CHAPTER 12

ARE YOU AWAKE? COGNITIVE PERFORMANCE AND REVERIE DURING THE HYPNOPOMPIC STATE

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Kleitman (1963) observed that "immediately after getting up, irrespective of the hour, one is not at one's best" (p. 124). It is a paradoxical phenomenon—being more impaired upon awakening from sleep than upon going to sleep—that has been documented for a wide array of behavioral tasks. Typically, such impairment is modest and short-lived due to gradual awakening or to a slow transition out of the hypnopompic state. It can be dramatic, however, if the arousal from sleep is abrupt, regardless of whether the sleep occurs at night or during a daytime nap (Dinges, Orne, Evans, & Orne, 1981). Further, the intensity of the hypnopompic state as evidenced by the severity of cognitive impairment can be most profound if the awakening occurs during the first half of the night or if the person has been awake for a protracted period of time and is aroused after only an hour or two of recovery sleep. In such situations, the sleepiness evident in the hypnopompic state results in vastly impaired performance compared with that seen in a sleep-deprived subject (Dinges, Orne, & Orne, 1985b).

The dramatic nature of hypnopompic disorientation, confusion, and performance impairment has been described by a variety of names, including "sleep drunkenness" (Broughton, 1968, 1973), "postdormital sleepiness" (Association of Sleep Disorders Centers, 1979), and "sleep inertia" (Lubin, Hord, Tracy, & Johnson, 1976). The former terms are now used primarily for sleep pathology, whereas the latter is widely applied to describe transient awakening (i.e., hypnopompic) impairment in healthy persons. This chapter is concerned with hypnopompic cognition during intense sleep inertia and the processes that might account for it.

SLEEP INERTIA AND PERFORMANCE DURING THE HYPNOPOMPIC PERIOD

The studies cited in Table 1 represent much of what is known about hypnopompic cognition and the awakening process as it pertains to performance capability. Table 1 does not include a separate body of literature on dream reports at awakening. Virtually all the work conducted on performance during the hypnopompic state has derived from two theoretical perspectives, with slightly different emphases. The first reflects an interest in the functional differences between REM and non-REM (NREM) sleep stages in information processing potential; this is the performance analogue of the studies of sleep stage-dependent dream mentation. The approach typically taken is to awaken subjects from different stages of sleep and evaluate performance on a parameter of theoretical import, such as perception (e.g., Lavie & Sutter, 1975) or memory (e.g., Bonnet, 1983; Stones, 1977). The second approach derives from studies of human performance during periods of prolonged quasi-continuous wakefulness, when the adverse effects of sleep loss must be weighed against the adverse effects of sleep inertia upon abrupt arousal from sleep due to an emergency (e.g., Hartman & Langdon, 1965; Haslam, 1982). In these studies, emphasis is placed not on the preawakening stage of sleep but rather on the magnitude of sleep inertia, its duration, and the range of performances affected by it.

At the heart of sleep inertia is the nature of hypnopompic cognition and biobehavioral functioning on arousal from sleep. Although much more has been written about the hypnagogic state (Mavromatis, 1987; Schacter, 1976) than the hypnopompic state, the phenomenon of sleep inertia and its accompanying cognitive processes are ubiquitous. Because of this ubiquity and because it is typically a transient phenomenon (lasting between 1–20 min) during which cognition and performance can be grossly altered relative to other times, the hypnopompic period is often ignored in many studies on the effects of sleep on human functioning.

It is, however, precisely because of the dramatically altered cognitive performance, reverie, and subsequent amnesia of the hypnopompic period that sleep inertia is worthy of increased attention. At the very least, the phenomenon offers a window to the changes in cognition from sleep to waking. In its extreme form in an otherwise healthy individual, sleep inertia affords a model for cognitive impairment of a kind rarely seen in either experimental (e.g., sleep deprivation) or clinical (e.g., insomnia) studies. What follows is an especially dramatic example of sleep inertia that illustrates how profound the phenomenon can become if the appropriate paradigm is used. Following the ex-

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ample is a discussion of factors that contribute to it, including a review of evidence that sleep depth more than the preawakening stage of sleep is the crucial variable that influences hypnopompic cognition. We conclude with a theoretical framework that posits a common process (sleep pressure) underlying hypnopompic, hypnagogic, and sleep-related waking reverie.

A PARADIGM TO STUDY SLEEP INERTIA

To study the role of sleep pressure and various aspects of sleep infrastructure on hypnopompic performance, we conducted two studies of human cognitive functioning upon abrupt awakening from naps (Dinges et al., 1981; Dinges, Orne, & Orne, 1985a, 1985b). Like the studies cited in Table 1, the goal was to use a cognitive task as a probe to investigate the processes underlying hypnopompic cognition. Relatively short periods of sleep (1–120 min) provide a theoretically important way of studying cognitive performance during the hypnopompic state without confounding circadian variation and sleep infrastructure changes, which result when awakening occurs from longer periods of sleep (4–8 hr). Thus, studies of naps under 2 hr, at different phases of the circadian cycle and following varying amounts of prior sleep loss, permit assessment of specific aspects of sleep infrastructure and depth in relation to performance upon awakening.

In both studies, performance on a 3-min descending subtraction task (DST) was used as a cognitive probe on abrupt awakening. Details of the task have been published elsewhere (Dinges et al., 1985a). Briefly, it was developed to permit the subject to perform it while lying on a bed in the dark, thereby allowing assessment within a few seconds of sleep offset. The task requires an oral rather than a nonverbal response from the subject, which permits reverie intrusions to be observed. The subtractions are done silently, and the answers are said aloud. Both speed and accuracy are emphasized, and because the subtrahend and minuend change after each response, a considerable load is placed on memory. If a subject does not give a response on the DST for 20 s, the experimenter then says, "Please continue, guess if you have to," and thereafter pushes the subject every few seconds to respond. (This was necessary often during the hypnopompic period but was necessary only infrequently during baseline, presleep, or sleep deprivation phases of the studies.) Thus, no subject could score poorly on the task merely by not responding for the bulk of the allotted 3 min.

In our studies, DST performance was assessed repeatedly before each nap (to ensure that performance was asymptotic) and immediately following a motor response (answering a telephone) and an affirmative oral response (to the question "Are you awake?"), as well as repeatedly thereafter. In all 198 awakening nap protocols we have run to date, sleep was polysomnographically recorded, and the EEG was monitored during hypnopompic performance. Naps were either 1 hr (Dinges et al., 1981) or 2 hr (Dinges et al., 1985a) in duration, although subjects were not told how long they would be permitted to nap. They were aware, however, that they would be expected to perform

Table 1
STUDIES OF PERFORMANCE UPON AWAKENING

Study	Task
Dinges, Orne, Evans, and Orne (1981)	Simple reaction time
Dinges, Orne, and Orne (1985a)	
Okuma, Nakamura, Hayashi, and Fujimori (1966)	
Rosa, Bonnet, and Warm (1983)	
Webb and Agnew (1964)	
Wilkinson and Stretton (1971)	
Williams, Morlock, and Morlock (1966)	
Feltin and Broughton (1968)	Complex reaction time
Goodenough, Lewis, Shapiro, Jaret, and Sleser (1965)	
Scott (1969)	
Seminara and Shavelson (1969)	
Jeanneret and Webb (1963)	Grip strength
Tebbs and Foulkes (1966)	
Wilkinson and Stretton (1971)	Steadiness/coordination
Hartman and Langdon (1965)	Complex simulation
Hartman, Langdon, and McKenzie (1965)	
Langdon and Hartman (1961)	
Seminara and Shavelson (1969)	
Fort and Mills (1972)	Letter cancellation
Haslam (1982)	Logical reasoning
Akerstedt and Gillberg (1979)	Memory tasks
Bonnet (1983)	
Gastaut and Broughton (1965)	
Grosvenor and Lack (1984)	
Stones (1977)	
Dinges et al. (1981)	Mental arithmetic
Dinges et al. (1985a)	
Pritchett (1969)	
Wilkinson and Stretton (1971)	
Scott (1969)	Clock reversal

Table 1 (continued)

Study	Task
Carlson, Feinberg, and Goodenough (1978)	Time estimates
Koulack and Schultz (1974)	Vigilance, trailmaking
Lavie and Giora (1973)	Visual-perceptual
Lavie and Sutter (1975)	
Scott (1969)	
Scott and Snyder (1968)	

the DST immediately upon awakening from sleep. Awakening was done auditorily by a telephone, which rang continuously for 1 min and, if not answered, rang on and off every 2 s for another minute. If the subject still did not respond, his or her name was spoken over the intercom until a verbal response was elicited. After answering the phone, the subject was asked how much time had elapsed since he or she had last spoken with the experimenter (immediately prenap) and was asked to estimate his or her sleepiness on a 10-point analogue scale. Following this, the subject was instructed to hang up the phone, lie back down (in the dark), and perform the DST. The polygraph was kept running throughout awakening performance, which was audio tape-recorded. After electrode removal, DST performance was again assessed repeatedly.

In one major study (Dinges, 1986; Dinges, Orne, Whitehouse, & Orne, 1987; Dinges, Whitehouse, Orne, & Orne, 1988), the amount of continuous wakefulness (sleep loss) prior to a 2-hr nap opportunity was varied from 6 hr to 52 hr. This had the effect of producing marked differences in nap sleep-stage infrastructure and amount, which permitted an assessment of hypnopompic cognition as a function of varying sleep depths or intensities. At the most extreme intensity, sleep inertia was profound, and intrusions of hypnopompic reverie occurred during the DST at awakening. The following example illustrates the power of our experimental protocol to produce a dramatic hypnopompic condition that is characterized by social interaction with simultaneous performance impairment, hypnopompic reverie, misjudgment of sleepiness, and a dissociation between the electroencephalogram (EEG) and behavior.

SOCIALLY AWAKE YET FUNCTIONALLY ASLEEP

The nature of the cognitive impairment in DST performance that was evident at awakening, especially after subjects had been sleep-deprived and therefore had slept very deeply, was such that it was often accompanied by dramatic intrusions of reverie as subjects attempted to say the answers aloud. Table 2 provides a transcript of the interaction between one of our sleep-deprived subjects (18-year old healthy man) and the experimenter after the subject had been awake for 52 consecutive hours in the sleep-deprivation protocol, during

Table 2

DIALOGUE BETWEEN SUBJECT AND EXPERIMENTER DURING PRESLEEP PERFORMANCE OF DESCENDING SUBTRACTION TASK AND UPON AWAKENING FROM SLEEP

Minute	Speaker	Content
		Presleep Performance
0	Experimenter:	I would like you to do the subtraction task again. Remember to work as fast and accurately as you can. Your starting number is 931.
1–3	Subject:	931 922 914 907 901 *895* 891 888 886 869 862 . . . . . 509 503. [75 correct, 2 errors]
4–5	Experimenter:	That's good. On a scale from 1 to 10, where 1 is very wide awake and 10 is very sleepy, how do you feel now?
	Subject:	8
	Experimenter:	Please lie quietly with your eyes closed, but stay awake, until I tell you that you can go to sleep.
	Subject:	Okay.
	Experimenter:	Remember that the end of the nap will be signalled by the telephone ringing, which you should answer as quickly as possible. You will then be asked to do the subtraction task. Okay, you can go to sleep now.
		Postsleep Performance
128–129	Experimenter:	Are you awake?
	Subject:	Yes!
	Experimenter:	Can you hear me okay?
	Subject:	Yeah.
	Experimenter:	How long since I spoke to you last?
	Subject:	Um . . . um . . . 90 minutes.
	Experimenter:	On a scale from 1 to 10, where 1 is very wide awake and 10 is very sleepy, how do you feel now?
	Subject:	Um . . . about um . . . 6.
	Experimenter:	Hang the phone up and lie back. I would like you to do the subtraction task. [S having difficulty hanging up telephone.]
	Experimenter:	Can you see to hang it up?
	Subject:	There, got it.
	Experimenter:	Remember to work as fast and accurately as you can. Your starting number is 648.

Table 2 (continued)

Minute	Speaker	Content
130	Subject:	648 64 . . ah 63 . . ummm 500 and ah . . (sigh)
	Experimenter:	Continue!
	Subject:	500 and um . . let's see . . um . . 696 *685* 640, ah, um . . . *632* *631* *631*
	Experimenter:	Continue!
132	Subject:	What if people ran faster than normal people run home—than the normal person runs faster than the square root of two times . . . (mumbles incoherently) . . . and normal quote-unquote people take up two derivatives of normal people in skin brackets . . . [1 correct, 4 errors]
133	Experimenter:	That's good.

Note: After the presleep test, the subject, who had been awake for 52 hr, took a 2-hr nap. After the nap, the subject did not respond to the telephone bell but picked up the phone when his name was called.

which the DST was repeatedly performed. The script in Table 2 begins as the subject is lying on a bed, in a dark quiet room, just before being allowed to sleep. His presleep (postsleep-loss) DST performance for 3 min of 75 correct answers and 2 errors was below his presleep-loss levels of 82–99 correct answers and 0–2 errors; this net loss in number completed is characteristic of the cognitive slowing on subject-paced tasks that typically occurs with sleep loss (Dinges, 1989b).

Following this performance, as is typical for intensely sleepy persons, the subject fell asleep (Stage 1) within 30 s of being permitted to do so and had his first epoch of slow wave sleep (SWS) 4 min later (Dinges, 1986). He remained asleep for the entire 2-hr period. He had no REM sleep but accumulated 82.5 min (69% of total sleep time) of SWS (70 min of Stage 4 sleep), which is nearly as much SWS as healthy young adults of his age acquire in an average 8-hr nocturnal sleep (cf. R. L. Williams, Karacan, & Hursch, 1974)! He was in Stage 3 sleep at the time of the awakening bell. Although he did not answer the awakening telephone call until his name was spoken, which indicates an intense sleep depth, he interacted immediately thereafter, affirming that he was awake, estimating the time since he had last spoken to the experimenter, and providing a rating of his sleepiness.

Curiously, he estimated his sleepiness at a score of 6 at awakening, indicating that he was less sleepy than he had been before the nap (rating of 8). This was a clear dissociation between his self-report and his DST performance, which was far worse at awakening than it was after 52 hr of wakefulness, just prior to the nap. We have analyzed enough data to show that this hypnopompic misjudgment of sleepiness is common in subjects whose sleep has followed

a sustained period of wakefulness beyond 18 hr, but that it does not occur if the nap is taken before sleep deprivation (Dinges, 1988). The intensity of the sleep inertia seems to make it difficult to estimate how sleepy one feels. This finding is consistent with Sewitch's (1984) report that the length of continuous NREM sleep affects the normal sleeper's perception of having been awake or asleep, such that awakenings from SWS or after prolonged periods of NREM sleep most often result in subjects being least able to identify accurately whether they had been awake or asleep.

Following his time and sleepiness estimates and his interaction with the experimenter over the phone, the subject was asked to perform the DST. He was totally unable to execute subtractions correctly (only his repeat of the starting number was correct; his four subtraction errors are in italics in Table 2), and he could not remember where he was in the sequence, despite two prods to continue from the experimenter.

He also had considerable difficulty preventing hypnopompic reverie from intruding into his oral output. The spoken reverie that occurred 2 min into the awakening DST performance (132 min in Table 2) began after the experimenter prompted him a second time to continue. It is noteworthy that the apparent evocation of this spoken reverie by the experimenter is consistent with the observations of Broughton (1968, 1982) that parasomnic episodes such as sleep walking and enuresis can be triggered by external stimuli and, therefore, can be considered disorders of arousal or of partial arousal. In fact, the awakening performance behavior of this subject is reminiscent of the confusional arousals from SWS described by Broughton (1968), which he suggested were due to "impaired cerebral responsiveness or of functional deafferentation" (p. 1074). The actual reverie of our subject at 2-min postawakening is illustrative of the semicoherent material we have observed in the awakening reverie of other subjects. Neologistic phrases such as "normal people in skin brackets" are referred to, along with phenomenological material appropriate to the individual such as "the square root of two times" (this subject was a math major and, indeed, the reverie has the quality of a statement of a mathematical problem).

Interestingly, the subject indicated that he had dreamed during the nap; this response was made in a questionnaire booklet he completed while electrodes were being removed, 15 min after awakening. No dream content was asked for and, consequently, it is unknown whether the dream related to his hypnopompic reverie. This was not the only reverie he experienced, however. Despite coherent social interaction and the completion of performance tasks, he experienced other reverie intrusions into his cognitive performance oral output at 6 min and 35 min postawakening. Although reverie was less dramatic in many other subjects to the extent that it lasted only a few seconds rather than a full minute, it was apparent in most who had undergone intensive sleep loss in the form of one- to five-word intrusions into oral performance output. Such intrusions were most common in the hypnopompic period.

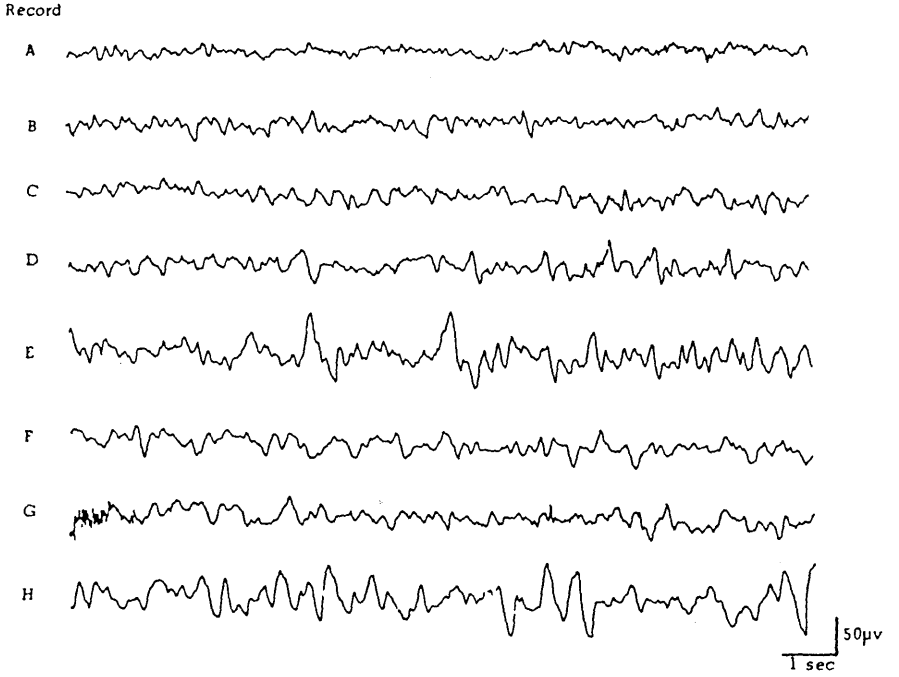

Figure 1 Vertex (C_z-A_1A_2) EEG recordings from a healthy young adult with eyes closed lying on a bed in a dark quiet bedroom, taken at eight different times. Record (A) after 6 hr of wakefulness—instructed to lie awake quietly. (B) after 52 hr of wakefulness—minute 1 after being instructed to lie awake quietly. (C) 2 min after B—still attempting to stay awake. (D) 1 min after C and after being instructed to go to sleep. (E) 2 min after D (asleep by polysomnographic criteria). (F) after 2-hr nap—minute 1 of DST performance at awakening (see text). (G) 2 min after F—during reverie of final minute on DST (see Table 2). (H) 2 min after completion of DST at awakening—instructed to remain awake, but *S* fell asleep.

FUNCTIONALLY ASLEEP YET ELECTROENCEPHALOGRAPHICALLY AWAKE

The EEG recording obtained during the awakening reverie of our subject was compared with recordings made under comparable conditions (e.g., eyes closed, prone in bed) prior to and following sleep deprivation, during wakefulness and sleep onset, in an effort to determine whether the reverie was accompanied by a clear change in EEG. Increased theta and delta activity in particular is characteristic of a period of reverie, and one would expect the EEG to be visibly different during reverie than during the prenap DST performance, when the subject was intensely sleepy but able to perform 75 correct subtractions in 3 min.

Figure 1 displays vertex EEG recordings made at eight times for this subject. The presleep-deprivation record (Record A) taken after only 6 hr awake is notably different from all others because it contains less slow wave activity (delta and theta) and more beta activity. All records (Records B–H)

from the sleep-deprivation period show increased slow wave activity. Of particular interest is the comparison between Records C and G. Although no EEG recording was made during the presleep DST performance, Record C (at 52 hr and 2 min of sleep loss) was obtained 2 min into the 3-min presleep wake, eyes-closed baseline (i.e., 1 min before the subject was allowed to sleep). Record G was obtained 2 min into the postsleep awakening DST performance, when the reverie at the bottom of Table 2 was elicited. There is no obvious difference between these records, and they are clearly different from the high-amplitude, slow-frequency waveforms apparent in the sleep records at 1 min and 2 min of nap onset (Records D–E) and when the subject fell asleep after completing the awakening DST (Record H). Despite the lack of EEG differences between Records C and G, differences at these times in DST performance and cognitive coherency are profound (Table 2).

SLEEP DEPTH AND SLEEP INERTIA

The depth of sleep achieved by our subject during the 2-hr (recovery) nap following over 2 days without sleep was intense, as it has been in every subject we have examined who was sleep deprived. Recovery sleep following sleep loss has long been known to involve a greater depth of sleep. Consistent with other studies, the greater depth of sleep was evidenced by a decreased latency to SWS (Dinges, 1986), an exceptionally high amount of SWS, especially during the first NREM cycle of sleep (Borbely, Baumann, Brandeis, Strauch, & Lehmann, 1981; Dinges, 1986; Feinberg, Floyd, & March, 1987; Hume & Mills, 1977; Webb & Agnew, 1967, 1971), decreased body movement during sleep (Naitoh, Muzet, Johnson, & Moses, 1973), a failure to respond to the awakening bell (Rechtschaffen, Hauri, & Zeitlin, 1966; Rosa & Bonnet, 1985; Williams, Hammack, Daly, Dement, & Lubin, 1964), and a rapid return to sleep following awakening (Bonnet, 1978). All of these parameters provide an index of sleep depth. Ironically, sleep depth often has not been considered in studies of performance upon awakening.

There has been considerable interest in determining which aspects of sleep account for sleep inertia or the performance impairment evident in the hypnopompic period. The typical experimental paradigm used to study it has consisted of abrupt, forced awakening from REM or NREM sleep stages. Thus, when performance during the hypnopompic period has been investigated (Table 1), the paradigm for studies of sleep-stage-related mentation has been most commonly used. In these studies, it is often assumed that the severity of performance sleep inertia is directly related to the preawakening stage of sleep, such that awakenings from SWS yield the most dramatic hypnopompic phenomena, whereas those from REM sleep yield more wakelike performances. Although the results of most studies that have investigated arousal from different sleep stages support this view, the bias of evaluating only the preawakening stage of sleep is so pervasive that other aspects of the sleep infrastructure are rarely examined to determine whether they are more consistently associated with the magnitude and nature of hypnopompic phenomena.

There is reason to hypothesize, however, that the intensity of hypnopompic reverie and the extent to which cognitive performance is impaired during awakening are most accurately characterized as being a function of the depth of sleep, of which the preawakening stage of sleep is but one facet (Dinges et al., 1985a, 1985b). This is evidenced in three ways: (a) awakenings exclusively from Stage 2 NREM sleep yield performance decrements that vary as a function of time of night (circadian phase) (Rosa, Bonnet, & Warm, 1983); (b) awakenings from recovery sleep following prolonged wakefulness yield decrements greater than those found for awakenings from the same stage of sleep prior to deprivation (Akerstedt & Gillberg, 1979; Fort & Mills, 1972; Rosa et al., 1983); and (c) abrupt awakenings from naps yield increasingly more severe cognitive performance decrements as the amount of NREM (Stages 2 + 3 + 4) sleep increases (Dinges et al., 1981) and as the amount of wakefulness prior to sleep increases, regardless of the stage of sleep from which subjects are awakened (Dinges et al., 1985a).

How can we account for the fact that most of the studies in Table 1 have observed greater hypnopompic impairment on awakenings from SWS relative to Stage 2 or REM sleep? This may have resulted because SWS is consistently associated with greater sleep depth, especially in the first NREM cycle (Dinges 1986; Feinberg et al., 1987). Generally, the preawakening depth of sleep may be more important than the depth of sleep at other times in the sleep period (Bonnet, 1983), making the preawakening stage of sleep a salient variable for hypnopompic cognition. On the other hand, at least one study has reported that the behavioral performance variables at repeated awakenings from recovery sleep following (40 hr and 64 hr of) sleep loss are generally more sensitive than sleep stages to different amounts of prior wakefulness (Rosa & Bonnet, 1985).

In fact, it is not clear that SWS is essential for severe hypnopompic performance impairment to occur in response to sleep depth. Bonnet (1985) observed that hypnopompic disorientation could be profound in subjects who were permitted to sleep but who were denied most of their SWS and REM sleep during repeated nights of experimentally induced sleep disruption every minute. In his study, auditory arousal thresholds increased dramatically by the second night of sleep disruption, indicating increased sleep depth, while subjects began to become confused on awakening:

> They often could not give ratings. One subject later recounted that at awakening she could hear the technician talking but his words did not seem to make sense. Other subjects could not perform simple tasks such as being able to respond with "a" when prompted for the letter that precedes "b". One explanation for this behavior is sleep drunkenness (i.e., arousal from very deep sleep resulting in confusion). (p. 18)

SLEEP PRESSURE AND THERMOREGULATION

It is my contention that sleep pressure, or the probability of transition from wakefulness to sleep, (Pr[W → S]) underlies sleep depth, SWS, and the magni-

tude of sleep inertia effects during the hypnopompic period. The simplest way to manipulate sleep pressure is through prior wakefulness. But which physiological process underlies sleep pressure and correlates with manifestations of sleep depth, especially pressure for SWS? Although biochemical changes must underlie any such process, and candidates have been proposed (e.g., see Chapter 2 by Hobson in this volume), there is reason to suggest that body core temperature and, presumably, brain metabolic activity may be an essential link in the hypnopompic process. Body core temperature shows a circadian rhythm, but sleep (especially the first SWS period of the night) also has the (evoked) effect of thermal down-regulation (Gillberg & Akerstedt, 1982). There is evidence that this effect may be enhanced whenever the pressure for sleep is increased. Aschoff, Giedke, Poppel, and Wever (1972) observed that "after two days without sleep, one night of uninterrupted sleep results in an exaggerated drop in rectal temperature" (p. 144). A significant covariation between oral temperature and performance upon awakening has been reported (Rosa & Bonnet, 1985). But the relation between SWS and temperature may go both ways (Sewitch, 1987). Experimentally induced lowering of rectal temperature at sleep onset has been associated with an increase in Stage 4 sleep and a lengthening of the first NREM/REM cycle (Sewitch, Kittrell, Kupfer, & Reynolds, 1986). Passive afternoon body heating has been observed to result in an enhanced drop in rectal temperature during the first 2–4 hr of subsequent nocturnal sleep (Horne & Staff, 1983).

One possible mechanism underlying the increasingly impaired performance at awakening of subjects who have experienced intense pressure for sleep is a decline in cerebral metabolism resulting from thermal down-regulation exacerbated by sleep pressure. The purpose of such down-regulation is unclear, although speculation is easy (e.g., the increased need for protein synthesis, which is favored during sleep, especially during periods of lowest basal metabolic level). Whatever its purpose, such basal drops may have a longer time course than the EEG manifestation of specific sleep stages and may make coherent cortical function impossible until metabolic or specific biochemical activity has been increased through the passage of time (e.g., circadian variation), a change in sleep stage (e.g., accumulation of REM sleep), or increased physical activity at awakening (e.g., getting out of bed)—all of which are intercorrelated such that REM sleep is more likely to occur as body core temperature is rising, and awakenings are more likely to occur from REM sleep (Dinges, 1989a). Unfortunately, despite many studies of hypnopompic performance, none have assessed body core temperature as a covariant of sleep inertia beyond looking at circadian variability.

The hypothesized widespread metabolic decline in cortical activity that would covary with pressure (and therefore with depth) of sleep would probably make it exceedingly difficult for a person to perform well if aroused abruptly from sleep when basal metabolic levels are low. Virtually every type of performance, especially all cognitive performances involving memory and attention, would be adversely affected. There is no reason to believe, however, that

the subject could not interact socially at some simple level such as name acknowledgment; indeed, this is what appeared to be the case with our subject. On the other hand, when performance is demanded, reverie can intrude, especially in response to a stimulus (e.g., the experimenter says "continue"). The nature of such semicoherent reverie may be determined by the extent to which the "functional deafferentation" suggested by Broughton (1968) has taken place. The reverie would, therefore, be the result of insufficient neural metabolism for coherent mentation. The inhibition needed for directed cognition would be lacking.

THEORETICAL MODEL OF REVERIE BASED ON SLEEPINESS

The concept of pressure for sleep, which is expressed as the probability of transition from wakefulness to sleep, (Pr[W → S]), is in effect a definition of sleep tendency or of sleepiness. To the extent that sleep-related reverie occurs without exception during sleep stages, especially during Stage 1 sleep, then a sleepiness model can account for it. That is, as the probability of a transition to sleep increases, the probability of reverie will increase: Increased pressure for a thermoregulatory down-regulation may underlie this phenomenon. Whatever its physiologic basis, it can account for the "semi-dreaming" (Kleitman, 1963, p. 221) of sleep-deprived subjects, which was observed in the earliest human sleep loss study (Patrick & Gilbert, 1896) as well as for both hypnagogic and hypnopompic reverie.

Figure 2 displays the manner in which sleep pressure manifests itself in behavior and physiology, depending on the context. If the context is one in which the subject is attempting to remain awake, as in sleep-deprivation studies, then increased microsleeps (Stage 1 intrusions), cognitive slowing, response blocks or lapses, and response habituation will be increased. Semi-dreaming or dreaming while awake during tasks that require oral output will be more likely. If the subject is attempting to go to sleep, then increased sleep pressure will be evident in decreased sleep latencies (e.g., as seen on the multiple sleep latency test) and hypnagogic reverie will be more likely to occur sooner. If the context is one of attempting to awaken from sleep, then increased sleep pressure will result in increased sleep depth, and the probability of hypnopompic reverie will be increased. There is, therefore, no reason to posit that the reverie resulting from these three instances is qualitatively different. The various effects of sleep pressure in Figure 2 are intercorrelated. The word used to describe the resulting reverie is more aptly thought of as a descriptor of the context in which each occurs, not of any unique characteristic of the mentation.

Differences can occur, of course, in the severity of reverie intrusions (i.e., pressure for sleep). Hypnopompic mentation may be the most dramatic form of reverie because it involves transition from sleep to wake (sleep inertia), whereas waking and hypnagogic mentation involve transition from wake to sleep ("wake inertia"). In all three cases, the process underlying the emanation

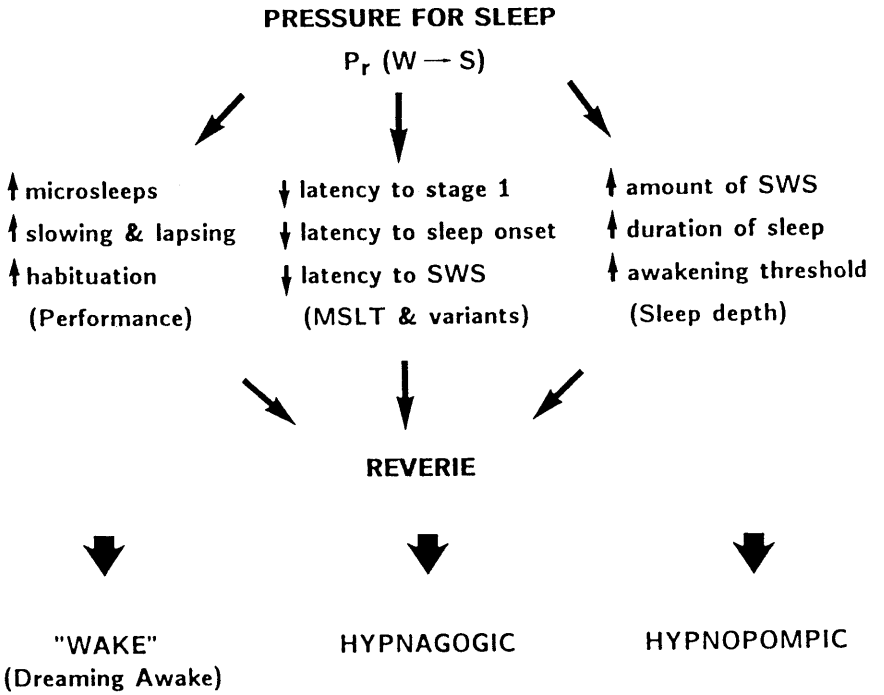

Figure 2 Theoretical model of how sleep pressure (i.e., probability of a transition from wake to sleep) can manifest in sleep-related behavior and physiology, depending on the context, and result in reverie. To the left is the sleep-deprivation context, in which the subject is attempting to remain awake but microsleep intrusions result in "semi-dreaming" reverie. Center is the sleep onset context, in which the subject is attempting to go to sleep and intense sleepiness leads to rapid sleep onset and hypnogogic reverie. To the right is the awakening context, in which the subject is attempting to transition out of sleep but sleep inertia resulting from increased sleep depth leads to hypnopompic reverie. In all three cases, the resulting reverie is the product of increased physiological sleepiness.

of reverie and the nature of cognition in general may well be the same. What happens to this process as REM sleep (and circadian time) accumulate remains to be determined.

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