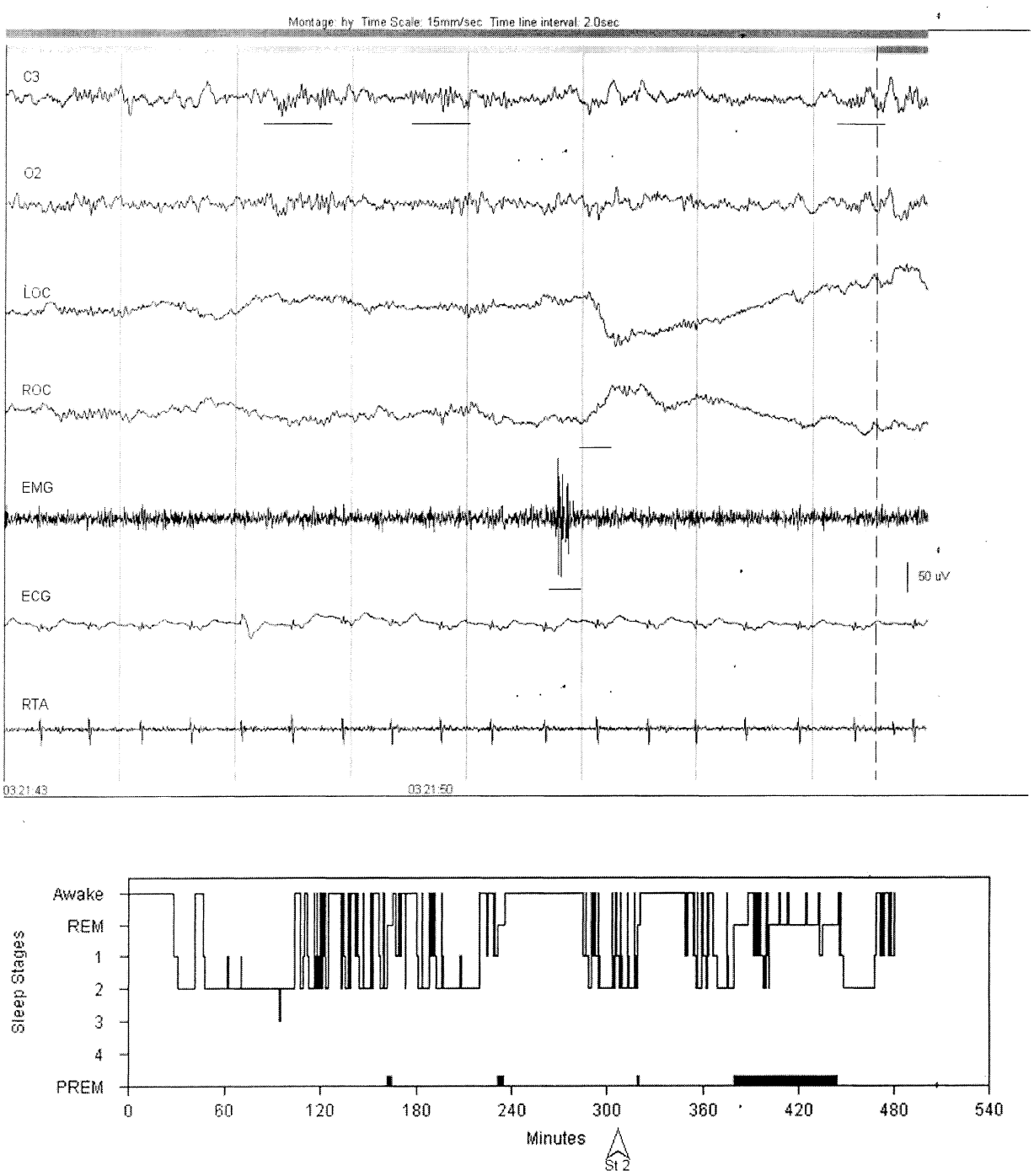

Figure NR6. Hypnogram and PSG tracing of late night stage 2 sleep from 43-year-old male post-traumatic nightmare (PTNM) patient. This patient had the most REM sleep signs of the entire sample and a fragmented sleep hypnogram on both nights (see Fig. NR7). He also reported dreaming vividly throughout 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

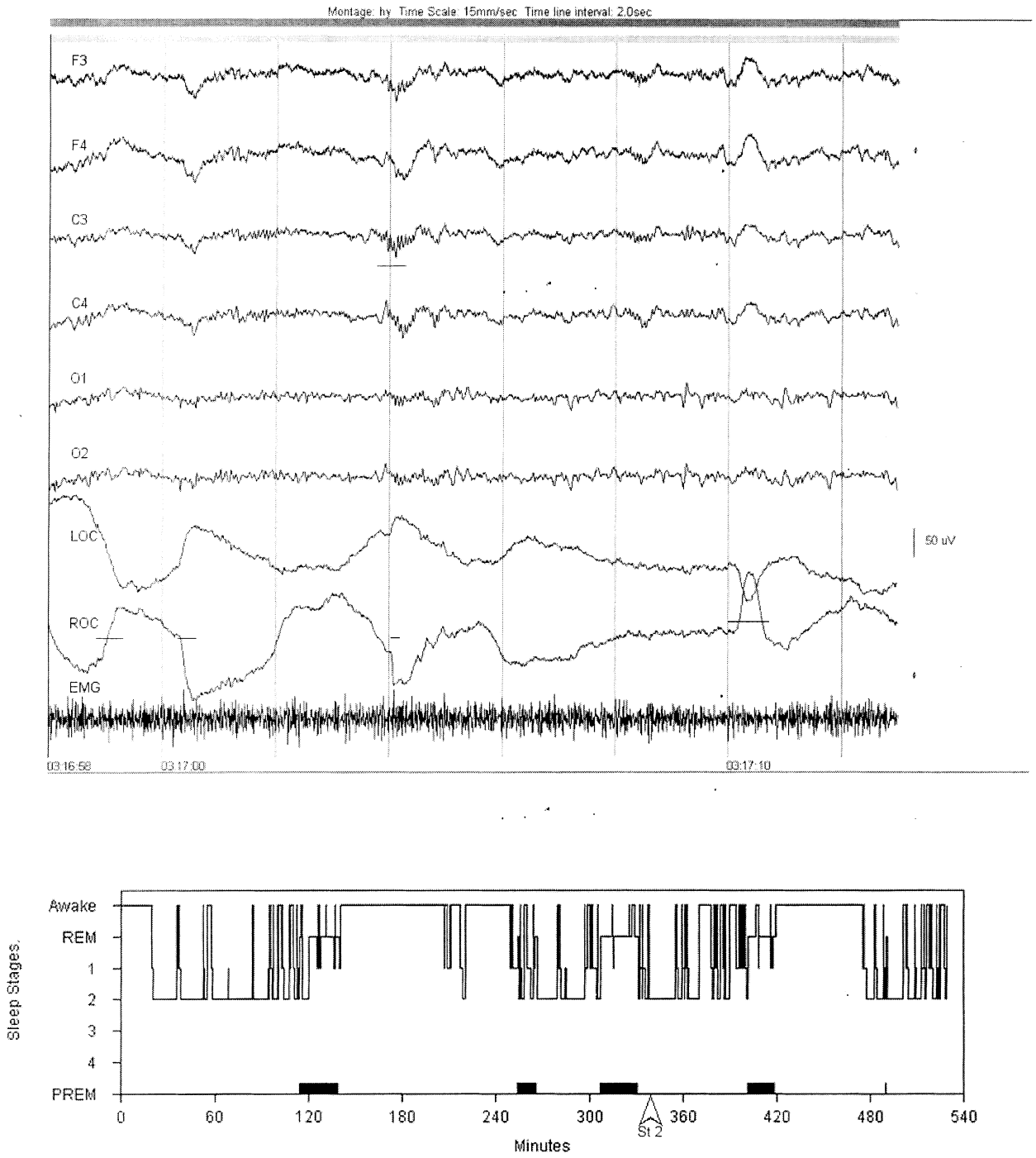

Figure NR7. Hypnogram and PSG tracing of stage 2 sleep from same patient as in Figure NR6 but on the following night. The epoch occurs less than 10 min after a lengthy REM sleep episode. Medium fast and rapid eye movements are visible; one of these occurs in exact synchrony with a sleep spindle. Legend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

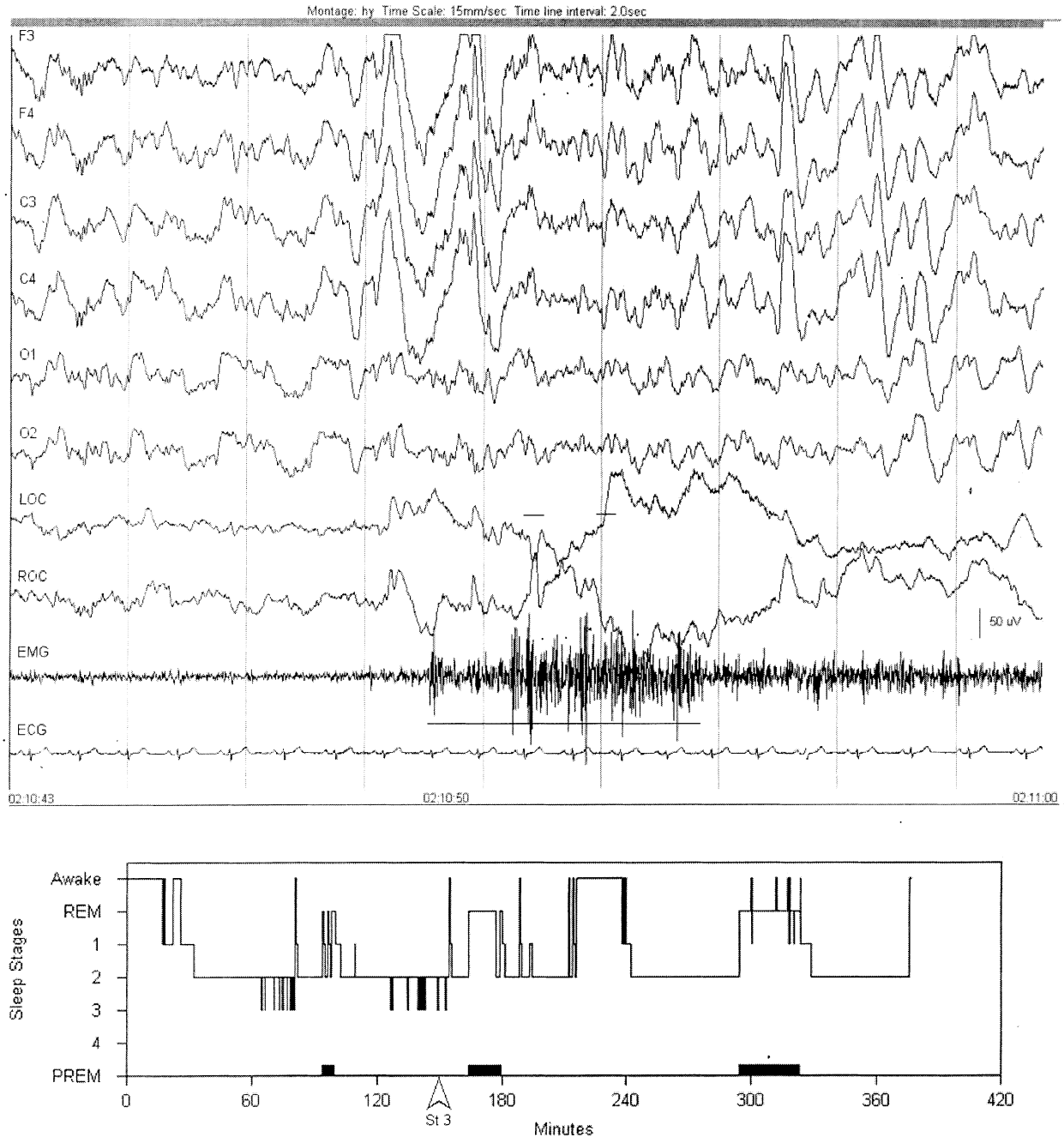

Figure NR8. Hypnogram and PSG tracing of stage 3 sleep from a 30-year-old female INM patient. A burst of medium fast-to-rapid eye movements coincides with a 5-sec burst of chin muscle activity against a background of quiescent EMG. A brief awakening occurred several minutes later. Legend as in Figure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

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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-