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.

© 2000 Cambridge University Press 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"

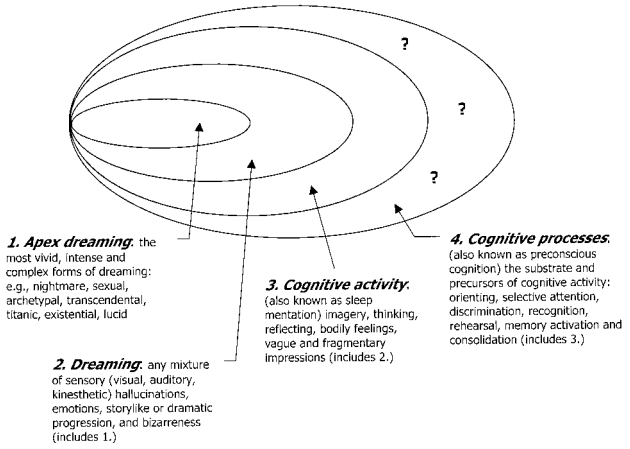

Figure 1. Four levels of specificity in defining sleep mentation. With an increasingly specific definition of sleep mentation, differences between REM and NREM mentation become more apparent. The two most specific levels (1 and 2) tend to occur much more exclusively in REM sleep. Cognitive activity (3) 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).

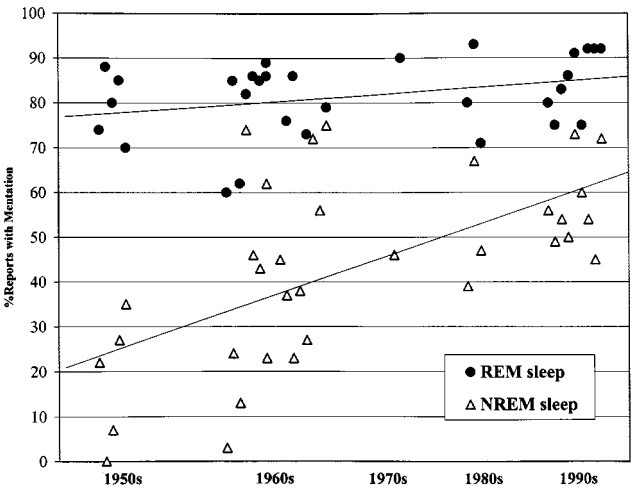

Figure 2. Summary of 35 studies of mentation recall from REM and NREM sleep over five decades. The percent of verbal reports that yielded some form of cognitive content after awakenings from NREM sleep increased from the 1950s to the 1990s, whereas the comparable percentage from REM sleep awakenings 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.

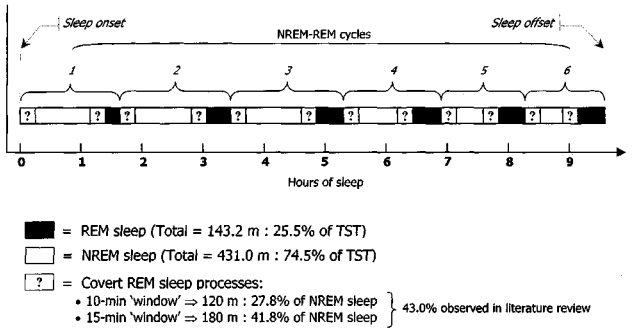

Figure 3. Probability model of covert REM sleep processes over six NREM-REM cycles: Normative results for 111 healthy nonmedicated subjects (127 nights). Illustration (to scale) of the normative sleep results listed in Table 2. The probability of obtaining covert REM sleep processes after a random awakening 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.