WHY WE NAP

EVOLUTION, CHRONOBIOLOGY, AND FUNCTIONS OF POLYPHASIC AND ULTRASHORT SLEEP

CLAUDIO STAMPI, EDITOR

BIRKHÄUSER

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Humans (and some other mammals) are unique in their monophasic sleep patterns - most other animals sleep several times a day. But many humans also nap or briefly "nod off" during the waking hours, and certain work and life styles require "polyphasic" sleep patterns of interspersed periods of sleep and wakefulness. Only recently have the underlying bases of polyphasic sleep and its effects on human behavior been studied, and this volume by international experts in the fields of sleep research, circadian rhythms, and human performance (neuroscientists, physiologists, psychologists, psychiatrists, chronobiologists) brings together the latest findings and reviews major aspects of this important and fascinating new field. Readers will include sleep researchers and neuroscientists interested in research on states of consciousness, and also industrial psychologists, ergonomisu, and professionals who need information on efficient programming of work-rest schedules.

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Foreword by Jürgen Aschoff

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To Alessandra
who provided me with a unique
opportunity to observe the human
innate ability to live in a polyphasic
world, and

to Diana
who, as a result, demonstrated how
easy it is to break the habitual
monophasic nocturnal sleep
when the motivation is strong enough

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Foreword

JÜRGEN ASCHOFF

"Very bad habit! Very bad habit!"
Captain Giles to Joseph Conrad
who had taken a siesta.
— Conrad: *The Shadow Line*

On the Multiplicity of Rest-Activity Cycles: Some Historical and Conceptual Notes

According to its title this book tries to answer the profound question of why we nap —and why Captain Giles was wrong in blaming Conrad for having napped. However, in this volume the term *nap* is not used in the narrower sense of an afternoon siesta; instead, emphasis is placed on the recurrent alternation between states of alertness and drowsiness, i.e., on rest-activity cycles of higher frequency throughout the 24 hr. In view of this focus, two authors (Stampi, in Chapter 1, and Ball, in Chapter 3) rightly refer to the psychologist Szymanski who was among the first to describe "polyphasic" activity patterns. Hence, I consider it appropriate to open this foreword with a few historical remarks.

At the time when Szymanski (1920) made the distinction between "monophasic" and "polyphasic" rest-activity patterns and sleep-wake cycles, respectively, not much was known about the mechanisms of such temporal structures. Although the botanists quite some time ago had demonstrated the endogenous nature of the "monophasic" sleep movements in plants, the hypothesis of an (still unknown) external driving force was favored by those who studied rhythms in animals and humans (Aschoff, 1990). It must be noted, however, that Szymanski himself, summarizing his

recordings of activity in children and adults within a range from minutes to hours, derived the "principle of activity by internal needs," and even used the term *organische Uhr* (organic clock): "The fluctuations in the disposition to work . . . constitute the mechanism of that organic clock from which consciousness reads the time" (Szymanski, 1922; my translation from German). He also came close to the notion of a system which later on was called *circadian* (Halberg, 1959). Once he had kept a canary in continuous darkness (DD) for 73 days, and he found that not only did the (mainly) monophasic rhythm persist but that its phase eventually was shifted by 6 hr. In view of these findings he posed the question of whether it might be possible that, in DD, a "new type of rest-activity cycles emerges which deviates from the norm" (Szymanski, 1914).

In his studies on humans, Szymanski made use of spring-suspended beds and chairs which allowed him to record movements of all kinds of frequencies, down to respiration. Apart from the main sleep-wake cycle, he noticed a prominent rhythm with a period of 2.5 hr during wakefulness and sleep, with bouts of activity separated by pauses of about 45 min, and superimposed by faster rhythms in the range of minutes. He also observed that, intraindividually, the variability of the various rhythmic components increased as the periods became shorter (Szymanski, 1922). This concurrent representation of activity rhythms with different frequencies becomes most obvious in typically polyphasic species such as rodents. The period that can be extracted from a record depends on the recording device and on the temporal scanning grid applied in the analysis. The actogram of a mouse, recorded with a spring-suspended cage under a light-dark cycle (LD), reveals a "pure polyphasic" (cf. Stampi, Chapter 1, this volume) pattern with 12 bouts of activity per 24 hr when the data are summarized in 10-min bins; in the same set of data, a period of approximately 4.5 hr appears (with only minor variations in amount of activity from L to D) when a 1-hr grid is applied; finally, the typical circadian pattern (with two nocturnal peaks) becomes evident when the data are organized in 2-hr bins (cf. Fig. 1 in Aschoff, 1957).

The very fact that rhythms of quite different frequencies coexist in the behavior of an individual, and often interact with each other (cf. later), is not at variance with our present view that we are dealing with at least two distinctly different classes of rhythms. There is, on the one hand, the circadian system that evolved in adaptation to time structures in the environment, which is driven by a localized pacemaker (or a set of pacemakers) within the central nervous system, and the period of which, when measured in constant conditions, usually does not differ by more than ±3 hr from an overall mean of about 24 hr (Aschoff et al., 1982). On the other hand, there is a variety of rhythms whose periods range from 1 to > 5 hr, which have no immediate relationship to periodicities in the environment, and for which it is unlikely that they are controlled by a common mechanism (Schulz and Lavie, 1985; cf. also later). The period of these

"ultradian" rhythms is said to be 10 times more variable than the circadian period (Gerkema and Daan, 1985), and it is obviously more affected by internal as well as external factors. As has been shown in birds and mammals, the frequency of an ultradian rhythm may depend on food availability, on period and phase of the circadian rhythm, and on the season, either by direct effects of the changing photoperiod or via changes in the circadian system. Furthermore, a phase-setting effect on ultradian rhythms by the circadian system has been demonstrated in 8 mammalian species (for references, see Aschoff and Gerkema, 1985). The reverse possibility, i.e., a shift in phase of the circadian rhythm by an ultradian bout of activity, has been suggested by a few actograms of voles (cf. Fig. 4 in Gerkema and Daan, 1985).

In spite of bidirectional interactions between the two classes of rhythms, there is no doubt that ultradian rhythms can be expressed independently of the circadian system. This statement is based on two sets of observations. During ontogeny, as well as during the emergence of hibernating mammals from torpor, ultradian rhythms appear prior to the circadian rhythm, and there are data which indicate that, at old age (and during entrance into hibernation?), the circadian system may become disintegrated, with only ultradian components being left. From this sequence of events, it could be concluded that the ultradian time structure is more "basic" than the circadian one. However, one has to remember that the appearance or disappearance of an overt rhythm does not necessarily reflect a change in the state of the generating mechanism; it may also be due to a coupling or uncoupling of the behavioral element to or from an ongoing central rhythm, as demonstrated in hibernating mammals by the episodic awakenings timed on a circadian scale (Pohl, 1964; Daan, 1973). In the rat, the circadian pacemaker is rhythmic prenatally and prior to its expression in an overt rhythm (Fuchs and Moore, 1980). In view of such uncertainties in defining "priorities" in the development of rhythms, the notion of an independent ultradian system is more rigorously supported by the observation that ultradian rhythms persist, and may even be more clearly expressed, in animals which have lost circadian rhythmicity after lesions of the suprachiasmatic nuclei (Gerkema and Daan, 1985; Honma and Honma, 1985). It remains to be seen whether these rhythms are driven by real pacemakers (cf. several chapters in Schulz and Lavie, 1985). In the vole, the retrochiasmatic area and the arcuate nucleus seem to be essential for the expression of ultradian rhythms in wheel-running and feeding (Gerkema et al., 1990).

The multiplicity of periods represented in ultradian rhythms, their nonstationarity, and the often inconsistent interrelationships among the components, impede attempts to arrive at a unifying concept of mechanisms. The theory of a Basic Rest-Activity Cycle (BRAC), advanced by Kleitman (1961), presupposes rhythmicity of about 90 min during wakefulness as well as during sleep, which is based on a common generating

mechanism. An impressive amount of data collected from studies on humans seems to support this hypothesis (Kleitman, 1982). However, to document an ongoing process that controls rhythms throughout the day and night, the demonstration of equality in *period* is suggestive but not sufficient—a persistence in *phase* over time is the major argument. Its documentation is impeded by the probability that the (persisting) ultradian rhythm is reset at the beginning and/or the end of a major sleep episode. To my knowledge, phase continuity of a rhythm with a 1.5-hr period has so far been documented only in studies with narcoleptic patients (Schulz et al., Chapter 16, this volume).

These findings agree with the BRAC model, but other rest-activity cycles for which a persistence in period has been documented during circadian arrhythmicity are not accommodated by it, e.g., 2.5-hr behavioral rhythms in voles (Gerkema and Daan, 1985) and in rats (Honma and Honma, 198S).

In conclusion: In contrast to the circadian system, the ultradian rhythmicity is formed by a multiplicity of oscillating units. The two domains agree in being the means to keeping internal order within the organism. This order provides the base for advantages either in the organization of internal processes or in the response to environmental conditions, and often these aims are achieved by an interaction between both domains.

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Preface

CLAUDIO STAMPI

> You must sleep sometime between lunch and dinner, and no half-way measures. Take off your clothes and get into bed. That's what I always do. Don't think you will be doing less work because you sleep during the day. That's a foolish notion held by people who have no imagination. You will be able to accomplish more. You get two days in one—well, at least one and a half, I'm sure. When the war started, I *had* to sleep during the day because that was the only way I could cope with my responsibilities.
>
> -Sir Winston Churchill[1]

Sir Winston Churchill was not the only person of renown who reputedly survived solely on naps. The ultrashort sleep and multiple daytime naps taken by Leonardo da Vinci, Napoleon, Salvador Dali, and Thomas Edison are already part of the anecdotes and legends perpetrated about famous nappers. Interestingly, Edison tried to make sleep almost unnecessary (he considered that to sleep eight hours a night was a "deplorable regression to the primitive state of the caveman"[2]), and indeed he partially achieved his goal, for his invention of the electric light placed man in an around-the-clock world.

Contrary to what the main title may suggest, this volume is not just about the afternoon siesta. Rather, the focus is on the recurrent alternation between states of alertness, which is experienced throughout 24 hr by most, if not all, living animals. The great majority of species show typical polyphasic rest-activity patterns. Monophasic behavior (one prolonged sleep episode per day) —the one usually displayed by adult humans and a

[1]Quoted in: Walter Graebner (1965): *My Dear Mister Churchill.* London: Michael Joseph, p. 55.
[2]Quoted in: Jeremy Campbell (1986): *Winston Churchill's Afternoon Naps.* New York: Simon and Schuster, p. 172.

handful of other mammals —is a remarkable exception to this general rule. When and why have such few species developed the ability to sustain wakefulness for relatively long periods (16-18 hr)? Is adult human behavior indeed monophasic, or is it just a masked expression of an underlying polyphasic drive with multiple alternations between states of alertness?

Human napping, whether in the form of an afternoon siesta or as the multiple napping behavior in the infant, in old age, or in adults during time-free studies, has never been considered more than an incidental phenomenon and has not attracted much experimental attention. Only recently have the underlying bases of polyphasic rest-activity (or sleep-wake) patterns and their relationship with human behavior been studied. The question of whether humans are tied to a pattern of sleep that requires one long sleep per 24 hr is not of trivial importance. It is estimated that 25% of the active population in industrialized societies work some sort of shifting or irregular schedule. Strategies for responding to a crisis (e.g., natural or manmade catastrophes, earthquakes, volcanic eruptions, oil spills) involve sustained human functioning beyond 24 hr. In these circumstances, the 8-hr nocturnal sleep is disrupted at best, and in many cases is completely impossible. How efficiently —and how healthfully —can humans operate on several short periods of sleep if their work schedules require it?

Polyphasic sleep-wake and multiple napping strategies have a potentially practical application in many areas concerned with maintaining and enhancing performance at different times of day and under a wide variety of around-the-clock activities, such as shiftwork in industrial settings, crew and passenger safety during air, sea, railway, and other transport operations, space flights, and human functioning in sustained emergency, survival, rescue, and defense operations. Industry has become increasingly interested in the past few years with the problems of human performance, mood, and safety during continuous operations. The scheduling of these shift systems and the judicious placement of sleep and naps within them are a key concern in these applied arenas.

However, the search for solutions for improving sleep management in high-demand situations is not the only reason for pursuing research in this direction. Polyphasic sleep research raises a number of fundamental issues, and provides remarkable tools for understanding the regulatory mechanisms of the sleep-wake system, as this volume attempts to demonstrate. This book explores the areas of sleep evolution and development, basic ultradian and circadian rhythms, occupational and performance aspects of polyphasic and ultrashort sleep, and their implications in sleep disorders medicine.

The present volume springs from a Workshop on Polyphasic and Ultrashort Sleep-Wake Patterns held in the Castello di Gargonza (Italy) in May 1988 under the auspices of the Commission of the European Communities (Medical and Public Health Research Programme). The Editor of this book

had felt the need for such a workshop—and for this book—for quite some time. Although the number of scientists involved in areas directly or indirectly related to polyphasic sleep research is relatively limited, there has never been an attempt to coordinate the different findings, to debate the state of the art and, most important, to establish guidelines for future studies in this pioneering field of research. It was felt that this would be of importance to a number of areas and applications.

The workshop focused on the theoretical, biological, chronobiological, medical, methodological, and with special emphasis on the human performance aspects of polyphasic and ultrashort sleep strategies. Particular attention was given to the issue of sleep management under sustained performance operations. The participants reported not only their own experiences and studies on the topic but, in particular, contributed with much discussion about several important issues related to polyphasic sleep. A number of guidelines for future research in this newly developing area was discussed and developed during the workshop, including the possible application of such sleep strategies in extreme or sustained operations. The workshop was attended by a selected group of scientists coming from different, though interrelated, backgrounds. They included, among others, sleep researchers, sleep disorders physicians, psychologists, physiologists, chronobiologists, and shift work, naval, and aerospace medical specialists.

The present volume has a broader scope with respect to the original structure of the Gargonza meeting. The topics and issues that are discussed in the book include not only the contributions presented at the workshop but also more recent studies, analyses, and reviews conducted by their authors and colleagues subsequent to the meeting. Additional chapters directly related to the central theme being debated have been written by invited authors who were not able to attend the meeting. As a result, the volume represents a comprehensive, extensive, and updated review of the major areas related to ultrashort and polyphasic sleep.

The themes discussed in the volume are of interest not only to sleep scientists, chronobiologists, and sleep disorders physicians but also to physiologists, psychologists, psychiatrists, and to all those who are concerned with work under emergency, extreme, or survival situations, as well as to those professionals who deal with the maintenance of optimal human performance and alertness under circumstances of sleep loss or irregular work schedules (e.g., paramedics, shiftworkers, and their managers, mission control specialists). The book is also addressed, and should be of interest, therefore, to professionals, to syndicate, union, and government leaders who are concerned with how sleep can be obtained in a way that will lessen or prevent the decrease in performance that often accompanies shift or sustained work schedules, and that sometimes leads to accidents, sleep disorders, and other health problems or loss in productivity.

The study of a putative polyphasic tendency .or capacity of the human sleep system represents a broad physiological interest both for speculative

issues related to the function of sleep and for its practical implications for human functioning. The Editor hopes that this volume, and the exciting issues and experimental data presented, will serve as a starting point and will stimulate further research in the area of polyphasic alertness behavior.

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WHY WE NAP

Evolution, Chronobiology, and Functions of Polyphasic and Ultrashort Sleep

1

Evolution, Chronobiology, and Functions of Polyphasic and Ultrashort Sleep: Main Issues

CLAUDIO STAMPI

In one of the first studies that examined the rest-activity behavior in a variety of animal species, Szymanski (1920) reported that several mammalian species exhibit numerous cycles (up to 10-12) of activity per day. Szymanski was the first to apply the term *polyphasic* to this fundamental and ubiquitous behavioral pattern, typical of most mammals, in which bouts of activity and rest alternate several times per day. Recent studies estimate that the majority (over 86%) of mammalian genera show typical polyphasic rest-activity patterns (Campbell and Tobler, 1984; see also Ball, Chapter 3, this volume).

The remaining mammalian species, which include many primates, show a very' distinct daily pattern. Normally, one consolidated period of sleep is followed by a continuous period of activity without any sleep, or with the intermission of a relatively brief rest-quiescence period. It appears that such species have developed the ability to sustain wakefulness for relatively prolonged periods (16-18 hr), without apparent need for intervening sleep, and without deficits in functioning. This is the *monophasic* activity pattern that is typical of, and well known for, the adult human,

Monophasic activity behavior appears therefore to be an exception to the general rule of the animal kingdom. Why have humans and a handful of other mammals developed such a rest-activity pattern? What survival advantages and sleep functions relate to monophasic behavior, as opposed to polyphasic sleep? Where in evolution does monophasic behavior become dominant and what factors relate to this change? Indeed, is adult human behavior *purely monophasic,* or is it just a variant of a (damped) polyphasic behavior? Or, in other words, do humans *need* to take sleep in one continuous period, or can sleep be taken in several naps?

The question of whether humans are irrevocably tied to a monophasic

sleep period is not of trivial importance. Sleep occupies a major portion of the life span of most species, including *Homo sapiens,* and yet its functions and processes are still poorly understood. Sleep is one of the most pervasive behavioral controls in nature, to the point that its quality, duration, and pattern can dramatically affect wake functioning and performance- The operational decrements in performance resulting from lack of sleep or irregular sleep-wake patterns are well known and are becoming increasingly important in today's "around-the-clock" society. It is estimated that about 25% of the active population in industrialized countries works some sort of shifting schedule. Insufficient or inappropriate rest has been the cause of, or a strong contributing factor to, several recent accidents that reached catastrophic proportions (e.g., Chernobyl, Three Mile Island).

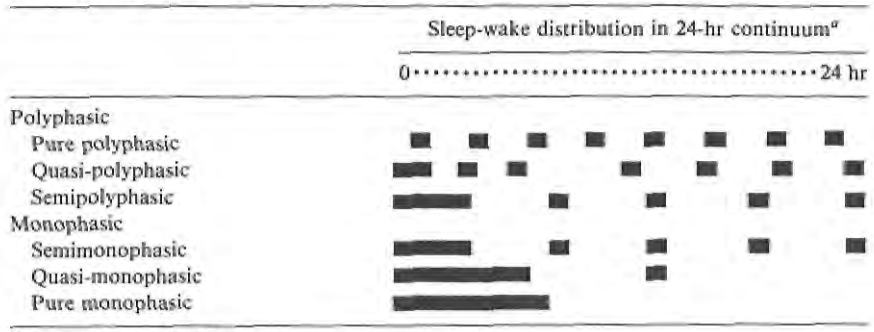
In this framework, it is essential to understand the factors affecting the need for sleep at different moments of the 24-hr continuum, especially during irregular or abnormal patterns of rest and activity, when nocturnal, uninterrupted 8-hr sleep is not possible. Polyphasic sleep-wake strategies have potential practical applications within many areas concerned with maintaining and enhancing performance at different times of day and under a variety of circumstances. Yet, while the search for solutions for improving sleep management in the workplace or in high-demand situations represents a crucial and necessary objective, these are not the only reasons for pursuing research in this direction. As discussed in this and subsequent chapters of this book, polyphasic sleep research raises a number of fundamental issues, and provides remarkable tools for understanding the regulatory mechanisms of the sleep-wake system.

Definitions

Before these issues are addressed, the differences between various types of phasic behavior should be defined. What are proposed here are purely semantic definitions because, from a methodological perspective, it is often difficult to classify a certain species within a specific category. For example, some species vary in their sleep-wake behavior according to such factors as age, season, habitat, or exposure to danger. Hence, they may be classified in different categories depending on changes occurring in these factors. For this reason, this classification scheme applies to *behaviors* rather than to *species* (e.g., one species may show different phasic behaviors). The reader should keep such methodological aspects in mind when examining Table 1.1, where schematic examples of sleep-wake behavior in the 24-hr continuum are presented (based on animals taking a total of 8 hr sleep per day).

Phasic behavior is divided into two major categories: *polyphasic* and *monophasic.* Each major category is divided into three subgroups, ac-

TABLE 1.1. Definition of *polyphasic, monophasic,* and intermediate behaviors



[a]See text for explanation.

cording to whether the behavior under study strictly respects the definitions of one or the other category. Hence, a *pure polyphasic* behavior occurs when episodes of sleep and activity are evenly distributed throughout the 24 hr (see Table 1.1). *Quasi-polyphasic* behavior applies when sleep-wake episodes are not evenly distributed throughout the 24 hr (e.g., when there is a preference for diurnal activity and nocturnal rest, or vice versa). Behaviors are considered polyphasic *(pure* or *quasi-)* provided that *less* than 50% of total sleep time is taken continuously. If *more* than 50% of total sleep time is taken as one continuous episode, this will be included in the *monophasic* category. Analogously, *pure monophasic* behavior occurs when *ail* sleep is taken in the form of a single daily episode. *Quasi-monophasic* applies to when more than 50% of sleep is taken in one episode, and the remaining amounts in one or more episodes. Finally, there is an intermediate category, which can be termed indifferently either *semipolyphasic* or *semimonophasic.* This occurs when exactly 50% of sleep is taken as a single episode, and the remaining portion as one or more episodes.

Examples for all groups and subgroups can be given by examining human behavior across ages or under different conditions. At birth, humans are *pure polyphasic,* with regular episodes of sleep and wakefulness at about every 4 hr. Soon they become *Quasi-polyphasic,* since longer sleep episodes tend to occur in the nocturnal period, although many naps are still present in the daytime. When uninterrupted sleep occurs for more than 50% of total sleep time, children become *quasi-monophasic.* Finally, many, but not all, adult humans may eventually become *pure monophasic* when they have just one sleep episode per day. Under conditions preventing normal nocturnal sleep, such as during shift work or sustained operations, adult humans may have a reduced nighttime (or daytime) sleep episode, supplemented by one or more naps, hence following a *semi-* or *quasi-polyphasic* behavior, or any variant of the above.

Phasing Behavior under an Evolutionary Perspective

What factors contributed to the radical differentiation of adult human sleep, from the ubiquitous polyphasic rest-activity pattern in nature, into its apparently monophasic pattern? Is monophasic sleep just a variant of the general rule? To address these issues, it is important to examine in detail the patterns of sleep duration and placement within the 24-hr day in an evolutionary perspective. However, an evolutionary assessment of sleep is certainly not a simple task, because it is complicated by the methodological difficulties that may arise in the evaluation of sleep duration in animals, not least of which is the definition of sleep itself. As thoroughly discussed in a comprehensive review of sleep duration across phylogeny (Campbell and Tobler, 1984), some studies relied solely or mainly on behavioral observations, whereas others included (or were limited to) electrographic recordings. It clearly emerged that electrographic recordings without simultaneous behavioral observations may be of limited value in the examination of sleep duration. However, since the criteria for defining behavioral sleep are often inconsistently applied, a purely behavioral characterization of sleep in the absence of electrographic correlates may also lead to unreliable or misleading information.

There is another major problem encountered when monitoring sleep in animals who are not in their natural habitat, the situation characteristic of the greater portion of studies on animal sleep. The inactivity induced by the confined environment may profoundly alter the sleep pattern observed. We know that humans in similar circumstances show profound differences in their sleep-wake patterns, and also take more sleep (see Chapter 6). Analogously, there is general agreement that the sleep durations recorded from an animal well adapted to laboratory conditions reflect the animal's *maximal* capacity for sleep, rather than its typical sleep pattern under natural conditions. Finally, another "dilemma" encountered by many sleep investigators is how to classify "drowsiness" or "light sleep," which is found in many animals (e.g., in ruminants). Zepelin and Recthschaffen (1974) have addressed the problem by attributing half of drowsiness time to the sleep quota, and half to wakefulness, but even the authors themselves were not completely satisfied with this solution. At present, no general agreement concerning the differentiation between sleep and such intermediate state(s) exists; obviously, therefore, any analysis involving sleep episode durations will be dramatically affected by the different interpretations given to "drowsiness" by the investigators.

Such limitations must be kept in mind when sleep questions are raised and discussed. Is monophasic or polyphasic sleep the original evolutionary pattern? Are there demonstrable genetic bases for the phasing of sleep? Have there been evolutionary tendencies toward either pure monophasic or pure polyphasic patterns? Whatever the function served by sleep, it could be

argued that by promptly minimizing the buildup of a putative "need-for-sleep" factor during wakefulness — by sleeping as often as possible—the polyphasic pattern would result in an adaptive benefit for the animal. Hence, in this case polyphasic sleep would represent an advantage. Conversely, polyphasic sleep may not be appropriate for species requiring larger amounts of time and effort ("cost") to find, create, or move to a safe sleep site, or for those for whom staying awake during a specific portion of the light-dark cycle —for example, at night for humans and many other species —may be either risky, or less appropriate for hunting, feeding, or other essential activities. Such species may have developed the ability for sustaining wakefulness for prolonged periods — hence developing monophasic or quasi-monophasic patterns.

Intuitively, though, the simplest explanation for the reason why some animals require polyphasic sleep (or are prevented from a monophasic behavior) appears to be related to energy demands. Very small mammals have very high metabolic rates. These animals are forced to spend most of their waking time foraging for food—for example, species of shrew have to "eat their own body weight of food" (Horne, 1988, p. 233) each day to survive. It is quite likely that animals with such high energy expenditure levels cannot afford to sleep uninterruptedly for prolonged periods: they must keep eating as often as possible. Indeed, due to their high metabolic rates, some of these animals will not survive for more than a few hours if they are prevented from feeding. Similarly, the polyphasic sleep-wake behavior in human newborns may be generated by a need for frequent feeding. However, a precise understanding of this matter across species is still open for speculation; while a substantial correlation was found between total sleep time and metabolic rate (Zepelin and Rechtschaffen, 1974), there have been no studies investigating the relationship between metabolic rate and phasing behavior.

Are animals Living in the tropics any different from those living in colder environments with respect to phasing behavior? Does environmental temperature, or an animal's thermoregulatory processes, influence the expression of polyphasic sleep? In Chapter 2 Pier Luigi Parmeggiani reviews basic and fundamental aspects of the interaction between thermoregulation and sleep. These regulatory mechanisms are particularly important for the control of the ultradian wake-sleep cycle. In particular, Parmeggiani shows that the anterior hypothalamic preoptic thermoregulatory structures influence the wake-sleep cycle in mammals, whether or not in the presence of thermal loads. Ambient temperature may elicit selective depression or enhancement of specific sleep (or wake) states in terms of their duration and frequency. Parmeggiani's chapter demonstrates the important role played by interactive regulatory mechanisms occurring between ambient and/or body temperature, and the sleep-wake system.

What are the relationships between the phasing of sleep and waking behavior across species? Although no systematic study has yet assessed a direct relationship between sleep phasing and the habitat demands of

certain species, the general impression is that mammals living under very dangerous or adverse conditions have highly polyphasic behavior. For instance, sleep is a particularly vulnerable state in the giraffe, an animal that takes 10 or more seconds to stand up. The giraffe lies down for periods between 3 and 75 min, three to eight times a night (Kristal and Noonan, 1979). Although most of this time is spent awake, it is assumed that the giraffe gets a total of only 2 hr of (fragmented) sleep per 24 hr. The blind Indus dolphin *(Platanista indi)* lives in turbid and muddy waters and has to keep constantly on the move to avoid injury on rocks. Hence, it would appear that sleep poses a particular hazard for this species, and indeed it was found that its sleep episodes last for about 90 sec only, for a total of 7 hr per day (Pilleri, 1979). Intriguing cases of polyphasic sleep occur in other aquatic mammals, such as the bottle-nosed dolphin *{Tursiops truncatus;* Mukhametov et al., 1977) and the Black Sea porpoise *{Phocoenaphocoena;* Mukhametov and Polyakova, 1981). These animals show approximately 1-hr periods of sleep involving one cerebral emisphere at a time, separated by 1-hr of wakefulness in both emispheres; this 3-hr cycle is repeated throughout the night. That is, the animals are never entirely asleep.

What survival trade-offs exist for polyphasic versus monophasic sleep? Is there a correlation between the degree to which an animal is preyed upon and phasing? Most of the issues mentioned above and many others are addressed by Nigel Ball in Chapter 3, in which he examines from different perspectives the biological significance of the phasing of sleep in animals. As Ball points out, one approach for understanding the role played by phasing would be to manipulate such behavior experimentally by inducing polyphasic sleep in monophasic species or vice versa. One of the most drastic approaches for inducing polyphasic sleep in monophasic or quasi-monophasic mammals is obtained by suppressing their circadian rhythms via lesions of their supra-chiasmatic nuclei (see Chapter 3). In humans, induction or facilitation of different types of polyphasic behavior has been done extensively by adopting a variety of methods. Findings from these studies are discussed later in this chapter as well as in Chapters 6, 7, 8, 10, 12, and 13.

Are there qualitative or quantitative differences in the sleep achieved by animals with monophasic or polyphasic sleep patterns? Specifically, are short sleep episodes equally, more, or less efficient than equivalent-duration longer sleep episodes in meeting an animal's total sleep need? Very few studies have addressed this last issue and, as is discussed in subsequent sections of this chapter (and in Chapters 10 and 12) they have been generally limited to humans.

Ontogeny of Phasing Behavior

Evidence that polyphasic sleep may not be totally unknown to humans does not arise just from the striking observation that such behavior is so prevalent in nature. Many other factors suggest that adult humans may in

fact have a "natural" ability to adapt to such patterns; such factors will now be briefly examined.

Sleep is not monophasic throughout the entire life span of humans. Indeed, polyphasic sleep is the typical pattern in infants, who show a 3- to 4-hr sleep-wake behavior. Interestingly, an infancy period of polyphasic sleep is not restricted to the human species. Why do species whose adult sleep pattern is monophasic invariably have polyphasic young? The study of the transition from the typical polyphasic infantile sleep pattern, toward increasing consolidation and lengthening of night sleep (quasi-polyphasic), with final reduction or disappearance of daytime sleep (naps) at or before puberty (quasi-monophasic), played a significant role in the history of sleep research. These early pioneering reports (Gesell and Amatruda, 1945; Kleitman and Engelmann, 1953; Parmelee et al., 1964) also showed that there is a rapid decline in human sleep duration, from about 16 hr at birth, to 14 hr by the first month, to 12 hr by the sixth month, reaching the average of about 8 hr per day in the young adult. What determines the change from the polyphasic nature of infant behavior into the usual adult monophasic pattern? Does this tendency toward a prolonged nocturnal sleep period reflect the development of a genetically determined fixed system, the maturation of basic rest-activity rhythms, a change in sleep need and function, or a response to social pressures for prolonged daytime wakefulness?

The frequent reappearance of daytime naps in the elderly has also been a subject of considerable interest (Tune, 1969). Given that circadian rhythms show a tendency toward flattening of their amplitudes with increasing age, this has often been alleged as a possible cause of sleep fragmentation. Or does this partial return to multiple daytime napping, observed not only in the elderly, but also at most ages when the opportunity exists (see Chapter 6), mean that adult humans may exhibit a "masked" or "damped" polyphasic sleep tendency? The mechanisms of such developmental changes across ontogeny are reviewed in two chapters of this volume. In Chapter 4 Piero Salzarulo and Igino Fagioli examine in detail the dramatic and fundamental maturational steps in the physiology of sleep and wakefulness in the first year of human life. The processes occurring during this period are crucial for explaining and understanding the transition from polyphasic to monophasic behavior. The studies they review clearly show that toward the end of this phase the internal organization of sleep appears to anticipate many aspects of that of the adult. However, there are several important differences. For example, although at this age (5 mo-1 yr) infants still show several daytime naps, Salzarulo and Fagioli found that the secondary, midafternoon peak in slow wave sleep (SWS) that appears to be present in the adult (see Chapter 9) does not occur in the child. Wilse B. Webb, in Chapter 5, presents a global overview of the ontogenetic development of sleep in humans, and extends the analysis from birth to the elderly. He introduces a behavioral model of sleep regulation and discusses its implications for ultrashort and polyphasic sleep schedules. Webb assumes that

sleep characteristics are primarily determined by a sleep demand component, a circadian timing component, and behavioral facilitators and inhibitors.

Behavioral determinants are certainly crucial factors in allowing or preventing the expression of a putative polyphasic sleep tendency or ability. Basically, two opposite methodological approaches have been used to understand the role played by behavioral (exogenous) and biological (endogenous) components. One approach is to place subjects in environments that facilitate or *encourage* the expression of the endogenous biological sleep drive. Such conditions include: isolating subjects from time cues; confining subjects to bed, with or without specific instructions prohibiting sleep; and restricting behavioral options in the environment (so-called disentrained environment, with no activities such as reading or writing allowed; see Chapter 6). The second approach to the study of behavioral facilitators and inhibitors is to *interfere* with the endogenous sleep-wake system by means of different types of manipulations, such as by submitting subjects to irregular, abnormal, or even polyphasic schedules.

Encouraging the Expression of Biological Components of the Sleep-Wake System

The first type of studies, those conducted in unstructured environments, is reviewed by Scott Campbell (Chapter 6). These studies, which have greatly advanced our understanding of the circadian regulating system, have demonstrated that in addition to the major sleep episode, there is a strong propensity for "afternoon naps" (subjective time) taken around the time of body temperature maximum. Incidentally, despite the fact that naps were specifically prohibited in all but a few time-free studies, subjects still managed to nap quite often. However, the "postlunch dip" may not be the only time at which sleep (or nap) tendency is high. Under less-structured environments multiple naps do occur throughout the 24 hr, and many subjects exhibit polyphasic sleep similar to that observed in nonhuman species. Such spontaneous naps do not recur randomly throughout the day. Rather, their striking regularity allowed speculation on the existence of an ultradian 4-hr component of the sleep-wake cycle that may be superimposed on the more robust circadian and midafternoon components. In fact, 4-hr cycles in sleep propensity have been found in many studies conducted in unstructured environments (e.g., Nakagawa, 1980; Zulley, 1988). These findings are probably not surprising to investigators familiar with the 4-hr sleep-wake pattern observed in infants. More recently, 4-hr cycles in the expression of SWS have also been found in the frequent daytime naps in a population of narcoleptics (see Chapter 15).

It is worth mentioning that anthropological studies conducted in tribes

active at night show that human sleep can be highly polyphasic in certain cultures. Although they have different cultures and ways of life, both the Temiars of Indonesia and the Ibans of Sarawak have similar polyphasic sleep-wake behaviors (Petre-Quadens, 1983). Their average nocturnal sleep episode duration ranges between 4 and 6 hr, and nighttime activities (fishing, cooking, watching over the fire, rituals) at any one time involve approximately 25% of the adult members. Daytime napping is very common in both tribes: at almost any time of day, about 10% of the adult members are asleep. Whatever the cause of these polyphasic sleep patterns, whether the expression of an inborn ultradian rest-activity tendency or other factors, such populations exhibit extremely flexible and fragmentary sleep-wake cycles. The minimal contact with modern civilization could be one of the reasons for the preservation of this possibly ancestral sleep pattern.

Interfering with the Biological Sleep Drive

A considerable body of studies have manipulated, in one way or another, and with more or less extreme constraints, what are considered to be the *normal* human sleep-wake habits. Yet, before such studies are discussed, it is important to point out that every day and night, all over the world, millions of individuals and workers are exposed to one form or another of abnormal or irregular sleep schedules. This occurs, for example, after time-zone transitions, during shift work, and during more specialized quasi-continuous operations. Under these conditions work and sleep are being attempted at unsuitable times of the circadian cycle, that is, they are taken when this is *possible* rather than when it might be more *appropriate* (as determined by habit and by endogenous factors). In such circumstances sleep is often shortened or split into several episodes (polyphasic sleep). In the first of a series of chapters in this volume to examine the effects of altered sleep-wake schedules, David Minors and James Waterhouse (Chapter 7) describe their investigations on what effects irregular sleep-activity schedules have upon the circadian timing system. They have also looked at whether circadian rhythms are influenced by the timing of "split" sleep. When an irregular sleep-wake cycle is imposed on subjects isolated from time cues, synchrony with the solar day is lost; their rhythms free-run with a period greater than 24 hr, as a sign of lack of regular Zeitgebers. Minors and Waterhouse have demonstrated that such free-running rhythms are prevented, and stable 24-hr rhythms obtained, when sleep is split into two 4-hr periods, with one of these at conventional sleep time (2400-0400). Rhythms remain stable even when the other 4 hr are taken irregularly. For this reason the nocturnal 4-hr sleep was termed "anchor" sleep, and findings were confirmed by performing "constant routines" before and after anchor

sleep. Minors and Waterhouse also present a new analysis protocol by which the phase of the endogenous components is assessed without the need for constant routines.

These findings show the great potential of anchor sleep for improving stability in circadian rhythms, and hence adjustment to shift work, for those working irregular schedules. This topic is also discussed by Stampi in Chapter 10, where he reports studies in which the concept of anchor sleep has been pushed a little further. In these studies subjects successfully followed a polyphasic sleep schedule consisting of 4 hr of nocturnal anchor sleep, plus 4 hr divided into multiple 20-, 50-, or 80-min daytime naps.

The Ultradian Components

As was brought up earlier in this chapter, the study of the ultradian components of the sleep-wake system is fundamental for understanding whether adult humans may have a natural ability to adapt to polyphasic sleep patterns. Such studies have indicated that pressure for sleep may not depend solely on homeostatic mechanisms (i.e., the amount of prior wakefulness), for there are also robust oscillatory components. Among the various techniques that have been adopted to investigate the "sleep propensity function," one is of particular interest in this context. It imposes what appears to be an extreme exaggeration of the concept of polyphasic sleep. Subjects are asked to live on a 20-min "day" for periods up to 48 hr: they are instructed to attempt sleep for 7 min and remain awake for 13 min (earlier versions had a 5/15 schedule). Can individuals get any sleep under such an extreme schedule?

Using this paradigm, Peretz Lavie (Chapter 8) has shown that subjects can sleep for up to 90% of the 7 min allotted bed time at times when sleep pressure is normally high (e.g., at night). In other words, sleep latencies become very short at these times. These studies also confirmed the existence of primary (nocturnal) and secondary (midafternoon) "sleep gates" in which transition from wakefulness to sleep is facilitated. Interestingly, the paradigm also revealed a "forbidden zone" for sleep in the early evening in which even sleep-deprived subjects show greater difficulty in falling asleep.

Ultradian variations in diurnal "sleepability" were also detected by Lavie, showing preferred cycles in the 1.5- to 2-hr range {supporting Kleitman's (1961) prediction of recurrent fluctuations in alertness during wakefulness—the Basic Rest-Activity Cycle, or BRAC], as well as in the 4-hr range, as found in other studies mentioned earlier in this chapter. It is known, on the other hand, that within sleep there are recurrent phases during which sleep is more frequently terminated, which have been associated with REM sleep. What is the functional value, in the 24-hr continuum, of such multiple "gates" that facilitate initiation of sleep during wakefulness, or

wake-up during sleep? *Do* they serve the function of providing multiple transition points to ensure a smooth passage from one state to the other, should such need arise? This issue is of considerable practical importance in examining the potential of polyphasic sleep schedules in continuous work scenarios. The polyphasic approach of submitting subjects to multiple naps several times per day (though less extreme than Lavie's 20-min day) is also at the root of one of the most widely used diagnostic tests for evaluating daytime sleepiness: the Multiple Sleep Latency Test (MSLT; Carskadon and Dement, 1977).

The Siesta, or the "King" of Naps

The studies described above have demonstrated the robust propensity for a period of midafternoon sleep or inactivity. This tendency, however, is not limited to unstructured environments, as shown by the hundreds of MSLTs routinely done in sleep laboratories. Afternoon naps are a common feature of the sleep of healthy adults who possess the flexibility in work-rest schedules that permits napping to take place. Is afternoon napping merely the consequence of a meal (the "postlunch dip"), or an effect of the higher ambient temperature at that time of day? Or is there a biological basis for the siesta? Is this phenomenon limited to man? Well before sleep researchers started to look closely at the matter and to suspect the existence of a biological rhythm for the midafternoon quiescent phase (e.g., Broughton, 1975), those investigating circadian rhythms were already quite familiar with the issue. Jurgen Aschoff (1966), in his paper "Circadian Activity Pattern with Two Peaks," had described the wide occurrence, across species, of a daily bimodal endogenously generated pattern of rest and activity. It may be surprising to some that a recent survey conducted in Switzerland on a representative sample of the adult population showed that as much as 24% of those surveyed considered themselves "nappers" (Wirz-Justice et al., 1991). Another study in the United States showed that 61% of the population naps at least once a week (see Chapter 9). Interestingly, the Swiss study found that as soon as men were working part-time or became unemployed, the incidence of naps more than doubled.

There is considerable evidence that most persons can nap if asked to do so. What distinguishes nappers from nonnappers? What are the positive or negative effects of naps? Do naps adversely affect subsequent nocturnal sleep? As David Dinges shows in Chapter 9, there is no evidence that naps compromise nocturnal sleep duration or quality. He examines the extent to which napping patterns are present in healthy adults and looks also at their infrastructure, showing that naps are not miniatures of nocturnal sleep compressed in time. Rather, their architecture depends on many factors including time of day, nap duration, and prior wakefulness. Three types of

napping behavior are identified. *Replacement* naps are taken in response to subjective fatigue, usually as a consequence of reduced nocturnal sleep. *Appetitive* naps are taken without regard to fatigue, as part of a habitual biphasic sleep cycle. *Prophylactic* naps may be taken as a strategy to "store" sleep in advance of a period of sleep loss, such as during shift work.

Factors Affecting Nap Recuperative Value

Although "sleep inertia" —the phenomenon of impaired alertness usually experienced upon awakening from sleep —can also occur upon awakening from naps, this inertia almost always gives way to beneficial effects on mood and performance. Indeed, naps have often been shown to have powerful —and disproportionate to their length — recuperative effects on performance. Because sleep inertia is normally dissipated within 15 min upon awakening, its effects do not appear to pose any limitations under normal conditions, that is, following a nocturnal sleep episode or a midafternoon nap. However, during polyphasic schedules individuals go to bed —and hence wake up —many times per day. Sleep inertia may be detrimental to effective functioning immediately upon awakening for individuals engaged in polyphasic schedules. Do sleep-inertia levels and duration increase, decrease, or remain the same under polyphasic sleep? In other words, is there a "learning" or adaptive effect that may allow gradual reduction of this potentially limiting factor? To what extent is sleep inertia affected by factors such as sleep stage at arousal, nap architecture, time of day, and prior wakefulness? Or, more generally speaking, what are the most important factors that influence the global restorative power of a nap?

Sleep inertia, as well as many other "technical" factors affecting nap recuperative value, all of which must be carefully considered in the design of polyphasic or ultrashort sleep schedules, are discussed extensively and from different perspectives in Chapters 10, 12, 13, and 14. These chapters are part of the two central sections of the book introducing and reviewing the concepts of sleep management under conditions of continuous work. Indeed, an increasing number of occupations require individuals to follow prolonged, around-the-clock periods of quasi-continuous work. Often they also involve performance of essential services and crucial high-responsibility tasks (e.g., space mission control crews, rescue teams after a catastrophe). In such situations, demands for activity, performance, or attention can be prolonged or recur at short intervals. This prevents the individual from sleeping in the habitual monophasic 6- to 8-hr nocturnal manner, and in such situations traditional shift-work schedules are not applicable. Sleep tends to be dramatically reduced and disrupted, resulting in severe decrease in performance and decision-making efficiency.

Phasic Behavior and Sleep Management under Sustained Performance

How can the need for sleep be satisfied during prolonged sustained performance to maintain reasonable levels of functioning? If humans could adapt to polyphasic sleep patterns, as most of their not-so-distant relatives do in nature, this strategy would appear to be the most obvious solution. This was proposed by the author (Stampi, 1985, 1989), who showed evidence that adult humans may indeed have an endogenous ability to adapt to polyphasic patterns. This is suggested not only by the evolutionary, ontogenetic, and sleep-wake rhythmic regulatory mechanisms mentioned so far. A surprising degree of facility to adapt to multiple napping regimens is suggested by a number of laboratory and field studies in which adult humans were engaged in various types of polyphasic schedules. Such studies (reviewed by Stampi in Chapter 10) include: (1) the first experiment of "interrupted" sleep (Husband, 1935); (2) subjects living under short "days" (90-, 160-, or 180-min sleep-wake cycles); (3) the spontaneous polyphasic sleep patterns normally followed by isolated cultures, as described previously; or (4) polyphasic sleep adopted as a result of extreme demands; (5) scheduled polyphasic regimens, with or without "anchor" sleep and with or without sleep reduction; and (6), studies of nocturnal sleep fragmentation.

The putative ability of humans to adapt to various types of napping regimens, as suggested by these findings, raises a number of challenging conceptual and methodological questions, relevant to the design of polyphasic schedules for actual sustained operations. To what extent must the timing of multiple sleep episodes respect the underlying endogenous rhythms ("gates") of the sleep-wake system? Conversely, how does adopting polyphasic patterns affect or disrupt such rhythms? Do adult humans vary in their constitutional ability to adapt to polyphasic patterns, and what determines this ability? This may or may not be analogous to what occurs under rotating shift work, where some people adjust easily, whereas others, especially after age 45, appear totally incapable of adapting. What are the consequences of polyphasic sleep for health and functioning, and for how long can these schedules be undertaken?

One of the key questions of sleep management is to understand what is the *minimal* sleep duration necessary to maintain an acceptable level of performance. There is a general consensus that individuals can adapt to 4.5-5.5 hrs of nocturnal sleep if this is achieved gradually. However, as Paul Naitoh points out in Chapter 13, under most environments that demand sustained work, a period of 5 hr of uninterrupted sleep is too "luxurious" and cannot be set aside because it conflicts with work schedules. What other sleep reduction strategies exist that may allow "capturing" of minimal sleep? Such techniques, including polyphasic sleep, are described

in Chapters 10 and 13, where their relative advantages and disadvantages are also discussed. The effects of partial and total sleep deprivation on mood, performance, and subsequent recovery sleep architecture are also described.

Paul Naitoh proposes a conceptual model of ultrashort sleep that could allow individuals to determine how long a nap period should be to achieve sufficient performance recovery, after a given amount of wakefulness and at a given point in the circadian phase. One of the principles of the model is that factors affecting nap recuperative value may vary considerably from individual to individual, a point also made in Chapter 14. There, Robert Angus and colleagues report on a research program conducted over several years in the field and in the lab, designed to estimate the effects of sustained mental work and sleep loss on a range of cognitive abilities. The aim of their studies was to provide guidelines for performance limits and rest requirements for military and civilian personnel engaged in around-the-clock operations. Many important methodological issues related to the assessment of performance are brought up. They have also found that 2-hr naps after approximately 40 hr of continuous work and sleep loss are beneficial and can provide temporary increase in performance.

Minimal Sleep

Paul Naitoh introduces the concept of sleep "quantum," that is, the smallest fundamental quantity of sleep required for restoration to take place. This appears to be on the order of 10 min, as studies of sleep fragmentation (Bonnet, 1986) and of solo sailors' sleep patterns (Chapter 10) suggest. Are there "optional" portions within sleep, as has been suggested (Horne, 1988), that could be avoided under sleep reduction regimens? Assuming that minimal or "core" sleep is on the order of 5 hr per day, are 5 total hours of polyphasic sleep more or less recuperative than 5 continuous hours of sleep? The "continuity" theory of sleep (Chapter 13) predicts that only a period of uninterrupted sleep is recuperative. However, in Chapters 10 and 12 Stampi reviews studies and presents preliminary data in which individuals under polyphasic and ultrashort sleep schedules functioned at levels equal to, or higher than, when they were under comparable amounts of monophasic sleep.

Examining the same problem from a physiological perspective, it could be asked whether a polyphasic sleep pattern alters total sleep need. That is, does the minimum amount of sleep optimally required increase, decrease, or essentially remain unchanged? In other words, is sleep under a polyphasic schedule more or less efficient? Preliminary experimental evidence (see Chapter 12) appears to confirm a hypothesis recently proposed (Stampi et al., 1990) that, contrary to the "continuity" theory of sleep, polyphasic

sleep appears to be the *only* way to achieve dramatic levels of sleep reduction (even beyond the 5-hr "limit") without compromising performance effectiveness.

It appears, however, that this hypothesis was proposed and "tested" as early as half a millennium ago. Among the numerous legends that circulate about Leonardo da Vinci, one deserves particular attention in this context: apparently Leonardo adopted ultrashort sleep to enhance his productivity. He would sleep 15 min out of every 4 hr, for a daily total of only 1.5 hr of sleep. While Stampi was unable to find any historical proof concerning this intriguing enigma (see Chapter 12), one report and one recent study, in addition to the issues brought up so far, suggest that Leonardo's apparently "impossible" idea makes significant biological sense.

In Chapter 11, Giancarlo Sbragia, a renowned Italian dramatist and actor, reports on how he learned of, and became intrigued by, Leonardo's peculiar sleep pattern and why he decided to try it on himself. He describes how difficult it was to adapt, in the first 2-3 wk, to such a schedule, and how he eventually succeeded in adjusting to a 22.5-hr waking day. He vividly explains how, after this initial adaptation period, this schedule appeared natural, and describes the "new world" that he discovered. And he also reports on the reasons that lead him to end his experiment after 6 months, reasons that *were not,* interestingly enough, due to fatigue, lack of sleep, or poor waking performance.

Stampi became quite intrigued by this report and decided to conduct an objective study on the matter. A series of experiments (Stampi et al., 1990; Stampi and Davis, 1991) is described in Chapter 12, in which a young volunteer attempted to follow Leonardo's ultrashort sleep schedule. In the first study the subject adapted particularly well to the 3-wk experimental period, to the point that he subsequently volunteered for a prolonged, 2-month study. Although he was not able to perfectly achieve Leonardo's extreme levels of sleep reduction, he succeeded in reducing his sleep to an average of less than 3 hr per day. These were divided into 6 naps of 30 min each, one every 4 hr. Sbragia's personal observations were confirmed, since performance was not significantly reduced compared to baseline levels. It also appeared that the gradual transition from monophasic to polyphasic ultrashort sleep patterns carried over several days played an important role in allowing smooth adaptation to the schedule.

Contrary to the usual findings of sleep-deprivation studies (see Chapters 10 and 13), in which only part of REM, and stage 2 sleep, but not total SWS amounts, are penalized, *all* sleep stages were proportionately reduced in this study. This was probably due to the much longer duration of this, compared to previous, studies. It may suggest that, in the short term, "optional" REM and stage 2 sleep can be reduced (within certain limits) to the advantage of "obligatory" SWS, but that the picture may change considerably over the long term.

This finding is just one among several examples of the value that the

study of polyphasic sleep schedules may have in contributing to the understanding of the laws that govern the sleep-wake system. Indeed, whether future research may prove that adult humans can adapt to polyphasic sleep, and whatever the value of such strategies under continuous work scenarios, polyphasic sleep is demonstrating its usefulness as a remarkable experimental tool.

Polyphasic Sleep and Sleep Disorders

This concept is further elucidated by studying polyphasic sleep from a different perspective, that is, as a consequence of, or associated with, the presence of sleep disorders. The use of a pathological model of sleep-wake alternation may be a means for understanding normal physiology. Patients with various forms of excessive daytime sleepiness exhibit a variety of phenomena including repeated microsleeps, short or prolonged diurnal sleep episodes, or remarkable increases of sleep per 24 hr, with or without nocturnal sleep fragmentation. In such circumstances patients are often overwhelmed by sleepiness which results in taking involuntary naps, or "sleep attacks," thus generating an involuntary polyphasic sleep pattern. Indeed, while voluntary naps taken by healthy individuals can be instrumental for functioning at high levels throughout the 24 hr, if naps occur involuntarily and are overwhelming, they may have just the opposite effect. Narcolepsy represents one of the clearest manifestations of this inability to maintain a stable monophasic sleep-wake cycle. The last two chapters of this volume are dedicated to exploring different aspects of this sleep disorder with regard to polyphasic behavior.

Is narcolepsy the pathological expression of an exaggerated tendency toward polyphasic sleep, that in healthy individuals is otherwise controlled? One possible approach to the reduction of excessive daytime sleepiness in narcoleptics is the judicious *scheduled* use of daytime naps, as a substitute for, or a complement to, medication. Preliminary evidence of the beneficial effects of scheduled naps has been reported (Godbout and Montplaisir, 1986; Roehrs et al., 1986; Rogers and Aldrich, 1988). It appears that in narcoleptics certain naps may be more refreshing than others, and improve subsequent performance. This appears to be associated with naps containing SWS. The effects of scheduled or unscheduled naps of different durations and at different times of day is reviewed by Michel Billiard and colleagues in Chapter 15. They also report on their own studies in which a 4-hr rhythm in SWS has been found in narcoleptic patients. Billiard and colleagues speculate that this 3- to 4-hr sleep-wake rhythm is normally present in healthy adults, as discussed previously in this chapter, but that it is made much more apparent in this pathological condition.

In Chapter 16, Hartmut Schulz and colleagues report on how they have

unsuccessfully tried to transform the polyphasic sleep-wake pattern of a narcoleptic patient into a strict, stable monophasic pattern. Although nocturnal sleep efficiency increased, even 3 wk of imposed daytime sleep restriction were not enough to stabilize this polyphasic pattern. By subsequently studying subjects in temporal isolation, they found that this condition did not change the polyphasic appearance of daytime naps. Narcoleptics have also been studied under the 7/13 min ultrashort sleep schedule, as Peretz Lavie reports in Chapter 8. By using this technique, he found that, contrary to normals who show BRAC periodicities in the appearance of REM only at night, narcoleptics have marked 80-min cycles of propensity to REM also in the daytime.

Conclusions

In summary, the research and issues reviewed in this chapter, many of which are developed in greater detail throughout the volume, pose a challenge to the concept that adult humans are irrevocably tied to a rigid monophasic sleep-wake system. Rather, they argue in favor of the hypothesis that, if need be, adult humans may adapt without major difficulties to some sort of polyphasic behavior. Indeed, the ability for a multiple napping behavior in adult humans may be the behavioral expression of an underlying biological rhythm of daytime sleep propensity.

However, the author would like to caution against misleading interpretations of these conclusions. What is being proposed here *is not* that polyphasic sleep is preferable to monophasic sleep, nor that everyone should now switch to a multiple napping behavior "panacea." It appears obvious that quasi-monophasic sleep —monophasic sleep plus occasional naps —is what comes most *naturally* to the majority of adult humans and a few other species. If somewhere in evolution such species have developed the ability to sustain wakefulness for relatively prolonged periods, most likely this ability occurred in response to some sort of important and advantageous adaptive pressure.

Yet, there is no indication that in this process such species have *completely lost the ability* to switch back to their primitive sleep-wake pattern. Indeed, the latter occurs throughout an important portion of their life span, or when the need arises. In other words, there is no proof that monophasic behavior occurs only to the exclusion of polyphasic sleep, nor that it requires its elimination from the system. To make use of an analogy, there is no reason why a skilled captain of a Boeing 747 should find major difficulties when flying a small, single-engine, one-passenger Cessna in which he probably learned the first rudiments of aviation many years earlier. The fact that he developed the ability to fly a sophisticated aircraft *may* require some additional *adaptation* time when he returns to the small

aircraft. Obviously, he cannot pretend to fly 6000 miles, or carry 400 passengers, with the small aircraft. On the other hand, the 747 is limited in certain ways with respect to the Cessna (e.g., it requires huge amounts of fuel, longer runways, complex maintenance, and so on). Therefore, in absolute terms, one aircraft is no better than the other: choosing which one to pilot depends on the situation and on the scope of any given mission. This last observation leads to the second hypothesis presented in this chapter.

Should adult humans be forced to reduce sleep by considerable amounts, polyphasic sleep may be *more efficient* than monophasic sleep. Unfortunately, experimental evidence for this hypothesis is limited to a handful of preliminary studies; this, in turn, may allow for some speculation on the matter. It could be argued that to be able to sustain wakefulness for periods of usually around 16 hr, humans may need to sleep more (total sleep) than if they were sleeping more often. That is, it is possible that a portion of prolonged nocturnal sleep is either "optional," as proposed by Horne (1988), or that it serves the function of filling "reserves" that enable the subsequent sustained wakefulness to take place. Under a dramatically reduced sleep regimen (e.g., 3 hr of sleep per day), this "optional" or "reserve" portion of sleep cannot be obtained (there is not enough sleep time). Hence, if such reduced sleep is taken in one episode per day (monophasic), this would prevent the ability to function effectively for much longer than a relatively limited time (e.g., a few hours), for no "reserves" could be accumulated. Conversely, if under a restricted sleep regimen short sleep episodes are taken many times per day, and *before* performance decrements occur, these amounts would be sufficient to keep the individual going for a few hours, after which time he would need to sleep again.

Such short sleep episodes would contain mainly or only "essential" or "core" sleep. If this is the case, there would be no need to spend additional sleep time for accumulating "reserves," even though subjectively the individual may probably still be feeling the need for additional sleep when the alarm clock sounds. This would explain why the only noticeable difficulty observed in the subject (Chapter 12) living under the "Leonardo da Vinci schedule" occurred, occasionally, at wake-up time. In fact, from time to time he would experience a considerable difficulty in waking up from certain naps; however, 10 to 20 min later he would usually become perfectly alert, remaining as such throughout the entire subsequent waking interval, without any signs of performance degradation or sleepiness. It is obvious, though, that under these conditions the individual may enter a rather delicate balance. However, a delicate balance that preserves the ability to function appears more advantageous than no balance at all and very poor performance effectiveness, as normally occurs, after a few days, to anyone sleeping just 3 continuous hours per day.

In other words, the recuperative value of sleep on performance may not

be linearly correlated with sleep duration; this is suggested by many studies presented in this volume. Indeed, even under sleep deprivation, short naps normally produce remarkable recuperative effects, disproportionate to their duration. Rather, nap recuperative power could be best represented by some sort of exponentially decaying (in time) function, providing high recuperative value at the beginning of sleep, which is gradually reduced (exponentially) as sleep continues. This function may be analogous, and perhaps parallel, to the exponential decay observed in delta wave (or SWS) activity during sleep (Feinberg, 1974; Borbely, 1982). As a corollary of this concept, which may carry practical interest for any "normal" individual living a "normal" life-style (i.e., not just for those dealing with quasi-continuous work), short naps unquestionably provide a strong refreshing power.

There is no doubt, however, that the most obvious conclusion emerging from the issues brought up in this chapter and throughout the whole volume is that the "laws" of sleep are still far from being totally understood. In this respect, Webb's (1975) comment on Samuel Johnson's description of sleep as a "gentle tyrant" is particularly appropriate: "To live on the best terms with a 'gentle tyrant' one must learn the rules by which he governs. Being gentle, he permits us certain freedoms to manifest our individual variations and differences; being a tyrant, he will not permit us to live in total freedom, and abuses carry their ultimate consequences." It is hoped that this volume will stimulate debate and especially more research with respect to the themes presented here, and that this may enlighten us about whether polyphasic ultrashort sleep, or any of its variants, are included in the rules accepted by the "gentle tyrant."

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I

Evolution, Development, and Regulation of the Sleep-Wake Cycle

2

Thermoregulation and Control of the Ultradian Wake-Sleep Cycle

PIER LUIGI PARMEGGIANI

In this chapter the relationship between temperature and wake-sleep regulation is considered primarily with respect to the ultradian wake-sleep cycle, that is, to the single sequence of wakefulness, synchronized sleep (NREM), and desynchronized sleep (REM) episodes. The issue is whether the influence of temperature on the wake-sleep cycle is either nonspecific, as a result of thermal comfort or stress influences, or specific, as due to the activity of thermoregulatory mechanisms. The experimental evidence (cf. Parmeggiani, 1987) shows that positive (warm) or negative (cold) thermal loads induce selective changes in the wake-sleep cycle. This occurs along with the overall increase in circadian waking or sleeping times characterizing the behavioral responses to thermal stress or comfort, respectively. Actually, such changes consist of selective depression or enhancement of single sleep stages in terms of duration and/or frequency. This shows that specific alterations are elicited by temperature in the mechanisms controlling the wake-sleep ultradian periodicity. In this respect, it is useful to consider whether the behavioral and autonomic thermoregulatory responses are either consistent or inconsistent with the behavioral and autonomic changes of sleep. On this basis, it is possible to understand why positive or negative thermal loads influence the ultradian wake-sleep cycle in so many ways.

A detailed analysis of sleep phenomenology is beyond the scope of this chapter. Only phenomena consistent with the issue discussed in this chapter will be taken into consideration. The interested reader is referred to Parmeggiani's (1980a) extensive review.

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Behavioral and Autonomic Phenomena of Sleep

Synchronized sleep is characterized by somatic and autonomic quiescence that minimizes energy expenditure. In this respect, the different sleep postures are behavioral thermoregulatory responses that cope with the sleep-dependent decrease in metabolic heat production. The most important autonomic event of synchronized sleep is a tonic increase in the parasympathetic outflow and an attenuation in sympathetic activity (Berlucchi et al., 1964; Baust and Bohnert, 1969). Accordingly, a downward regulation of cardiovascular (cf. Mancia and Zanchetti, 1980) and respiratory (cf. Phillipson and Bowes, 1986; Orem, 1988) activities is observed. Concerning thermoregulation (cf. Parmeggiani, 1980b; Heller and Glotzbach, 1985), body temperature decreases during synchronized sleep as a result of heat loss due to vasodilation in the heat exchangers (e.g., ear skin, upper airway mucosa) and to sweating (in humans).

Motor activity is impaired by general muscle atonia during desynchronized sleep (cf. Jouvet, 1967). The basic autonomic event is a tonic decrease in sympathetic outflow (Baust et al., 1968; Iwamura et al., 1969; Reiner, 1986) in the presence of tonic parasympathetic activity (Berlucchi et al., 1964; Baust and Bohnert, 1969). Moreover, random changes in autonomic activity, consisting of phasic increases and decreases of sympathetic and parasympathetic outflows, respectively, occur particularly in concomitance with rapid eye movements and myoclonic twitches. In short, during desynchronized sleep the homeostatic regulation of physiological functions, like circulation, respiration, and particularly thermoregulation, is impaired with respect to wakefulness and synchronized sleep (cf. Parmeggiani, 1980a).

Thermoregulatory Responses during Sleep

Thermoregulatory responses to thermal loads may be classified as behavioral (changes in posture and motility related to the search for thermal comfort) and autonomic (vasomotion, piloerection, changes in muscle tonus, shivering and nonshivering thermogenesis, panting, sweating). Long-term adaptation to thermal loads will not be considered here.

In mammals, thermoregulatory responses to changes in body or ambient temperatures are controlled by anterior hypothalamic-preoptic (AH-PO) integrative mechanisms that drive subordinate brainstem and spinal somatic and autonomic mechanisms (cf. Satinoff, 1978). In particular, positive thermal loads elicit vasodilation in the heat exchangers (skin, upper airway mucosa), panting, sweating, and decrease in muscle tonus, whereas negative thermal loads provoke vasoconstriction in the heat exchangers, increased muscle tonus, shivering and nonshivering thermogenesis, and piloerection. The reader is referred to extensive reviews (Parmeggiani, 1980b; Heller and

Glotzbach, 1985) for a detailed analysis of the changes in thermoregulation during sleep.

Behavioral thermoregulatory responses to thermal loads are observed only during wakefulness and synchronized sleep. In particular, the posture during synchronized sleep varies according to ambient temperature. In contrast, the muscle atonia during desynchronized sleep is unrelated to ambient temperature, and impairs any behavioral thermoregulatory activity.

Autonomic thermoregulation in response to thermal loads is normally operative during synchronized sleep. The AH-PO temperature regulator operates with a set range that is lower than that of wakefulness according to the functional quiescence of synchronized sleep. Autonomic thermoregulatory responses are suppressed or depressed during desynchronized sleep.

On the basis of the previous considerations, a distinction between sleep-promoting and sleep-regulating influences of temperature is appropriate (Parmeggiani, 1987).

Sleep-Promoting Role of Thermoregulatory Mechanisms

During wakefulness, central or peripheral thermal loads may increase or decrease sleep propensity, depending on whether sensory influences (EEG synchronizing or desynchronizing) and thermoregulatory somatic and autonomic activities (motility, posture, vasomotion, metabolic rate, circulatory and respiratory responses) are consistent or inconsistent with sleep processes. Thus, a moderate positive load better promotes sleep than a negative one, for the effects induced in somatic (e.g., decrease of muscle tonus) and autonomic (e.g., vasodilation due to decrease in sympathetic outflow to heat exchanger vasculature, sweating) activities are the same that occur in synchronized sleep. In this case, synchronized sleep may be considered as a synergic concomitant of thermoregulation in the adaptation to a positive thermal load. The contrary is true for negative thermal loads which, therefore, maintain or promote wakefulness. Nevertheless, since sleep may also occur under adverse thermal conditions, the sleep-promoting role of thermoregulatory structures is only facultative in wakefulness, and their functional importance is inversely related to the existing degree of sleep propensity.

Sleep-Regulating Role of Thermoregulatory Mechanisms

During synchronized sleep, thermoregulatory mechanisms temporarily become an essential part of the mechanisms controlling the further evolution of sleep (Parmeggiani, 1987). The changes in AH-PO temperature (exam-

ined below) are consistent indicators of such a regulation in normal conditions. Actually, they show that the occurrence of desynchronized sleep episodes depends upon a specific functional level of the AH-PO thermostat. However, the sleep-regulating function of AH-PO structures may be overridden by their thermoregulatory responses to heavy thermal loads. As a result, the duration of synchronized sleep is prolonged and arousal instead of desynchronized sleep may eventually occur. In such conditions, desynchronized sleep is depressed or suppressed also for long periods until the accumulation of an increasing desynchronized sleep debt produces sufficient pressure to overwhelm the AH-PO thermoregulatory drive (cf. Parmeggiani, 1977). In this case, brainstem effector mechanisms of desynchronized sleep escape from the normal AH-PO control.

In conclusion, thermal loads affect the ultradian wake-sleep cycle because they override the functional control of AH-PO structures on the cycle by increasing their thermoregulatory activity. This is easily conceivable, if the somatic and autonomic thermoregulatory effects of negative thermal loads are considered. Concerning positive thermal loads, a distinction between light and heavy is appropriate. The reason why this is the case is discussed in the next section.

Functional Significance of AH-PO Temperature Changes during Synchronized Sleep

The onset of synchronized sleep is characterized by a decrease in AH-PO temperature with respect to wakefulness. The amount of such a decrease at the end of the synchronized sleep episode is related to both ambient temperature and the occurrence of either desynchronized sleep or wakefulness (Parmeggiani et al., 1975). When synchronized sleep is followed by desynchronized sleep, the decrease in AH-PO temperature is greater at low (e.g., 0°C) than at room (e.g., 20°C) temperatures. However, when synchronized sleep is followed by wakefulness, the decrease is the same at both ambient temperatures.

These results demonstrate that the decrease in AH-PO temperature during synchronized sleep is dependent on sleep processes affecting the activity of the AH-PO thermostat so as to allow heat loss also at low ambient temperature. In the absence of a thermal load, that is, at neutral ambient temperature, sleep processes do not interact with thermoregulation, and the functional AH-PO adjustment producing heat loss easily occurs. In this case, the probability of desynchronized sleep occurrence depends only upon the ultradian need of desynchronized sleep and, in some species, upon the phase of the circadian rhythm of body temperature (see the following section). Under the influence of a thermal load, however, the probability of desynchronized sleep occurrence, that is, of a normal

evolution of the ultradian wake-sleep cycle, is determined not only by the previously mentioned factors but also by the effect of such a load on AH-PO thermoregulatory activity. In other words, the sleep-specific adjustment of AH-PO activity inducing heat loss may be supported or counteracted by central or peripheral thermal influences.

The range of AH-PO temperatures, which is compatible with the occurrence of desynchronized sleep, may be considered as a temperature "gate" whose width is affected by several factors such as ambient temperature, body size, thermal insulation, and age. However, the sleep-specific functional adjustment of the AH-PO thermostat is always the same. The different AH-PO temperature decreases during synchronized sleep in different environmental conditions indicate only whether such adjustment is unchallenged or opposed by environmental influences on the AH-PO thermostat.

Concerning the functional changes of AH-PO thermoregulatory structures in more detail, experimental data already show that the sleep-specific level of activity is the same occurring under the influence of light central or peripheral positive thermal loads. The only difference is that heat loss during sleep is the result of a downward regulation of body temperature induced centrally by sleep processes.

The previous results and considerations are consistent with studies showing that experimentally induced changes in AH-PO temperature affect the wake-sleep cycle. Light AH-PO cooling increases waking time (Sakaguchi et al., 1979). This is conceivable, for thermoregulatory activity such as vasoconstriction in the heat exchangers, increase in muscle tonus, shivering, and piloerection, entails autonomic adjustments that are opposite to those induced by synchronized sleep processes (see above). In contrast, light AH-PO warming promotes both synchronized and desynchronized sleep (Sakaguchi et al., 1979; von Euler and Soderberg, 1957; Roberts and Robinson, 1969; Roberts et al., 1969; Parmeggiani et al., 1974, 1980). AH-PO warming elicits changes in thermoregulatory effectors, like decrease in muscle tonus, vasodilation, and sweating, that are coincident with those brought about by sleep processes. So, the somatic and autonomic patterns of synchronized sleep are enhanced and not opposed by thermoregulatory mechanisms. Therefore, the effect of small central thermal loads is specific at neutral ambient temperature, since cooling promotes wakefulness and warming promotes sleep. The relationship between sleep and warming and between wakefulness and cooling is observed also with respect to the influence of peripheral thermal loads, although the stimulus nonspecific quality of comfort or stress may easily affect the wake-sleep cycle independently of thermoregulatory mechanisms. Evidently, AH-PO heavy positive and negative thermal loads exert only unspecific arousing influences, partially depending on the increase in sympathetic activity and the strong activation of circulatory and respiratory functions. Finally, it is important to mention that in the case of prolonged desynchronized sleep

deprivation elicited by negative thermal loads, synchronized sleep time increases with respect to total sleep time (cf. Parmeggiani, 1977). Therefore, synchronized sleep cannot be considered simply as a thermoregulatory response to positive thermal loads.

Control of the Ultradian Wake-Sleep Cycle in Relation to the Circadian Rhythm of Body Temperature

The previous considerations concerning AH-PO control of the ultradian wake-sleep cycle also apply to sleep occurrence in relation to the circadian oscillation of body (rectal) temperature in the absence of thermal loads (Parmeggiani, 1987).

As already mentioned, the duration of synchronized sleep varies inversely with the ease with which the AH-PO thermostat may be so adjusted as to induce the somatic and autonomic functional changes underlying heat loss. Thus, synchronized sleep may show different durations according to the phase of body temperature, a broad indicator of the activity level of the AH-PO thermostat under the influence of the circadian pacemaker. Synchronized sleep may be followed by either a desynchronized sleep or an arousal episode. On this basis, desynchronized sleep probability is theoretically greater around the minimum than around the maximum of the circadian oscillation of body temperature.

As a matter of fact, in humans desynchronized sleep occurs around the temperature minimum in conditions of both internal synchronization or desynchronization of the wake-sleep cycle (Czeisler et al., 1980; Zulley, 1980). In particular, the peak probability of this stage of sleep is reached just beyond the minimum and corresponds to the initial part of the rising slope of the average rectal temperature. This small inconsistency with respect to the animal model cannot be explained at present. At any rate, since the actual AH-PO temperature gate of desynchronized sleep cannot be determined in humans, it is worth considering the possibility that the average rectal temperature may give somewhat distorted information on the autonomic adjustments bringing about desynchronized sleep.

In conclusion, the AH-PO control mechanism of sleep exerts a restricting influence on the circadian occurrence of desynchronized sleep. The fact that desynchronized sleep in humans is associated with the minimum of body temperature is consistent with the concept discussed in this chapter. It is clear, however, that this conclusion deserves to be tested further with respect to other factors interacting at AH-PO level. The AH-PO control mechanism is particularly important in so far as the surrendering of homeostatic regulation in desynchronized sleep (cf. Parmeggiani, 1980a,b) is normally confined to actually harmless functional and/or environmental conditions.

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3

The Phasing of Sleep in Animals

NIGEL J. BALL

The tale of the hare and the tortoise typifies a fundamental enigma in the study of sleep: how can it be that animals, which we would like to think behave rationally (even if not consciously), can deny themselves clear rewards for the vaguely understood and often excessively delayed benefits of sleep? This is a particularly unpalatable proposition for animals that interrupt unfinished activities for sleep. It seems to us humans that animals should complete their day's activities, then find a secure sleeping location and sleep well, yet such a pattern is rare in nature. Less than 14% of mammalian genera, for example, can be considered strictly monophasic (Campbell and Tobler, 1984). To explain the variety of the phasing of sleep is to confront both the reasons for sleep and the principles behind the scheduling of behavior. In this chapter several different perspectives will be used to evaluate the biological significance of the phasing of sleep in animals.

Semantic and Methodological Perspectives: What Is Polyphasic Sleep?

Polyphasic sleep occurs when the dominant periodicity of the sleep-activity rhythm is less than 24 hr (e.g., Tobler, 1989). Thus to sleep for 8 hr and then to stay awake for 16 hr is to have a monophasic pattern, while 12 alternations of sleep and activity on a 2-hr period over the 24 hr is indisputably polyphasic. However, some animals alternate wake and sleep all night, but have no daytime sleep, while others have one consolidated sleep period, but occasionally also sleep later in the day. For this review, the

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definitions from the introductory chapter of this book have been slightly modified. *Strictly monophasic* is used for sleep patterns that either have only one uninterrupted episode of sleep each 24 hr, or have at least 12 consecutive hours per 24 hr in which there is no sleep. *Polyphasic* is used to imply a fairly even distribution of sleep throughout the 24 hr. *Strictly polyphasic* is used where the major sleep pattern is of a regular ultradian period. *Biphasic* is a particular type of polyphasic sleep in which two major sleep periods, with the shortest being more than a quarter of the duration of the longest, alternate with two periods of activity.

Related to the phasing of sleep is the phenomenon of the *sleep cycle*, (which would perhaps be better referred to as a "sleep rhythm" since the constancy of sleep patterns has been rarely demonstrated statistically; cf. Broom, 1979). Sleep cycles have been defined in two main ways: as the average rapid eye movement sleep (REMS)-episode interval, with or without a minimum threshold (Ursin et al., 1983; Shiromani, 1989); or as the duration of sleep between the onset of one REMS period and the next (Zepelin and Rechtschaffen, 1974; Zepelin, 1989). The first method allows comparison with other biological-rhythm studies that use activity levels, for example, to calculate ultradian rhythms, while the second method removes the variability due to wakefulness and perhaps provides a better measure of sleep. In this second method the amount of wakefulness allowed in a sleep episode is often arbitrary. Even those animals with monophasic sleep patterns may still have regular ultradian sleep cycles. The Basic Rest-Activity Cycle (BRAC) (Kleitman, 1963) is an ultradian rhythmicity believed to extend throughout the 24 hr and often, but not always (e.g., Okudaira et al., 1984), manifested by a variety of physiological and behavioral rhythms that may include sleep.

Tobler (1989) used the ratio of the amounts of sleep in the light and dark parts of the circadian cycle as an index of sleep phasing. Two related measures are used here. The "sleep-in-darkness index," (SIDI) is the percentage of the total sleep time (TST) occurring in the dark phase of an approximately 12:12 light-dark (LD) photoperiod, while the monophasic sleep index (MSI) is the percentage of sleep that can be considered monophasic. It is calculated as the percentage of difference between the SIDI and a strictly polyphasic sleep pattern (assumed to be SIDI = 50%). Thus an animal sleeping only at night, or only during the day, would have an MSI of 100%. The advantage of these indices is the avoidance of both difficult decisions about arousals and semantic problems of what constitutes an episode of sleep.

These definitions work well for mammals and birds, for which there are clearly defined sleep states (Zepelin, 1989; Amlaner and Ball, 1989). A comparison of the number of species with various sleep phasings as quantified by the sleep-in-darkness index (see Figure 3.1 and data in Table 3.1) shows that both mammals and birds contain what may be broadly

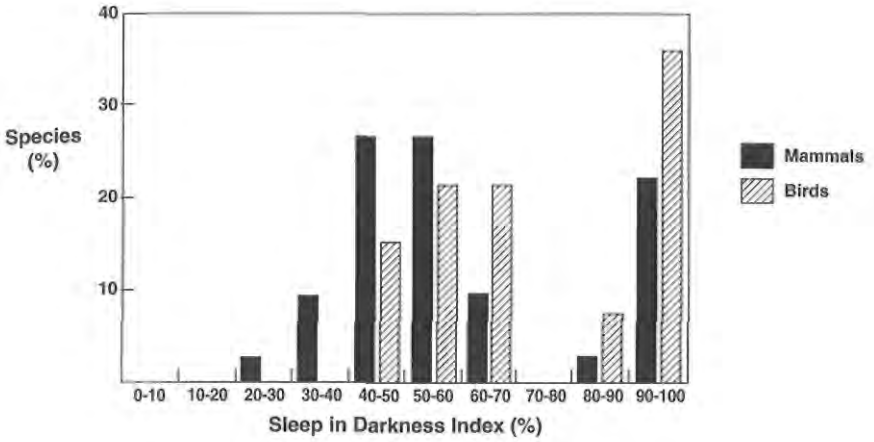

FIGURE 3.1. The variety of phasing in birds and mammals. A frequency histogram of the percentage of species of 14 bird studies and 23 mammal studies exhibiting various percentages of total sleep time in darkness. Data from Tables 3.1 and 3.2, and from Tobler (1989).

considered monophasic and polyphasic species. The phasing of sleep is not merely a mirror image of the phasing of activity. In spite of the prevalence of nocturnally active mammals, few of the species that have been studied electrophysiologically are both monophasic and diurnally sleeping, and few of the polyphasic species identified by this sleep-in-darkness index are diurnally active. Birds also tend to sleep more at night than mammals (median sleep-in-darkness indices are 61 and 53%, respectively). Finally, there is possibly a kind of "forbidden zone" of sleep at night: no birds or mammals in this sample showed 70-80% sleep in darkness. This gap implies a divergent selection pressure such that *either* evenly polyphasic *or* almost strictly monophasic sleep patterns are favored, and it has implications for human sleep which will be discussed below.

For other vertebrates, the division between wakefulness and sleep is much less clear, and the components of a sort of continuum of arousal states tend also to be distributed according to circadian patterns (Hartse, 1989). Thus, Karmanova (1982) distinguished several additional sleeplike states and states of immobility that dominate the inactivity of poikilothermic vertebrates. These states are characterized by immobility and raised thresholds of arousal, which are also the fundamental defining attributes of sleep. It is unfortunate that there is no consensus between East and West about these states or their relationship to REMS and to non-rapid eye movement sleep (NREMS), since our understanding of the phasing of sleep is considerably affected.

evolution: very active animals tend to have unusual methods of achieving sleep (cetaceans, Mukhametov et al., 1977; swifts, Cramp, 1985). Fish of the class Osteichthyes are similarly mostly polyphasic (Karmanova, 1982). However, if some sleeplike states are excluded, and only the most extreme sleep states quantified, then some fish species are monophasic (Tauber, 1974). It can therefore be concluded that the basic animal sleep-activity pattern is a largely obligatory phase of inactivity and sleep in which the most extreme sleep behaviors occur, alternating with a variably facultative polyphasic pattern of sleep in the remainder of the 24 hr.

3. Have There Been Evolutionary Tendencies toward Either Strictly Monophasic or Strictly Polyphasic Sleep Patterns? The strictly monophasic birds and mammals seem to be relatively more common among the more recently evolved taxonomic groups of their respective classes. The avian order Passeriformes, which consists of almost half of the known species of birds, and which was the last taxonomic order to appear (Brodkorb, 1971), has several families that are strictly monophasic (see Table 3.2). In mammals (see Table 3.1), the primates, rodents, and possibly some of the large grazing mammals have strictly monophasic members (Campbell and

TABLE 3.2. The phasing of sleep in birds

Order	Species	TST[a] (hr/24 hr)	SIDI[a]	MSI[a]	SCL[a] (min)	References[b]
Sphenisciformes	Emperor penguin	10.5	54	8	—	1
	Little blue penguin[c]	8.2	61	22	0.88	2
Anseriformes	Mallard	6.4	52	4	0.87	3
Galliformes	Domestic fowl	11.8	61	22	0.53	4
Columbiformes	Pigeon	12.4	68	36	—	5
	White-winged dove	12.4	82	64	0.12	6
Strigiformes	Tawny owl	16.0	50	0	—	7
		6.8	48	4	—	8
		11.4[e]	*49*[e]	*2*[e]		
	Snowy owl	7.7	43	14	—	8
Passeriformes[d]	Rook	7.6	ca. 95	90	11.6	9
	Jackdaw	6.7	ca. 95	90	7.5	10
	Magpie	7.1	100	100	5.5	11
	Chaffinch	7.3	100	100	—	12
	Starling	9.4	92	84	5.1	13

[a]For key to abbreviations, see Table 3.1.

[b]*Key to references:* 1, Buchet et al. (1986); 2, Stahel et al. (1984); 3, Zepelin et al. (1982); 4, Karmanova (1982); 5, Van Twyver and Allison (1972); Tobler and Borbély (1988); 6, Ayala-Guerrero and Vasconcelos-Dueñas (1988); 7, Susic and Kovacevic (1973); 8, Karmanova (1982); 9, Szymczak (1987b); 10, Szymczak (1986a); 11, Szymczak (1987a); 12, Tymicz et al. (1975); 13, Szymczak (1986b).

[c]Not used in analysis.

[d]Unlike most orders of birds, the passerines exhibit nocturnal, strictly monophasic sleep, with only small amounts of sleep during the light that are contiguous with the nocturnal episode.

[e]Means indicated by italic numbers.

Tobler, 1984), unlike the more ancient taxonomic groups. However, the difficulty of classifying drowsiness makes the exact interpretation of the phasing of sleep in many mammals difficult.

Strictly polyphasic patterns of sleep are more common. Herbivorous species, such as mallard (*Anas platyrhynchos,* Zepelin et al., 1982; D. F. Schmidt unpublished data), tend to have polyphasic sleep patterns (see Figure 3.2), as do many nocturnally active species. These species tend to have a rather equal distribution of sleep in the light and dark phases, even though the incidence of activity may be more skewed (e.g., owls, *Strix,* Šušić and Kovačcević, 1973; Karmanova, 1982; guinea pigs, *Cavia,* Pellet and Beraud, 1967). I cannot detect other evolutionary trends in the development of strictly polyphasic sleep.

4. Are NREMS and REMS in Phase? In humans, the propensity for NREMS is influenced strongly by the duration of prior wakefulness, while the end of a NREMS episode and the propensity for REMS are associated with the circadian phase of the endogenous temperature cycle (Dinges, 1989). For many species, particularly among adults, the incidence of NREMS peaks shortly after the end of the phase of activity and the incidence of REMS is highest toward the end of the phase of inactivity (e.g., Tobler and Borbély, 1988, but also see Szymczak, 1987a). Circadian aspects of polyphasic sleep are discussed below, but a priori one might expect different phasing for NREMS and REMS, even if REMS usually occurs in temporal conjunction with NREMS.

5. Are There Quantitative or Qualitative Differences in the Sleep Achieved by Animals with Monophasic or Polyphasic Sleep Patterns? For there to be an evolutionary significance to the phasing of sleep, there must be adaptive

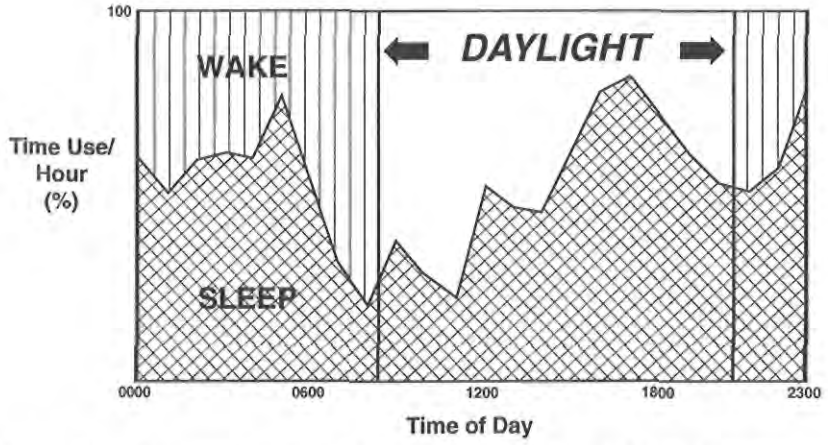

FIGURE 3.2. Polyphasic sleep in a herbivore, the mallard *(Anas platyrhynchos).* Mean percentage of 1-min spot checks of electrophysiological and simultaneous video data in which mallard were asleep ($N = 6$). (D. F. Schmidt, unpublished data.)

consequences that result from the adoption of either monophasic or polyphasic patterns. The function of sleep is still unclear, but some indication of its physiological consequences may be derived from examining the quantities and quality of sleep under different sleep phasing.

(a) Quantities of Sleep per 24 Hr. Mammals with monophasic sleep sleep less than those with polyphasic sleep [correlation of TSDT (hr/24 hr) with MSI (%): Spearman's $\rho = -0.513$, $N = 22$, $p = .01$; see Table 3.1]. This relationship was not substantiated in birds, probably because of the small taxonomically independent sample size (12). There are also insufficient data to statistically compare the quantities of individual sleep stages in either mammals or birds.

(b) Intensity of NREMS. In many mammalian species the intensity of NREMS, measured as the power in the delta band of the EEG, increases according to the length of prior wakefulness, and also declines during the inactive phase (Tobler, 1989). It is surprising, therefore, that monophasic species do not appear to have more intense NREMS than polyphasic species (Tobler, 1989), since, by definition, the duration of prior wakefulness is greater in the former. In birds, which lack the relationship between prior wakefulness and the delta power of the EEG (Tobler and Borbély, 1988), and in mammals, the polyphasic species have qualitatively lower arousal thresholds than monophasic species (e.g., Amlaner and Ball, 1983). It would be interesting to compare arousal thresholds throughout the 24 hr in both monophasic and polyphasic species, but, the EEG notwithstanding, it appears as if monophasic species may have more intense sleep than polyphasic species.

(c) Cycle Length. In birds, the cycle length of the monophasic passerines is greater than that of the polyphasic nonpasserines (Table 3.2, medians = 6.5 and 0.7 min, respectively), but this may result from the paucity of REMS in the passerines (Amlaner and Ball, 1989). In mammals there are few quantitative data, but again monophasic species tend to have longer sleep cycles [see Table 3.1; correlation of MSI (%) with sleep cycle length (min): $N = 16$, Spearman's $\rho = 0.55$, $p < .01$]. However, there is a clear correlation between body size and sleep cycle length in mammals (Zepelin, 1989), but both larger mammals and smaller birds tend to be monophasic and to have longer sleep cycles than their polyphasic equivalents. The lengthening of the sleep cycle in monophasic animals, if confirmed with analyses controlled for such variables as body weight, suggests an important outcome of monophasic sleep, namely, consolidation of sleep episodes. This also might be related to the smaller quantities of sleep in monophasic species: they might need less sleep because they are able to have more consolidated sleep.

(d) Experimental Changes: Creating Polyphasic Sleep. Sleep phasing can be manipulated experimentally. Lesioning the suprachiasmatic nuclei of small mammals produces a largely arrhythmic sleep pattern without altering the total amounts of NREM and REM sleep (Mistelberger et al., 1983). This

suggests that there is no direct relationship between phasing and amount of sleep in an individual animal.

Conclusions

The temporal patterning of sleep is complex, and depends on the types of inactive behavior included as sleep. Monophasic sleep appears to be a derived, perhaps specialized, evolutionary development, which has implications for adaptive advantages. An unknown divergent selection pressure may be involved in separating monophasic and polyphasic species. Although the consolidation of sleep that appears to be associated with a monophasic pattern may lead to reduced overall total sleep times, the converse does not occur when polyphasic sleep is experimentally induced within a species.

An Ethological Perspective: Scheduling Behavior

It is generally accepted that a reasonable generalization about the way that animals behave is that they will have been "designed" by natural selection to tend toward optimizing their behavior, that is, to maximize their total lifetime fitness. In reality, such optimality will be curtailed by genetic mixing, constraints and accidents of development, and the unpredictability of the environment. Ethologists have investigated the kinds of processes used to schedule behavior in an optimal fashion. Assuming that our model animal has a repertoire of behavioral activities, the choice of "ongoing" behavior seems to be influenced by the animal's internal state (e.g., how hungry it is) and by external cues (e.g., how large or tasty the food is). These two influences are easy to relate to: a late date the night before (producing an internal state of fatigue) or a boring lecture the morning after (an external cue discouraging wakefulness) both encourage daytime sleepiness, but to what extent do states and cues influence animal sleep patterns? Light intensity is a good example of an external stimulus that can mediate the phasing of sleep. Birds that normally only sleep at night may sleep in the day during solar eclipses (e.g., Kullenberg, 1955). The phasing of inactivity in the common vole *(Microtus arvalis)* changes as a function of the annual photoperiod (Hoogenboom et al., 1984). Warmth and/or sunlight also increase the propensity of sleep; birds that sunbathe, for example, will simultaneously sleep, even if they do not normally exhibit polyphasic sleep (N. J. Ball, personal observation). Sleep is also affected by internal need. Sleep deprivation can result in a more polyphasic pattern of sleep in the recovery phase (Lavie, 1989). Dark-eyed juncos *(Junco hyemalis)* studied under long daylength that induced nocturnal perch-hopping exhibited only

minimal daytime sleep (8.04%), even though their nighttime sleep was reduced by 75.3% (N. J. Ball, unpublished data).

Quantitative ethologists (e.g., McFarland and Houston, 1981) have used a model of animal behavior in which the animal is viewed as moving through a multidimensional state space. Each axis of this state space is a physiological deficit, and the origin represents the optimal condition for that animal in its usual environment. Any behavioral activity is then a vector in that state space, reducing deficits in some axes but inevitably and simultaneously increasing deficits along others. Thus when we sleep we might be decreasing our "sleep debt" (whatever that is), minimizing thermoregulatory deficits, or increasing our energetic and water deficits.

The importance of this approach to the study of polyphasic sleep is more apparent when the rules for changing behavior are considered. If the animal is to behave optimally, then it will be acting to minimize the total adaptive risks associated with its trajectory through this state space. There are two primary influences on these decisions. First, it is reasonable to assume that the adaptive risk to an animal of any position within the state space is the sum of the risks along each axis, and that the adaptive risk to an animal for any particular deficit is an increasing function of the magnitude of that deficit. This assumption implies that the costs associated with not sleeping for 16 hr and then sleeping for 8 hr are substantially greater than a regular, shorter, sleep-wake pattern, that is, there are substantial adaptive benefits to promptly minimizing deficits. Of course, always responding immediately to the largest deficit by choosing the behavior with the most appropriate vector would result in constantly switching between behaviors ("dithering"). However, a second primary influence on the decision to change behavior is the cost of changing, which can be defined as the costs incurred when changing to a new behavior before adaptive benefits result. Thus an animal might be sleepy enough to merit falling asleep, but might be in an insecure location. One of the costs of changing in this case would be the energy costs of moving to a secure sleep site, and these costs would delay or even postpone the decision to fall asleep.

How reasonable and applicable are these ethological ideas and assumptions? First, as discussed above, polyphasic sleepers tend to sleep more, not less, than monophasic species, which means either that the assumption that the cost of not sleeping is an increasing function of the duration of wakefulness is incorrect, or that the rate of accrual of benefits when sleeping is governed by an even more rapidly increasing function. Since the depth of sleep is probably greater in monophasic species (based on arousal thresholds), this second alternative may be acceptable. Second, the cost of changing behavior should be a relatively important influence on the phasing of sleep. Unfortunately, it is difficult to determine whether an animal has a monophasic pattern because of a high cost of changing *or* whether the high cost of changing has been adopted because the animal is monophasic. Starlings, *Sturnus vulgaris,* for example, often fly considerable distances to

roost communally, and are monophasic. However, under conditions of extreme cold they may sleep where they forage during the day (N. J. Ball, personal observation). Presumably, the cost of not finding a safe site in which to sleep is outweighed by the extreme necessity of recouping sleep lost by the cold, and by the higher cost of moving to a safe site in such cold weather.

It might be thought that animals subject to greater predation risk while asleep will have a higher cost of changing from wakefulness to sleep, partly from the additional predation risks while sleeping, but also from the travel costs of going to a more secure sleeping location. Such animals would be expected to have more monophasic sleep patterns (or to reduce the costs associated with sleep by lowering its intensity and developing polyphasic drowsiness). Even though many herbivores (which are more likely to be prey) are polyphasic, there is some support for this idea. The prediction holds for the sexually dimorphic wild mallard in which the brown-plumaged females, and even the brown males when in a brown "eclipse" plumage, spend more time asleep during the day than do the brightly colored males in breeding plumage (Lendrem, 1983). Fully tamed animals, which should be "released" from fears of predation, ought to be more polyphasic than wild stock or close wild relatives, and indeed wild passerines are monophasic even in the laboratory (e.g., Szymczak, 1987a), while tamed zebra finches *(Taenopygia)* are polyphasic (Schmidt et al., 1990).

A second major ethological perspective relates to the notion of "time-filling." For many animals, the 24-hr pattern of environmental light, temperature, humidity, and so forth, is so strong that a circadian patterning is almost inevitable. This means that animals must choose to behave not only in an optimal sequence but also at optimal times of day, and at optimal rates. Since daily requirements are not constant, we would expect animals to budget their time for the most strenuous of circumstances, leaving, under normal conditions, a buffer zone of time to be filled by a low-cost activity. It has been suggested that one of the functions of sleep is to act as this time-filling activity (Meddis, 1983), but there are other candidates, such as quiet wakefulness. Quiet wakefulness, otherwise known as "rest" or "loafing" or "idling," has been considered an "advanced" form of wakefulness resulting from cortical development (Kleitman, 1963). If an animal has both quiet wakefulness and sleep, then we might predict a polyphasic pattern to the former, even if we cannot find polyphasic sleep. By contrast, in those animals without quiet wakefulness, sleep may have the role of the time filler. Thus both shrews (Insectivora) and hummingbirds (Apodiformes) are small animals with high metabolic rates and substantial energetic demands. Shrews appear to use sleep as a time filler, while hummingbirds use quiet wakefulness. This difference probably arises because shrews, unlike hummingbirds, are able to feed throughout the 24 hr, but the latter can usefully visually scan their environment while inactive. However, this simple view of time-filling behavior does not explain why, for several animal species, the

percentage of time spent quiescent is less variable than the percentages of time spent in other, more active, behaviors (Herbers, 1981). Notwithstanding these exceptions, time-filling rarely seems to be an important function of sleep in the active phase of the day (although it might influence the intensity of sleep in the major period —see Horne, 1988).

In summary, polyphasic sleep would be expected to occur preferentially in species for which the benefits of sleep are high (or for which the costs of sleep deprivation are high) and/or in animals for which the cost of changing between wakefulness and sleep is low. The benefits of sleep are high as a result of either endogenous need or positive external conditions. These conditions, though generally applicable, do not provide an adequate explanation for the polyphasic-monophasic dichotomy seen in animals.

Mechanistic Perspectives: Wired for Sleep

Decisions

However precise and adaptable optimal decisions are, they are themselves costly, and animals, faced with repetitive problems over evolutionary time, would be expected to sacrifice some adaptability for low-cost, "hard-wired," or "preprogrammed" responses. Such preprogramming also allows different responses to be coordinated, which is especially important for sleep since the animal could then take advantage of the gross immobility inherent in almost all sleep by synchronizing the many different activities that are wholly or partially incompatible with body movement (Corner, 1985). This reasoning leads to the rather weak prediction that polyphasic sleep should be less frequent in animals with more active life-styles, since those animals will have less opportunity to distribute activities benefiting from immobility over the day. Hummingbirds may be an example of this: even though they are required to spend about 4 min in each of their 180 feeding cycles per day inactive in order to allow their crops to clear sufficiently for further feeding (Diamond et al., 1986), they are rarely observed to sleep (N. J. Ball, personal observation), perhaps because the metabolic inactivity of sleep would be incompatible with this rate-limiting step in digestion.

Sleep Mechanisms

Sleep serves, and is served by, different regions of the brain. Hence the structure of the brain is a likely influence on the phasing of sleep in animals. The phylogenetic development of the brain as it relates to sleep has been

reviewed by Broughton (1972). Invertebrates lack brain structures currently deemed necessary for the expression of REMS and NREMS. The most significant differences between the brains of the various vertebrate phyla, which otherwise appear to be organized on similar lines with similar levels of the major neurotransmitters in the major divisions of the brain (e.g., Aprison and Takahashi, 1964), relate to degrees of differentiation of the brainstem and to the origin and location of the associative centers. Amphibians lack obvious analogs of the brainstem structures that are responsible for mammalian sleep, while reptiles, culminating in the chelonians, show progressive development of these areas. Birds have more associative activity in the optic tecta and in cortical areas derived from the corpus striatum, whereas mammals have a larger neocortex. For birds and mammals these differences apparently influence sleep patterns only in small ways. Both groups have equivalent sleep stages and similar, but not identical, variability in sleep phasing (see Figure 3.1). Brain structure or activity does, however, influence sleep phasing. Long-term pontine cholinergic stimulation in rats decreases the inter-REMS episode duration while treatment with a muscarinic receptor blocker has the opposite effect. Both substances abolished circadian variability of the inter-REMS episode duration (Shiromani, 1989), indicating that dynamic action of the REMS control mechanism is required for the expression of ultradian variability. Avian sleep cycles are considerably shorter than mammalian cycles [medians: 3 and 20 min (Zepelin, 1989), respectively]. In a recent quantitative review of mammalian sleep patterns, Zepelin (1989) determined that the strongest morphological, metabolic, or ecological correlate of sleep cycle times was the logarithmic transformation of adult brain weight ($r = .88$). Even when the body weight and metabolic rate were statistically controlled, mammals with larger brains had longer sleep cycle times ($r = .64$). Zepelin noted that cycle length also increases during maturation, and concluded that a causal relationship with cholinergic activity of the brain might be responsible. In contrast, a study of seven species of felids indicates no relationship between sleep cycle time and brain weight (L. J. Squires, personal communication), and the same author notes that other intrafamilial comparisons (e.g., murids) also do not show this relationship. These exceptions might only be examples of a characteristic of correlative studies of brain weight — mathematical relationships often increase with the distance of the taxonomic relationship of the sample (Harvey and Krebs, 1990). This is partly a statistical artifact, but also indicates that the apparent relationship with brain weight is neither direct nor causal. However, Elgar et al. (1988) found that a positive relationship (partial $r = .663$, $N = 20$) between mammalian sleep cycle times and brain weight disappeared when humans were removed from the analysis, and in birds there is almost certainly a negative relationship between sleep cycle time and brain weight, although the appropriate brain weights are not available for statistical analysis. Thus the

studies to date are of low predictive value. Brain weight may be too gross or too indirect a measure of the underlying relationship, and future studies should perhaps examine hypotheses of causation more directly.

A Circadian Perspective: Rhythms from Within

Most animals, and even most cells, are capable of endogenous rhythmicity on time scales from a few seconds to years or more (Gwinner, 1975). However, these rhythms are most obvious, and probably most effective, when they are internally synchronized by an endogenous controller and synchronized to environmental rhythms by a Zeitgeber. Mammalian ultradian sleep-wake rhythms are synchronized by the hypothalamic suprachiasmatic nuclei, and bilateral lesioning of these nuclei produces arrhythmic sleep patterns (e.g., Mistelberger et al., 1983) in which ultradian structure of sleep bout length is also abolished (Edgar et al., 1989). The extensive neural connections between the retinas and the suprachiasmatic nuclei point to the predominance of light as a mammalian circadian Zeitgeber, although numerous other external stimuli have been experimentally demonstrated to be viable. Similar ultradian arrhthmicity can be produced in birds by destruction of the light-sensitive pineal gland (Gaston and Menaker, 1968), and the pineal is of major importance in the synchronization of most nonmammalian vertebrate circadian rhythms.

Endogenous rhythms are causally involved in the propensity for sleep, and hence are likely candidates for determining the phasing of sleep in animals. A large body of impressively quantitative research over the last 35 years has characterized the relationship between sleep (or activity) and endogenous rhythms in a variety of vertebrate species. Aschoff (1960) proposed that the daily level of activity increases with light intensity in diurnally active species, and decreases in nocturnally active species. The implications of this relationship on the phasing of sleep has not been fully evaluated. On the one hand, the decrease in the duration of the major phase of sleep should imply a homeostatically driven increase in polyphasic sleep, while conversely the hypothesized decrease in threshold of activity should make such polyphasic sleep less likely.

One problem with ascribing ultradian rhythms to endogenous mechanisms is the lack of obvious ultradian Zeitgebers, without which an ultradian rhythm may be of little benefit compared with other mechanisms for sequencing behavior. One exception is the antipredator advantage gained by rodents in synchronizing foraging behavior (Hoogenboom et al., 1984). It has been suggested that ultradian rhythms are based on harmonics of the circadian rhythm, but this has not been convincingly demonstrated.

A Summary: The Significance of Sleep Phasing in Animals

The fundamental sleep pattern is that of an obligatory phase of inactivity/sleep alternating with a variably facultative polyphasic pattern in the remainder of the 24 hr. The deepest sleep, or the most extreme sleep postures, may occur during the obligatory phase (Karmanova, 1982), and "poor" sleepers (Allison and Van Twyver, 1970) may be monophasic in the laboratory even though they sleep at other times of the day under more natural conditions. Some species of most phyla have developed a strictly monophasic pattern of sleep, while more species have a strictly polyphasic pattern in which the main sleep-wake rhythm is less than 24 hr.

It is clear from this short review that no one perspective provides an adequate explanation for the variety of phasing in sleep patterns. Evolutionarily, we may note that monophasic patterns appear to be derived and even specialized, but the adaptive benefit of such patterns is not striking. Indeed, with the exception of the order Passeriformes of the birds, which are discussed further below, the prevailing incidence of the fundamental sleep pattern argues strongly against universal benefits for variants. Ethologically, sleep patterns are hard to characterize as homeostatic or adaptive responses suitable for quantitative state-space analyses. Monophasic animals tend to sleep less, not more, than polyphasic species, and show no particular ecological characteristics: they do not tend to have particular diets or to be an extreme size. Some —for example, the largest grazing mammals —are poor sleepers, and these may be monophasic almost by default: they sleep so little in the laboratory that they have little opportunity to display polyphasic patterning. Mechanistically, the monophasic species are closely related to similarly constructed species that are polyphasic. It might be argued that some monophasic species have disproportionately large associative areas of the brain since the cerebral hemispheres are relatively large in humans compared with many other mammals, and the optic tecta are large in passerines compared to other birds (Pearson, 1972). However, other monophasic species do not conform to this pattern, and there are polyphasic and facultatively monophasic mammals with large associative areas. The circadian perspective fails to produce a solid rationale for most ultradian rhythms. Thus it appears that none of these perspectives provides a complete answer to the question of the phasing of sleep in animals, but all contribute to the explanation of specific cases. So why is there a dichotomy in phasing, and why did monophasic patterns develop and survive, albeit in a limited number of species?

We can note that there have not been persistent evolutionary tendencies toward strictly polyphasic sleep, especially of the deeper and more intense forms. Many cases of strictly polyphasic sleep appear to have external

causes (such as nocturnal activity, or constraints resulting from diet), and thus we may conclude that the benefits of strictly polyphasic sleep (e.g., minimizing overall costs) must usually be outweighed by the costs (perhaps the costs of changing from wakefulness to sleep, or decreased adaptive fitness resulting from raised arousal thresholds). In addition, the evolutionary tendencies toward more intense forms of sleep, and away from the torporlike or unspecific immobility of nonhomeothermic vertebrates, implies that sleep patterns, and sleep phasing, have been subject to evolutionary pressures, and can reasonably be expected to have been optimized. Since whole orders (i.e., the passerines) have largely adopted monophasic sleep, we can assume that the adaptive advantages of that pattern transcend details of day-to-day behavioral, ecological, or social considerations: the passerines are morphologically similar, but otherwise rather diverse. Monophasic species also have polyphasic young, and these facts together imply that a morphological advance (perhaps the large associative areas common to these groups) released, but did not dictate, the monophasic pattern in adults of this group. Perhaps an increase in "sleep-debt capacity" is responsible. The ability to function fully awake for most of the day without loss of performance due to sleepiness might provide opportunities that are not available to polyphasic species and also might facilitate monophasic sleep in these upwardly mobile groups: the passerines and humans have, after all, attained almost global numerical dominance for their ecological types.

A review of the polyphasic variants of human sleep patterns is beyond the scope of this chapter, but the apparent success of monophasic species, the extreme reluctance of passerines to sleep during the day, and the divergent selection pressure suggested by Figure 3.1 might caution against the viewpoint that polyphasic sleep would necessarily be advantageous to humans.

In conclusion, there are apparent barriers against the widespread adoption of strictly polyphasic or strictly monophasic sleep patterns, yet monophasic patterns when used are a successful adaptive strategy. Future research must determine what long-term costs such monophasic species pay, and exactly what facilitates this unusual ability.

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4

Sleep-Wake Rhythms and Sleep Structure in the First Year of Life

PIERO SALZARULO AND IGINO FAGIOLI

On the long path from the neonatal polyphasic sleep-wake rhythm to the adult monophasic sleep rhythm, the first year of life includes many important steps.

Early reports (Parmelee et al., 1964; Kleitman and Engelmann, 1953; see also Webb, this volume, Chapter 5) clearly stated a progressive diminution of sleep amount during the day, leading to two sleep episodes ("naps"), and an increase of the sleep amount during the night, together with a lengthening of the longest sleep period, a phenomenon emphasized more recently by Coons and Guilleminault (1984) as "sleep consolidation."

These essential phenomena could imply that the *number* of sleep episodes decreases with age, a fact that can be grasped by the inspection of the Kleitman (1963) and Parmelee et al. (1964) figures.

To look more closely at this problem and to investigate the related sleep structure, we reanalyzed recordings of 10 infants from a population previously studied (Fagioli and Salzarulo, 1982b). The infants were aged between 5 and 50 wk. Each infant was recorded polygraphically for a whole 24-hr period. For this study we retained three behavioral states, defined by combining behavioral and electrophysiological data:

Quiet sleep (QS): eyes closed, no eye movements, diminished body movements, EEG with slow waves and spindles (or "trace alternant")

Paradoxical sleep (PS): eyes closed (or alternatively half-open and closed), rapid eye movements, body or limb movements, low voltage EEG, chin muscular atonia, irregular respiration

Waking: eyes open, eye movements, irregular respiration, and additional body movements.

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The minimum duration of each state was 2 min, shorter changes being included in the preceding state.

Sleep Episodes: Number and Duration

In the present analysis, the sleep episodes that included wake epochs shorter than 15 min were considered as uninterrupted sleep episodes; the same procedure was applied to identify uninterrupted wake episodes. By applying these criteria a negative correlation was found between the number of sleep episodes and age (see Figure 4.1A). By splitting the 24-hr period into two halves (daytime: 0800–2000; nighttime: 2000–0800), we found a significant correlation only for the nighttime (see Figure 4.1B) and not for the daytime (see Figure 4.1C).

On the other hand, what is changing between younger and older infants is the distribution of sleep episode duration between night and day. In fact, the number of sleep episodes whose duration is either longer or shorter than the median [see Table 4.1(A)]—in other words, long and short episodes—in young infants (5–14 wk old) is similar during the daytime and during the nighttime ($\chi^2 = 1.004$, df = 1, n.s.). On the contrary, in older infants (19–50 wk old) long episodes are more frequent during the nighttime and short episodes during the daytime ($\chi^2 = 13.604$, df = 1, $p < .001$).

Moreover, nocturnal sleep episodes lengthen with age and represent an increasing proportion of total sleep time (see Figure 4.2). This trend is in agreement with the data mentioned above (Coons and Guilleminault, 1984) concerning the consolidation of nocturnal sleep.

A complementary aspect of this consolidation pattern is the alternation of short episodes of wakefulness and sleep, which indicates the difficulty of organizing "stable" sleep episodes. To obtain an index of instability of sleep and wakefulness, we investigated the sequence of two or more consecutive epochs of sleep and waking shorter than 15 min, which we defined as a "fragmented sleep-wake pattern." The total duration of fragmented sleep-wake pattern showed a significant decrease from young infants to older ones [$F(1,8) = 11.224$, $p < .025$], whereas the difference between nighttime and daytime [$F(1,8) = 3.240$, n.s.], and the interaction between factors [$F(1,8) = 1.479$, n.s.] failed to reach statistical significance [see Table 4.1(B)].

Sleep Architecture

In the first year of life, sleep undergoes important maturational steps, concerning both individual physiological activities (EEG, motility, ECG, etc.), and sleep states. The latter are particularly important, because they are involved in the analysis of sleep structure.

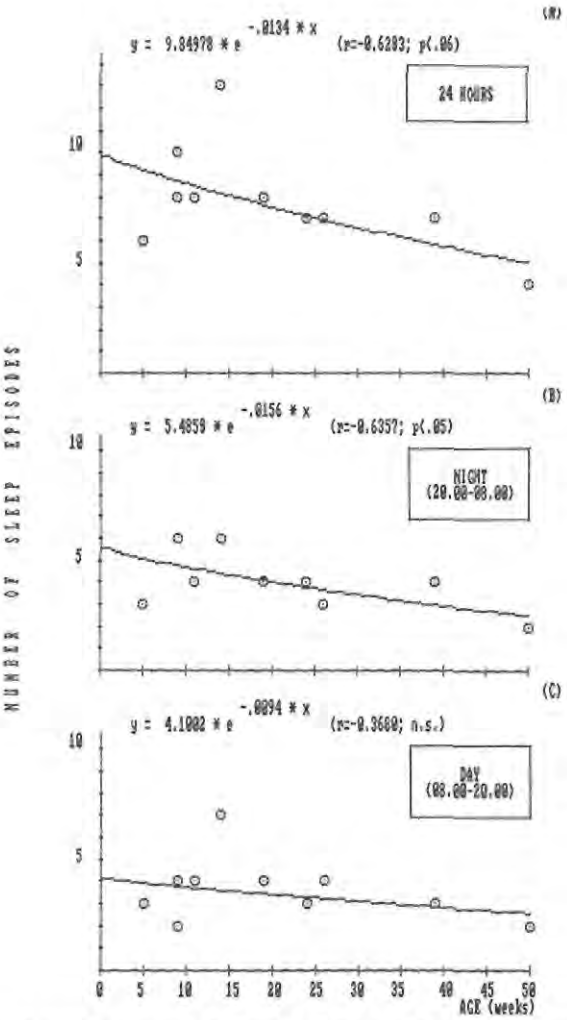

FIGURE 4.1. Number of sleep episodes as a function of age: (A) whole 24-hr period; (B) at night (2000–0800); (C) during the day (0800–2000).

Increases in QS have been described by several authors in daytime recordings (Dittrichova, 1966; Emde and Walker, 1976), in nighttime recordings (Navelet et al., 1982; Anders and Keener, 1985; Hoppenbrouwers et al., 1988), and in 24-hr recordings (Fagioli and Salzarulo, 1982b; Coons and Guilleminault, 1982). Data about PS are more controversial. While in the past it has been claimed that PS diminishes with age (Roffwarg et al., 1966), more recent nocturnal sleep recordings showed no consistent diminution with age (Fagioli and Salzarulo, 1982b; Navelet et al., 1982; Hoppenbrouwers et al., 1988).

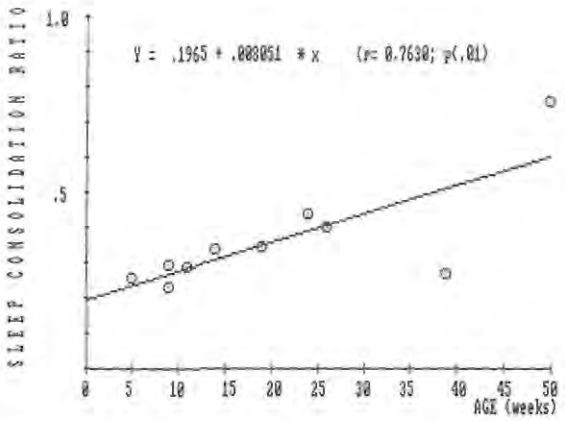

FIGURE 4.2. Sleep consolidation ratio (duration of the longest sustained sleep/duration of total sleep time) as a function of age.

In fact, QS is present in nearly all diurnal (90%) and nocturnal (92%) sleep episodes in young infants, and in all sleep episodes in older infants (i.e., between 19 and 50 wk). There are no differences between diurnal (90%) and nocturnal (80%) PS in young infants, while there is a strong difference in the older infants, in whom PS is present in 94.12% of nocturnal and in *only* 50% of diurnal sleep episodes. This suggests that after age 5 mo 50% of sleep episodes consist only of QS, a finding very similar to that observed in adult naps taken in the afternoon (Salzarulo, 1971). Other adult studies showed differences according to the time of the day (see Broughton, 1989).

As a consequence of what is noted above for QS and PS separately, many daytime sleep episodes of older infants have no QS-PS cycles, while most nocturnal sleep episodes do have QS-PS cycles [see Table 4.1(C)]. A QS-PS cycle (a phase of QS plus a phase of PS) was defined as the period starting from the beginning of QS to that of the next QS (or the spontaneous awakening) (Fagioli and Salzarulo, 1982a). This shows that diurnal sleep episodes are poorly organized in contrast with nocturnal sleep. In fact, diurnal sleep episodes are very often short (<28 min), and are spontaneously terminated before PS takes place. The spontaneous awakening from diurnal sleep episodes in QS is opposite to the general trend which consists of PS awakenings (Schulz et al., 1985). This mechanism could explain the findings mentioned above concerning the shorter duration of diurnal sleep episodes [see Table 4.1(A)], and the diminished ability to sustain a sleep episode when sleep begins in the daytime, particularly evident in older infants [see Table 4.1(D)].

Slow wave sleep (SWS) became in recent years an important marker for describing the "sleep process" (Borbély, 1982). In the adult, differences in

TABLE 4.1. Characteristics of *nocturnal* (2000–0800) and *diurnal* (0800–2000) sleep *in young* (5–14 wk) and *older* (19–50 wk) infants[a]

	(A) Number of sleep episodes		(B)	(C)	(D)
	shorter than median	longer than median	Duration of fragmented sleep-wake pattern	Sleep episodes without QS-PS cycles	Duration of sleep episodes by sleep onset time
			Young Infants		
Night	10	15	52.80 ± 46.29	44.00	63.0 (27.5–139.0)
Day	11	9	15.60 ± 10.01	55.00	48.0 (27.5–149.0)
			Older Infants		
Night	4	13	7.20 ± 9.96	23.53	118.0 (64.0–212.0)
Day	14	2	0.00 ± 0.00	68.75	32.0 (27.8–38.0)
			All Infants		
Night	14	28	30.00 ± 39.75	33.33	77.5 (37.0–169.0)
Day	25	11	7.80 ± 0.59	61.11	36.5 (27.6–72.5)
24 Hr	39	39	37.80 ± 42.21	46.15	59.0 (28.2–136.0)

[a] *Explanation of column headings:* (A) Number of sleep episodes whose duration is either *longer or shorter than the median (median duration = 59 min).* (B) *Total amount [mean ± SD* (min)] of "fragmented sleep-wake pattern" (i.e., the sequence of two or more consecutive epochs of sleep and waking shorter than 15 min). (C) Percentages of sleep episodes without QS-PS cycles. (D) Duration of sleep episodes according to the time of sleep onset (median and interquartile range).

SWS amount between diurnal and nocturnal sleep have been found (Knowles et al., 1986), and have been related either to the length of prior waking (Borbély, 1982) or to a circadian influence (Webb, 1986). In our studies (Schulz et al., 1989), infants begin to show SWS after age 4–5 mo. In infants aged between 19 and 50 wk, SWS could be found in many sleep episodes, more often at night (89.4% of episodes) than during the day (68.7%). A more detailed analysis of the distribution of SWS amount in successive 4-hr epochs showed a peak of maximum production between 2400 and 0400, and a minimum between 1200 and 1600 [$F(5,20) = 2.922$, $p < .05$] (see Figure 4.3). This distribution is different from that described in the adult (see Broughton, 1989).

On the other hand, in the longest nocturnal sustained sleep episode the SWS percentage is greater in the first cycle as compared to the next ones (Bes et al., 1991). However, the nocturnal decrease is not as robust as that observed in the adult. Since we are still in the presence of a polyphasic sleep rhythm that leads to shorter waking time before the nocturnal longest sustained sleep, we may speculate that the weak decrease of SWS during the night is accounted for by the short prior waking time. However, we cannot exclude other developmental factors: an investigation with infants of the same age and different prior waking times could clarify this problem.

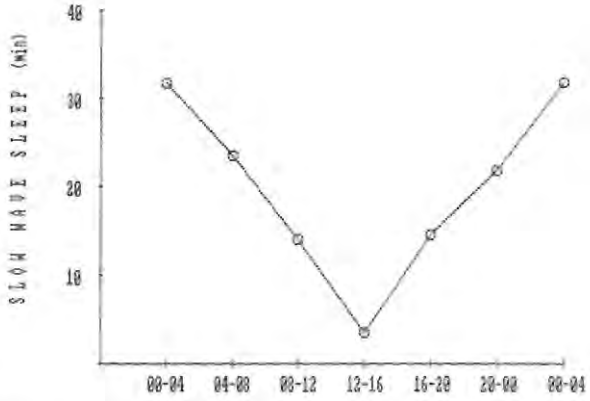

FIGURE 4.3. Mean amount of slow wave sleep (min) as a function of time of day (grouped in 4-hr epochs) in infants older than 19 wk.

Conclusions

During the first year of life there is a progressive tendency to diminish the number of sleep episodes, mainly during the night: after age 19 wk nocturnal sleep becomes more consolidated with longer sleep episodes. Its internal organization is in some way similar to that of younger infants, and also anticipates that of the adult. In fact, the duration of QS-PS cycles is around 50 min, while the recurrence time of SWS is about 100 min, leading to a 1:2 coupling between SWS and PS (Bes et al., 1991); this is a demonstration of the existence of a relative independence between PS cycle and SWS cycle, previously indicated for the adult by Lubin et al. (1973). At the same time, SWS proportion, which is high in the first sleep cycle, does not decrease progressively during the night. It takes several years before the adult sleep organization is reached (Bes et al., 1991).

This type of night sleep is followed by daytime sleep episodes that are often short, while still relatively numerous. Infants age 19–50 wk still have a propensity to fall asleep during the day, but also have greater difficulty in maintaining sleep than younger infants.

Some aspects of diurnal sleep anticipate those of adult naps (e.g., the proportion of sleep episodes including PS); in fact, the decrease of PS amount in the first year of life takes place during the daytime (Fagioli and Salzarulo, 1982b). The circadian distribution of SWS is clearly different between infants and adults. In addition to the major nocturnal peak in SWS, adults show a secondary peak of SWS in the midafternoon (for a review, see Broughton, 1989). This midafternoon secondary peak of SWS was not found in the infant population we investigated. On the contrary,

the midafternoon interval possibly represents the nadir of the SWS circadian rhythm in infants.

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5

Developmental Aspects and a Behavioral Model of Human Sleep

WILSE B. WEBB

This chapter is divided into three parts. First, a model or schema of sleep is outlined. Second, the ontogenetic development of sleep is presented within this schema. Third, conclusions are drawn about the effects of polyphasic variations in sleep patterns in human adults.

A Model of Sleep

I have elsewhere outlined a "behavioral model" of sleep (Webb, 1988). This is a variation on the Daan-Borbely model of sleep (Daan et al., 1984). It represents an attempt to develop a predictive model of the dimensions of sleep which are subdivided into sleep patterns, sleep structure, and subjective responses. In the present chapter the primary sleep variable to be considered is sleep patterning. Sleep patterns are defined as measures of total sleep amounts, episode lengths, and their placement within the 24 hr. As an example, a 2-yr-old human may have 12 hr of total sleep time with a circadian placement of a night episode of 10 hr and two daytime episodes of 1 hr in the morning and 1 hr in the afternoon.

The model states that sleep patterns are predictable from (i.e., a function of) three primary variables: sleep demand, circadian timing, and behavioral facilitation or inhibition. The relationship among these variables may be stated by the formula:

$$R\ (\text{sleep}) = f\ (\text{sleep demand} \times \text{circadian timing} \pm \text{facilitation and/or inhibition})$$

Demand, in this formula, is defined as a positively accelerated function of prior wakefulness and a negatively accelerated function of sleep occurrence.

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Circadian timing is defined by the time within 24 hr of the sleep episode. Facilitators and inhibitors are those behaviors that have been shown to increase or decrease the probability of sleep. For example, being erect, or anxious, or in an environment which, voluntarily or involuntarily, demands continued responding inhibits sleep. In contrast, being supine, relaxed, or in a low-demand environment facilitates sleep.

Operationally, this formula states that, if both sleep-wake timing and facilitation-inhibition factors are held constant over a series of sessions, sleep will be a function of sleep demand (prior wakefulness/sleep episode length). Or, if sleep demand and circadian timing are held constant, then sleep will be a function of behavioral determinants that inhibit or facilitate sleep. If sleep demand and facilitation-inhibition factors are held constant, while circadian timing is varied, sleep will be a function of the timing of the sleep episodes.

The parameters of these primary variables are affected by four modulators: species, neurophysiological conditions, individual differences, and developmental level (age). It is apparent, for example, that the sleep demand parameter in some species may be some 4 hr per 24, while that for other species may be 16 hr per 24. The circadian parameter in some species may be nocturnal and highly polyphasic, and for others it may be diurnal and less polyphasic. Moreover, each species has different inhibitory and facilitative factors. Neurophysiological modulators refer to those states that have been shown to be distinct modulators of the primary parameters; examples of their modulator effects include drugs and such pathological conditions as narcolepsy insofar as they modify the sleep demand or circadian parameters.

Here, however, I will not discuss species differences and neurophysiological modulation. Consideration will be limited to sleep among humans with "normal and healthy" neurophysiological status. This chapter focuses on the modulating effect of developmental status. Due attention will be paid to the modulating effects of individual differences. A detailed review of the modulating effects of development has been presented elsewhere (Webb, 1989). The following is a highlighted summary of this earlier report.

Sleep Demand and Age

If no systematic restraints are placed on sleep, it can be assumed that the total amount of sleep obtained in a 24-hr period reflects the demand for sleep. Put simply, the amount of sleep obtained is the amount required to fulfill the sleep demand.

The changes in sleep amounts in infancy have been well documented by the studies of Parmelee (Parmelee et al., 1961, 1964) and Kleitman and Engelmann (1953). Beginning with an average sleep total of about 16 hr in

the first few days of life, sleep rapidly declines to about 14 hr by the end of the first month, with a slower decline to about 12 hr by the sixth month (see also Salzarulo and Fagioli, this volume).

An older study (Foster et al., 1928) and a more recent study (Koch et al., 1984) describe the further decelerated sleep decline across the first 5 yr. There is a slow linear decline of about 30 min per year from age 1 through 5. Both studies reported somewhat higher sleep amounts than Parmelee et al. (1961, 1964) Kleitman and Engelmann (1953), with a terminal level of 11- to 12-hr averages at age 5. These figures are generally substantiated by Weissbluth (1987).

Beyond age 5 it is apparent that total sleep time becomes a less certain measure of sleep need, for sleep amounts begin to be influenced by such external factors as school and parental schedules. This is neatly illustrated by the study of Anders et al. (1978), who surveyed the sleep of adolescents between ages 10 and 13 and obtained data concerning sleep on weekdays versus sleep on weekends.

For children age 10, sleep on school days and weekends was essentially the same: 590 min or about 10 hr. This finding indicates a slow decline from age 5 (with about 12 hr) to age 10 of about 25 min per year. There is a further decline on weekdays over the next 4 years to 520 min. However, on weekends, the sleep was 40 min longer (560 min). The author noted that the sleep need (demand) "may not be changing . . . but that the school and social pressures on older children may decrease the amount of time available for sleep" (p. 62) during the week.

This confounding of total sleep as a sleep demand measure as influenced by social demands is apparent in adult populations, again exhibited by differences between weekday and weekend sleep (cf. Webb, 1985). However, it is not unreasonable to assume from extensive surveys, that sleep demand further declines from adolescence levels of about 10 hr to adult levels of about 8 hr.

Extensive questionnaire data from the American Cancer Society concerning more than a million men and women has been reported by Kripke et al. (1979). These data are self-reports on sleep amounts from people aged 30-90. The women reported 7.9 hr of sleep with essentially no change until age 70. The men reported a slightly lower level at age 30 (7.8 hr), with a linear increase to 8.0 by age 60. Both genders then show an overall increase with increasing age.

A further analysis of the Kripke et al. (1979) data reveals a striking aspect of the sleep of older persons. There is an increase in both higher and lower sleep amounts, indicating a differential development of higher and lower sleep demand levels in this age group. This is displayed in Figure 5.1. The figure plots the percentages of sleep of more than 9 hr per night and less than 6 hr per night across the age groupings. The difference in scale values for the longer and shorter groups should be noted. While there are increases in both these groups, it is apparent that the longer sleep tendencies are

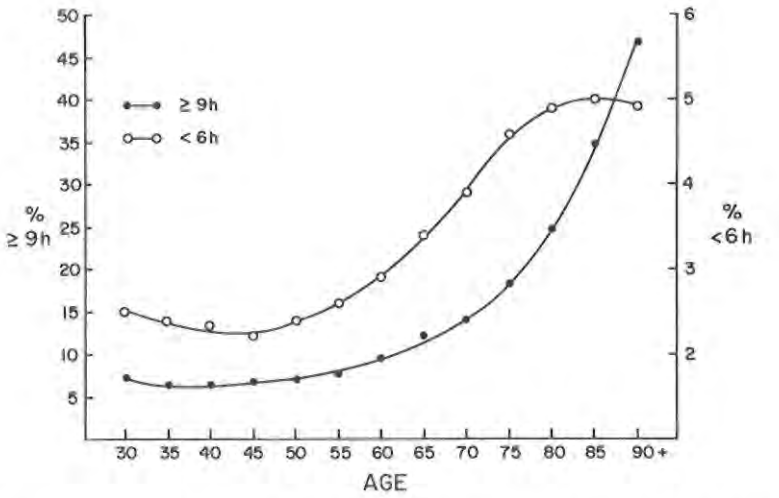

FIGURE 5.1. Percentage of population (by age groups) sleeping more than 9 hr and less than 6 hr per night. Reprinted with permission of Raven Press, from Webb W, Dinges D (1989): Cultural perspectives on napping and the siesta. In: *Sleep and Alertness*, Dinges D, Broughton R, eds. New York: Raven Press.

greater. But there is a decreased number of individuals in the middle range of sleep and an increase in the extremities.

This figure emphasizes a crucial variable in the discussion of sleep parameters: individual differences. The figure shows that between ages 30 and 50, about 2.5% of these persons slept less than 6 hr per night while 7.5% slept more than 9 hr per night. This is in accord with the consistent finding of surveys that report a standard deviation of about 1 hr. Thus individual sleep demands vary from less than 5 hr to more than 10 hr in the general population. This standard deviation is higher in both infant and elderly populations.

The Circadian Timing of Sleep

At birth, sleep is polyphasic with an average of about six sleep episodes distributed equally across the 24 hr. However, within the first week, there is an evident tendency for sleep to consolidate into a long sleep period (Parmelee, 1974). Parmelee et al. (1964) and Kleitman and Engelmann (1953) document an essentially linear increase in the amount of "nighttime sleep," which reaches an asymptote of about 10 hr by the twelfth week. Parmelee (1974) states that about 66% of the infants have established a clear pattern of nocturnal sleep by the fifth week, and 98% have done so by the twelfth week. Anders and Keener (1985) have carefully documented this

consolidation. They report a rise of nighttime sleep from 8.3 hr in the first 2 wk to 10 hr in week 24, and sleep remains at this level through the first year. The longest sleep period rises steadily from 233 to 430 min by week 20, and thereafter remains at this level.

The most remarkable change occurs, however, in daytime sleep. From the first week this begins to show a linear decline in total amount from about 7.5 hr to about 3.5 hr by the sixth month (Parmelee et al., 1964; Kleitman and Engelmann, 1953). Furthermore, these sleep periods are typically distributed into two sleep periods, one in the morning and one in the afternoon (Gesell and Ilg, 1937; Parmelee, 1974; Meier-Koll et al., 1978; see also Salzarulo and Fagioli, this volume).

From the first year through the fifth year, the changing circadian tendencies are quite clear. There is a stable amount of nighttime sleep combined with a continued decline in daytime sleep. This decline in daytime sleep is primarily a function of the elimination of nap periods. The pattern for 2-year-olds, as reported by Moore and Ucko (1957), indicates that 25% of the children studied took two naps, 66% took a single nap, and 8% napped irregularly or not at all. The deletion of the nap is well documented from early nursery school studies. The median percentages of children ages 2, 3, and 4 who did not take afternoon naps were 4, 17, and 40%, respectively. Reynolds and Mallay (1933) reported that 50% of the 2-year-old group took naps every day and all took naps at least 3 days a week. In contrast, none of the 4-year-olds took a nap every day and only 50% napped once or twice per week.

The data on older children is sparse. Simonds and Parraga (1982) cite mothers' reports of their own children that indicate that 17% of children ages 5–8, 9% of children ages 9–11, 9% of children ages 12–14, and 23% of children ages 15–18 took naps.

There is interesting experimental evidence to suggest a sharp decline in nap tendencies in middle childhood. Carskadon et al. (1983), using the Multiple Sleep Latency Test, reported a continuous rise in sleep latency with frequent "no nap" values from the prepubertal to pubertal maturity, with a subsequent decline of these scores (increased nap tendencies) thereafter.

With maturity, the presence of napping seems primarily to be a function of behavioral control. Naps are present in "seista cultures" and generally absent in northern climates and industrial settings (Webb and Dinges, 1989). They are also frequent in environments, such as college campuses, where schedules are not rigid.

With advancing age there is an evident increase in naps. In a stratified sample with sleep-log reports, Tune (1969) reported a linear rise in naps from ages 20 to 90. Webb and Swinburne (1971) observed older persons in a nursing home, aged 66–96, and found that only 1 of 19 persons went without naps over a 4-day period; 4 averaged three or more naps daily.

While naps do emerge in older populations, it is clear that their presence is limited. In the Tune study there was only an average of about one nap

each 4 days in his oldest group. Spiegel (1981) studied a group of retirees with an age range of 53-70 and found that 34% never napped, and 18% napped only occasionally.

Behavioral Facilitation and Inhibition

There is much asystematic evidence for the role of cultural and individual effects on the sleep response. Akerstedt (1988) has presented an extensive review of the role of shift work associated with subjective, behavioral, and physiological sleepiness. Webb and Dinges (1989) have reviewed cultural effects on naps; effects of geography and industrialization are also apparent. Numerous studies have documented the differences between weekend and weekday sleep tendencies and the effect of college schedules.

The striking effect of removal of time schedules which results in circadian shifting of sleep has been demonstrated in infants (Kleitman and Engelmann, 1953) and in time-free studies (cf. Webb and Agnew, 1974). Perhaps the most remarkable evidence of the effect of behavioral control (or its lack) is found in the studies by Campbell (1984). When individuals are confined to bed without behavioral control, sleep amounts increase by 50% and sleep episodes become fragmented and average less than 4 hr in length.

In short, it is apparent that, within limits, the sleep response is partially determined by the behavioral determinants of the individual, and that these, in turn, are modified by the developmental process. The infant sleeps in a behaviorally facilitative environment. As the child enters the social and school environment, the effects of these environments are manifested in weekday versus weekend differences in sleep. As the individual emerges into the adult environment, such factors as college schedules, shift-work conditions, and cultural patterns display their influences on the sleep response. Finally, with aging, such factors as physical restrictions, physiological impositions, and occupational changes emerge as determining factors.

Summary and Interpretations

Indexed by age, there are systematic changes in the overall amount and the patterning of sleep. Sleep demand levels change from an average of about 16 hr at birth to about half this level in the adult. In circadian timing, there is a rapid development from an initial acircadian polyphasic timing to a long sleep episode with two short episodes. These daytime episodes are eliminated by about age 5, and the long nighttime episode gradually shortens to a stable level in young adults. The young adult has intermittent daytime episodes that are heavily determined by cultural factors and

sleep-wake scheduling. Daytime episodes emerge more strongly in older persons but continue to be limited in their presence.

Three speculative interpretations can be placed on these data. It may be argued that these changes are primarily a function of the changes in sleep demand levels. Daytime sleep episodes are necessitated by the inability to fulfill the initial high sleep demand by means of a single major episode. As the demand level decreases and the major episode becomes consolidated, daytime episodes are eliminated. The reemergence of daytime sleep episodes in older subjects may be interpreted as a "replacement" for the deteriorating major episode. Alternatively, it could be argued that these patterns reflect an increasing maturity of a biphasic sleep-wake circadian system. The maturation of the waking circadian period permits the elimination of the daytime sleep episodes. In this interpretation, the emergence of the naps in older persons (and the increasingly fragmented major episode) are taken to be evidence of the destabilizing of the circadian system.

A third alternative would argue for developmental changes in both sleep demand levels and circadian tendencies. In this interpretation, the adult pattern would have a primary biphasic sleep-waking timing of about a 8/16 hr relationship, with a "residual" daytime tendency. This interpretation most effectively accounts for the intermittent presence of naps, their timing, and the reemergence of naps in older persons.

Comments on Polyphasic and Ultrashort Sleep Regimens

The orderly, species-specific, unfolding pattern of ontogenetic development in humans strongly suggests an evolved inherent system. Such systems are fixed systems, of ancient natural "wisdom," which have evolved to effectively relate species to their particular ecological niches. Ethologists such as Tinbergen (1969) and, more recently, sociobiologists have argued that attempts to "reschedule" or modify such systems face severe limitations or risk "misfirings" of these systems. The development of alternative "polyphasic or ultrashort" schedules would appear to be attempts to alter such a fixed system.

In the data presented in this chapter and, within the schema used, young-adult human sleep can be generally characterized as having a circadian pattern of a major sleep period and a major waking period and a sleep demand level of about 8 hr. For a detailed presentation of this patterning and the interaction of the circadian and demand variables, the reader is referred to Daan et al. (1984). Within such a schema, attempts to reschedule sleep-waking into alternative periods would result in a disruption of the "natural sleep" processes.

There is ample experimental data to support this conclusion. Kleitman

(1963) reviewed the early studies concerned with the "modifiability of the 24-hour periodicities." These studies included the "effects of environmental conditions" on rhythms (e.g., light-dark or feeding routines); "shifts in phase"; and "non twenty four hour" periodicities. Most of these studies were concerned with whether various markers such as temperature, endocrine levels, heart rate, or renal measures would track experimental schedules. While Kleitman concluded that there was "no foundation for assuming that some cosmic forces determine the 24-hour rhythm . . . it is impossible to state the causative factors that contribute to the apparent fixity of the 24-hour rhythm" (p. 180).

In the 1970s a series of studies focused on sleep-waking characteristics of varied sleep cycles (Weitzman et al., 1974; Carskadon and Dement, 1975; Moses et al., 1975; Webb and Agnew, 1975, 1977; Webb, 1978). These studies (Table 5.1) ranged from ultrashort schedules (1-hr wake time/0.5-hr sleep time) to ultralong schedules (32-hr wake time/16-hr sleep time). In each study, sleep was measured electroencephalographically and the schedule of wake and sleep time was systematically maintained.

The results of these studies are summarized in Table 5.1. It shows the percentage of sleep time that occurred during the scheduled sleep period of the maintained regimen. It is apparent that, as one moves from a 8/16 schedule, sleep becomes increasingly ineffective.

The dynamics of this process are quite apparent when one examines the sleep characteristics of the available sleep periods. Sleep demand levels are predicted to be a function of prior wakefulness. In the short regimens the "required" eliciting level is less than the 16-hr value. The lower the demand level, the longer the sleep latency, and these latencies occupied the available sleep period. In the longer regimens, while the sleep onset latencies are short, the sleep period cannot be extended across the available long sleep period due both to "completed" sleep demand levels and circadian tenden-

TABLE 5.1. Percentage of sleep time that occurred during the scheduled sleep periods in studies investigating ultrashort to ultralong sleep schedules

Reference	Wake-sleep schedules (hr)	Sleep (%)
Carskadon and Dement (1975)	1/0.5	60
Weitzman et al. (1974)	2/1	56
Webb and Agnew (1975)	3/6	80
	4/8	84
	6/12	88
	8/16	93
	10/20	92
	12/24	88
Webb (1978)	16/32	77

cies. Furthermore, if these periods were "scheduled" at circadian times that have been shown to further shorten sleep, the sleep length would be further shortened.

It is possible to argue that, if these regimens were extended, the resultant cumulated "sleep debt" may shorten latencies in the short regimens and lengthen sleep in the long regimens. This may be possible; but such a heavy, accumulated "sleep debt" would be so substantial that it would make for ineffective performance during the waking period.

In summary, studies using ultrashort and ultralong sleep schedules with optimal sleep-waking ratios, that is, 2:1 ratios, indicate that the underlying laws of sleep cannot be "repealed."

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II

Circadian and Ultradian Components of the Sleep-Wake System

6

The Timing and Structure of Spontaneous Naps

SCOTT S. CAMPBELL

Napping behavior in adult humans has often been considered to reflect a type of sleep that is fundamentally different from major sleep episodes taken at night (Dinges and Broughton, 1989; Kleitman, 1963; Wever, 1979). They are differentiated not only quantitatively, but qualitatively. Whereas major sleep is acknowledged to be the expression of an endogenously generated, regularly recurring, biological need, napping has generally been considered to be a compensatory response to previous sleep loss or a behavioral reflection of boredom.

Yet several important characteristics of napping, not least of which is its ubiquity, raise questions regarding the exact nature of this type of sleep. Webb (1978) outlined features of napping that correspond closely to characteristics common to all biological rhythms: they are (1) temporally repetitive, (2) species-specific, (3) often developmental, (4) innate and unlearned, (5) adaptive, and (6) endogenously generated. Perhaps the most crucial of these characteristics necessary to designate napping as a "biological rhythm" is the last: proof of endogenous generation.

However, as Webb points out, data addressing this point have been slow forthcoming. In most experiments designed to examine endogenous rhythmicity in humans, napping has been viewed as a confounding influence on the primary measures of interest. As such, naps have been specifically prohibited in all but a few studies (e.g., Schaefer et al., 1967; Webb and Agnew, 1974; Campbell and Zulley, 1985; for a review, see Campbell and Zulley, 1989a).

If napping behavior is, indeed, another behavioral expression of the endogenous sleep-wake system of adult humans, then naps should conform to the same general rules that govern the temporal placement, duration, and internal organization of major sleep episodes. Thus, naps should (1)

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maintain a characteristic pattern of occurrence within a given time frame; (2) maintain a stable phase relationship with other components of the circadian system, such as body core temperature; and (3) reflect the influence of both circadian and homeostatic factors in their structural composition. This chapter examines spontaneous daytime sleep episodes within the framework of these assumptions.

Patterns of Occurrence

It has been acknowledged for quite some time that, under the entrained conditions of daily life, most napping tends to occur in the middle of the afternoon. This "postlunch dip" in alertness was so named because it was thought not only to be temporally related but also causally related to digestion of the midday meal (Kleitman, 1963). Indeed, as recently as 1980, a relationship between the propensity for afternoon napping and the timing of lunch was still being considered (Nakagawa, 1980). Nakagawa studied healthy young subjects during 10-12 hr of continuous bed rest, from approximately 0800 to 2000. Despite specific instructions prohibiting sleep, subjects were periodically "overcome by an uncontrollable desire to sleep." Based on the finding that sleep comprised over 55% of the interval between 1400 and 1700 and that mealtimes tended to cluster at a time just prior to that interval, the author concluded that "there seems to be a significant correlation between the two [events]."

In contrast, several investigators have confirmed that daytime sleepiness and corresponding performance decrements reach a peak in the afternoon, whether or not a meal is ingested (Blake, reported in Colquhoun, 1971; Stahl *et al.*, 1983). This tendency for spontaneous naps to occur in midafternoon, regardless of meal timing, was further demonstrated by a study we conducted using a bed-rest protocol (Campbell, 1984). Subjects spent 60 consecutive hours in bed during which they were given no instructions relative to when and when not to sleep. Meals were served at irregular intervals throughout the bed-rest'period, and one subject fasted for the duration of the study. Over the entire study, meals were served during 37 waking episodes; no meals were served during 47 waking periods. There was no difference in the duration of the two groups of waking episodes, nor was there a difference in the duration of sleep episodes following waking periods in which meals were eaten and those following waking periods during which meals were not eaten. Moreover, no significant differences were found between the subject who fasted and mean values of the group, in any sleep or waking parameter measured. Such findings strongly suggest that the influence of meals on daytime sleep patterns is negligible.

While a substantial body of evidence indicates that the propensity for

afternoon napping is quite strong, the postlunch dip may not be the only time at which daytime sleep tendency is high. Both bed-rest studies described above found that subjects also showed a strong propensity to initiate naps at an earlier time of the day. Indeed, Nakagawa reported that his subjects spent a greater proportion of time asleep between 0800 and 1100 than during any other 3-hr time block. This, despite the fact that subjects had had a full night's sleep prior to the day of bed rest. Similarly, our subjects who were confined to bed for 60 hr initiated more sleep episodes between 1000 and 1200 (17% of all sleep episodes) than during any other 2-hr interval within the 24-hr day. Zulley (1988) also has reported that well-rested subjects confined to bed for 32 hr exhibited a strong tendency for napping in the late morning.

Thus it appears that spontaneous daytime naps do maintain a general pattern of occurrence within the 24-hr day. Under the entrained conditions of daily life, there is little question that the most common time for such naps is in the midafternoon. However, in less-structured environments there is a clear, and perhaps equally strong, propensity for naps to occur in the late morning. Several investigators have speculated that these multiple "preferred" nap times reflect the existence of an ultradian, 4-hr component of the human sleep-wake system that may underlie the more robust circadian component (Campbell, 1984; Lavie and Scherson, 1981; Nakagawa, 1980; Zulley, 1988).

Phase Relationships

That napping shows a general pattern of recurrence within the 24-hr day suggests that these daytime sleep episodes may maintain stable phase relationships with other aspects of the circadian timing system. The possible relationship between an apparent tendency for napping in the late morning and other components of the circadian system remains unclear. However, there is quite convincing evidence that midafternoon sleep propensity is intimately linked to the circadian oscillation in body core temperature.

The relationship between body temperature and major sleep is well established. When subjects are studied for extended periods in environments without time cues, major (subjectively, nocturnal) sleep occurs in coincidence with the daily trough of body core temperature (Wever, 1979). That is, subjects tend to initiate sleep just prior to their daily temperature minima and they awaken several hours following the minimum, on the rising portion of the temperature curve. This relationship between sleep and temperature is illustrated in Figure 6.1.

Although in most such studies napping is expressly prohibited, subjects sometimes have found it impossible to maintain wakefulness throughout the subjective day. In those cases, the subjects are typically requested to

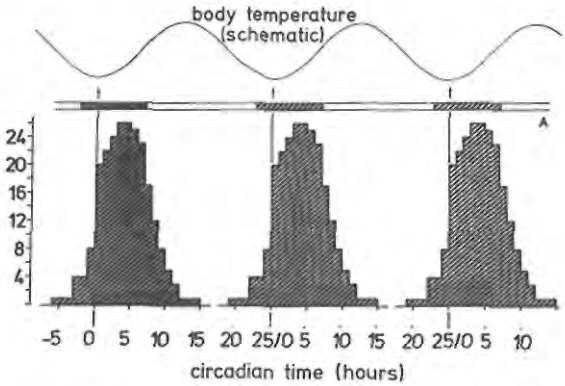

FIGURE 6.1. Distribution of the frequency with which major sleep episodes were initiated under time-free conditions for each hour of the circadian day relative to the circadian course of body core temperature. Successive temperature minima are identified by 0 and 25/0 on the time axis. Major sleep is typically initiated just prior to the temperature minimum. Reprinted with permission of Gustav Fischer Verlag, from Zulley J, Campbell, S (1985): The coupling of sleep-wake patterns with the rhythm of body temperature. In: *Sleep '84*, Koella W, Ruethar E, Schulz H, eds. New York: Gustav Fischer Verlag, pp 81–85.

indicate their intention to nap (and the end of the nap) by giving a designated signal. When the timing of such self-designated naps is analyzed, it becomes apparent that their occurrence is not randomly distributed across the circadian day, but rather that naps are initiated at "preferred phase positions" relative to temperature (Zulley and Campbell, 1985a). The results obtained from 6 subjects who reported napping during their time in isolation are illustrated in Figure 6.2.

Subjectively perceived naps occurred generally in two locations relative to the temperature cycle. One group of "naps" occurred at the same phase at which major sleep episodes occurred. Though these sleep periods were perceived by subjects as "naps," they lasted for an average of 6.3 hr (SD = 2.9 hr). It is clear both from their duration and the phase position at which they occurred that these sleep episodes do not fit the usual definition of a nap. They were perceived as naps probably because subjects felt that they had not been awake for a sufficient time since their last major sleep to designate the waking interval as a complete day (for further explanation of this point, see Zulley and Campbell, 1985a; or, Campbell and Zulley, 1989a).

A second group of self-designated naps that correspond more closely to the usual view of naps (mean duration: 2.4 hr; SD = 1.1 hr) showed a strong tendency to cluster approximately 180° away from the minimum of body core temperature, that is, around the temperature maximum (see Figure 6.2). While less robust than the propensity for sleep at the body temperature minimum, the finding that short naps typically occur halfway between successive temperature minima clearly suggests the existence of a second preferred phase position for sleep that is closely tied to the

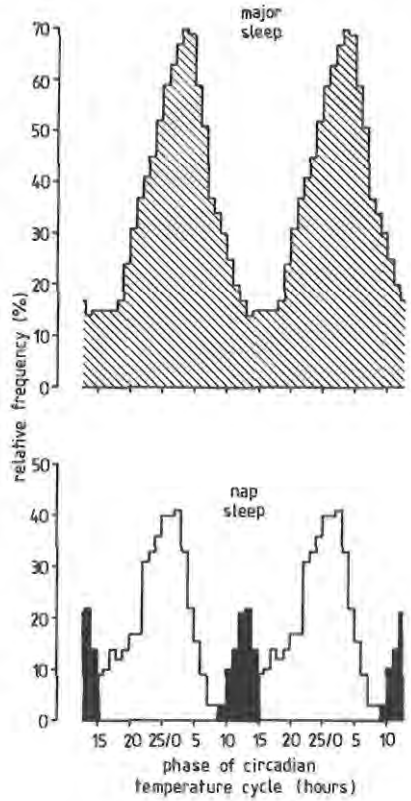

FIGURE 6.2. Location of the occurrence of subjectively perceived naps relative to the circadian course of body core temperature. Naps showed a tendency to occur at two general phase positions: around the minimum and around the maximum of body temperature. Data were obtained from 6 subjects living in time-free environments. Reprinted with permission of Springer-Verlag, from Zulley J, Campbell S (1985): Napping behavior during "spontaneous internal desynchronization": Sleep remains in synchrony with body temperature. *Hum Neurobiol* 4:123–126.

endogenous circadian timing system, a notion first proposed by Broughton in 1975.

Further support for this view comes from a subsequent study in which we examined the spontaneous sleep of 9 young adults living in a "disentrained" environment for 72 consecutive hours (Campbell and Zulley, 1985). Subjects had no cues to time of day and they were instructed to eat and sleep when inclined to do so. Moreover, in an effort to increase the likelihood that subjects would express biological sleep propensity in the unequivocal form of napping, we severely restricted behavioral options in the environment. Subjects were prohibited from reading, writing, listening to music, doing heavy exercise, and so on. The results of that study are summarized in Figure 6.3, which shows a summation histogram of subjects asleep during

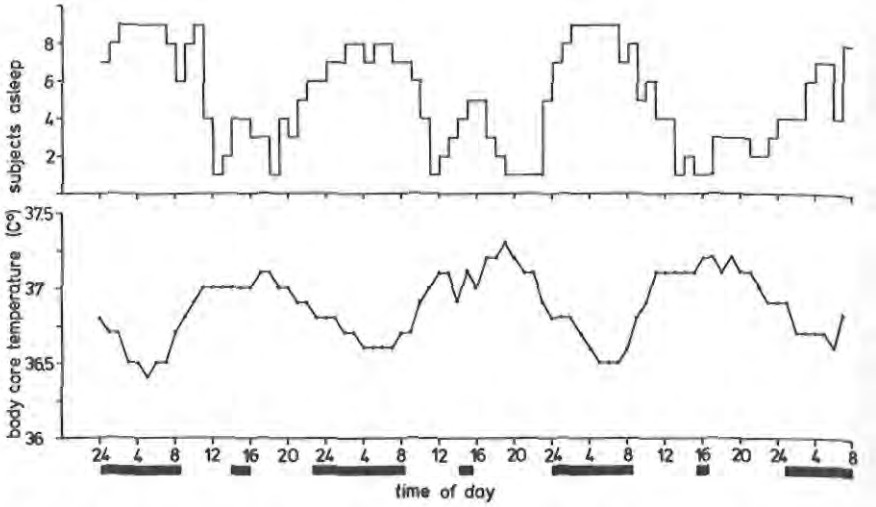

FIGURE 6.3. Relationship between sleep episodes recorded in a disentrained environment and circadian rhythm of body core temperature. Above the temperature curve (averaged for 9 subjects) is a summation histogram of the number of subjects asleep during any given hour during the experimental period. Below the temperature curve (black bars) are average onsets and durations of major sleep episodes and naps. *Reprinted with permission of Springer-Verlag, from Campbell S, Zulley J (1985):* Ultradian components of human sleep/wake patterns during disentrainment. In: *Ultradian Rhythms in Physiology and Behavior*, Schulz H, Lavie P, eds. New York: Springer-Verlag, pp 234–255.

each hour of the disentrainment period, the average course of body core temperature for the group, and the average onset and duration of major sleep episodes and naps taken by the group during the period.

Both major sleep episodes and naps showed clear phase relationships with body core temperature. As in all time-free environments, major sleep occurred in association with each day's temperature minimum. In addition, there was a clear tendency for average nap times to occur in correspondence with daily maxima in temperature. Specifically, when the onset of each nap was examined relative to the absolute temperature maximum in the corresponding circadian day, it was found that 23 of the 26 naps were initiated just *prior* to the maximum temperature value.

It can be concluded from these findings that spontaneous naps not only exhibit a pattern of occurrence within the entrained, 24-hr day, but that their rhythmic recurrence is closely tied to the circadian rhythm of body core temperature. Since the circadian course of body temperature has long been considered to be one of the best reflections of underlying mechanisms governing the circadian timing system (i.e., the "hands of the clock"), these data give further credence to the argument that napping is actually the expression of one component of the biological rhythm of sleep and wakefulness.

Influences on Nap Structure

Can it be said, then, that naps are simply miniature major sleep episodes timed to occur approximately 180° out of phase with normal nocturnal sleep? Is the structure of naps governed by the same homeostatic and circadian rules that dictate the form of major nocturnal sleep? If so, one would hypothesize reduced proportions of REM sleep in naps, since their occurrence coincides with the circadian phase at which low REM propensity has been demonstrated. Likewise, reduced amounts of slow wave sleep (SWS) in naps would be predicted, since the occurrence of this sleep state is presumed to be dependent on the duration of prior wakefulness, and wakefulness preceding naps is relatively brief compared to major sleep episodes.

When we examined the structural components of the naps taken during the 72-hr disentrainment study described previously, these predictions were generally confirmed. Naps had 30% less REM sleep than did major sleep episodes. Similarly, median SWS percent during naps was only 4.9% compared to 14.1% for major sleep periods. Other studies have found similar reductions in REM and SWS amounts, when compared with nocturnal sleep values recorded from age-matched subjects (Nakagawa, 1980; Campbell, 1984; see Table 2 in Campbell and Zulley, 1989a). In very general terms, then, the composition of naps may be said to follow the same rules governing that of major nocturnal sleep.

However, when analyzed more closely, it becomes clear that the composition and structure of naps are subject to a more complex set of rules. In disentrainment, we found two rather distinct groups of naps. While the two groups did not differ significantly in terms of their average durations, or in the amount of waking time preceding them, they showed certain differences with respect to their composition. On the one hand, there was a group of daytime sleep episodes (about half of total naps taken) in which normal sleep-stage sequencing always occurred, with an interval of SWS always preceding the first episode of REM sleep. Moreover, all stages of sleep were present in these naps. On the other hand, a second group of naps was characterized by a virtual absence of SWS and, in some case, the absence of both REM and SWS. Thus, one group of naps closely resembled nocturnal sleep in structure, while the other group more closely fit the usual view of naps as being relatively light sleep episodes.

Interestingly, the two groups of naps could be separated not only on the basis of their structural differences, but also by their differential placement within the overall nap distribution. The naps containing all sleep stages, occurring in normal sequence, clustered in the middle of the nap distribution (i.e., the body temperature maximum, roughly between 1400 and 1700), whereas the "incomplete" naps fell at either end of the distribution. Such clustering was particularly evident with respect to the presence of SWS (Campbell and Zulley, 1989b). Figure 6.4 shows the relationship between

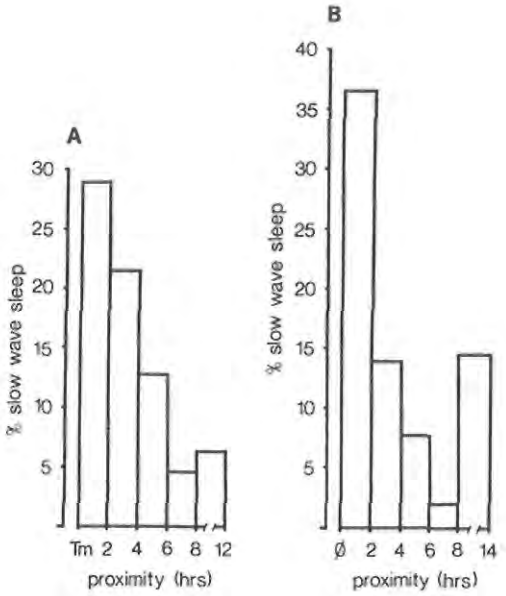

FIGURE 6.4. Average percentages of slow wave sleep (SWS) contained in naps as a function of their proximity to the respective circadian temperature maxima. In *Figure 6.4A proximity to absolute temperature maximum is shown. In Figure 6.4B*, proximity to cosine-fitted acrophase is shown. Naps occurring within 4 hrs of the temperature maximum contained significantly more SWS than naps occurring at other times. Copyright © 1989, The Society for Psychophysiological Research. Reprinted with permission of the publisher, from Campbell and Zulley (1989b).

proportions of SWS within naps and the body temperature maximum for the corresponding day. The strong association between SWS propensity in naps and circadian phase of occurrence is quite apparent. Naps occurring within 4 hr of the temperature maximum contained an average of 24.6% SWS compared to 6.8% SWS in naps initiated at other times. Similarly, Nakagawa (1980) found a higher percentage of SWS in naps initiated between 1400 and 1700 than in naps initiated during any other 4-hr block throughout the day. In neither of these studies was duration of prior wakefulness a significant factor in the differential placement of SWS.

In summary, the structure of naps as a whole appears to be determined by the same general rules that govern the makeup of major nocturnal sleep. The well-established circadian rhythm of REM sleep propensity (Czeisler et al., 1980; Zulley, 1980) that increases the likelihood of its occurrence around the temperature minimum, results in reduced proportions of that sleep state during naps because of their tendency to cluster around the temperature maximum. Similarly, the well-documented link between SWS propensity and prior wakefulness (Webb and Agnew, 1971; Knowles et al.,

1986) restricts SWS amounts in naps, since they are typically preceded by only a few hours of waking. However, in the case of SWS, there also appears to be a strong circadian component. Such a circadian influence on the occurrence of SWS in major nocturnal sleep has only been hinted at (Webb and Agnew, 1971; Hume and Mills, 1974).

Conclusions

The findings reviewed in this chapter argue convincingly for the hypothesis that napping behavior in adult humans is the behavioral expression of an underlying, endogenous biological rhythm of daytime sleep propensity. Spontaneous naps do not occur randomly throughout the day. Rather, they recur with a striking regularity. Moreover, the data indicate that both the timing and composition of naps are probably regulated by the same mechanisms that dictate the occurrence and structure of major nocturnal sleep. Just as nocturnal sleep is tied to the *minimum* of body core temperature, naps are tied to the *maximum.* The composition of both major nocturnal sleep and of naps is determined by two principal factors: circadian phase (for REM sleep) and prior wakefulness (for SWS).

Does the finding that the occurrence of SWS in naps has a circadian component reflect a fundamental difference between naps and major nocturnal sleep? Certainly, the same robust circadian effect on SWS has not been observed in nocturnal sleep, where it has been estimated that over 90% of the variance in SWS amounts may be accounted for by duration of prior wakefulness (Knowles et al., 1986). However, a possible circadian influence has been suggested by some investigators. Both Webb and Agnew (1971) and Hume and Mills (1974) reported a modest circadian influence on nighttime SWS, though both groups concluded that the effect was not significant relative to that of prior wakefulness. Nevertheless, such findings do suggest that the same mechanisms are involved in determining the composition of night and day sleep, albeit to differing degrees.

With these findings in mind, it would seem difficult, if not impossible, to justify placing napping outside the endogenous sleep system comprising nocturnal sleep. Yet, sleep researchers and chronobiologists, as well as the general public, frequently continue to view naps in that way. For example, the large majority of mathematical models designed to "explain" human sleep do not include daytime sleep episodes in their parameters and cannot mimic their occurrence within the 24-hr day (see, for example, Moore-Ede and Czeisler, 1984). Likewise, most experimental protocols continue to prohibit napping, since it is often viewed as a source of experimental variance (Webb, 1978). Perhaps the most explicit demonstration of "discrimination" against the study of spontaneous napping in the laboratory is that most human sleep studies continue to record polysomnograms only

between about 2300 and 0700, rather than around the clock. This is equivalent to a geologist studying only the large mountain ranges because "that's where most of the rocks are."

The inclination to view naps as something other than an inherent part of the human sleep-wake system is likely to decline as we continue to learn more about the nature and function of these daytime sleep periods. Indeed, substantial progress has been made in this regard within the past several years, as evidenced by this book and similar volumes dedicated to the study of napping and polyphasic sleep. Moreover, the judicious use of naps to relieve, or prevent, fatigue in the workplace is gaining wider acceptance, and such applications will, in turn, lead to a better understanding of the role of napping within the sleep-wake system. Until that time, however, napping probably will continue to be viewed by many in terms of the definition that Webb (1978) quoted from a fictional Doctor Kentish, that is, as little more than "any rest period up to twenty minutes duration involving unconsciousness but not pajamas."

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7

The Impact of Irregular Sleep-Wake Schedules on Circadian Rhythms and the Role of "Anchor" Sleep

D. S. MINORS AND J. M. WATERHOUSE

In humans the inherent period of the internal oscillator controlling overt circadian rhythmicity is, on average, 25 hr (Wever, 1979). However, when we live under normal nychthemeral circumstances, the internal circadian oscillator is synchronized to a period of exactly 24 hr. The adjustment is achieved by means of rhythmic cues from the external world, for example, the alternation of light and dark, the sleep-wake cycle, social influences, known as Zeitgebers (Aschoff, 1954; Wever, 1979; Minors and Waterhouse, 1981a; Moore-Ede et al., 1982).

Such synchrony is upset by changes in the routine of sleep and wakefulness, as after time-zone transitions and during shift work, and there is believed to be a link between an abnormal phasing of circadian rhythms and what is commonly called "jet-lag syndrome" and the general malaise experienced by workers subjected to shift changes. The problems are particularly marked for those who live irregular schedules of sleep, duty, and leisure, such as aircrews and military personnel. Not only will such schedules lead to occasions when work (often lasting for an extended period of time) and sleep are being attempted at inappropriate times of the circadian cycle, but they also will lead to a loss of Zeitgebers with a 24-hr period (Graeber et al., 1986). That is, sleep —and, as a consequence, activity, meals, light, and social influences — will have to be taken when there is opportunity rather than when it might be "best" (as determined by habit and by biological or social factors). In such circumstances, sleep is often taken not over a single period of 8 hr but rather split into several episodes. Our basic program of research, summarized in this chapter, has been to investigate what effect such irregular sleep-activity schedules have upon the circadian timing system and whether circadian rhythms are influenced by the timing of split sleeps.

WHY WE NAP
Evolution, Chronobiology, and Functions of Polyphasic and Ultrashort Sleep

In analyzing our results we have developed experimental and mathematical methods that have enabled us to separate the component of a measured rhythm that is due to the internal oscillator (the endogenous component) from that due to external factors (the exogenous component) such as mealtimes, sleep-activity, time spent awake.

Subjects and Materials

Our experiments have been performed upon volunteer university students, aged 18-21, who have been studied in an isolation unit in groups of 2-5. Some details of the numbers of subjects and experimental protocols are given in Table 7.1. In the isolation unit the ambient air temperature and humidity were maintained constant; during the subject's wakefulness it was lit at 300 lux but was unlit during times of sleep. Further details of this unit have been described elsewhere (Elliott et al., 1972). Initially, subjects were studied for a 5-day control period living on a customary nychthemeral routine. They slept between 2400 and 0800 and ate meals at their customary times. After this control phase, subjects entered the experimental phase, during which they were asked to sleep at irregular times according to one of six designs, even though they continued to eat at times as nearly as possible the same as those during the control phase. Lighting was under the control of the subjects; they were instructed to take all sleep periods in the dark.

Throughout all experiments, subjects micturated on rising from bed and

TABLE 7.1. Details of numbers of subjects and protocols

Group	Number	Anchor sleep	Constant routine start times A,B[a]	Comments
A	8	–	–	Normal 8-hr sleep
B	4	–	–	Irregular 8-hr sleep
C	5	–	–	Irregular 4-hr sleep
D	8[b]	2400–0400	–	
E	4	0400–0800	–	–
F	4	0800–1200	–	–
G	2	1200–1600	–	–
H	4	–	0400,0400	Normal 8-hr sleep
I	8	2400–0400	0400,0400	–
J	8	2000–2400	0400,0400	–
K	8	2000–2400	0400,2400	–
L	7	2000–2400	0800,2400	–

[a]A, following the control phase (sleep times: 2400–0800); B, following the experimental phase (anchor sleep).

[b]Two of the subjects had a reversed sequence of irregular sleep compared with the other 6 subjects.

every 2 hr thereafter while awake. The volume of all urine passed for each collection period was noted and an aliquot refrigerated for subsequent analysis for sodium, potassium, chloride, creatinine, inorganic phosphate, calcium, and urate. For each period of collection of urine the flow rate and excretion rates of each of the constituents in urine were determined. Rectal temperature was also measured using a thermistor probe placed 10 cm beyond the anal sphincter. During the hours of wakefulness this temperature was measured by the subjects every hour; during sleep it was telemetered hourly.

Rhythms have been sought by the single cosinor analysis (Nelson et al., 1979). This involves fitting a 24-hr cosine curve to each day's data using a linear least squares technique. The maximum of this fitted cosine curve, termed the acrophase, is used to assess the phase of the rhythm on that day.

We will consider our experiments in three phases: (1) the original "anchor sleep" experiments (Minors and Waterhouse, 1981b); (2) experiments using constant routines (Minors and Waterhouse, 1983); and (3) reanalysis of data by a method that attempts to divide temperature data that have been obtained under nychthemeral conditions into endogenous and exogenous components.

Phase 1: The "Anchor Sleep" Experiments

Our original experiments were designed to investigate how the human circadian system is affected by irregular sleep-wake cycles and by splitting the habitual 8-hr sleep period into two 4-hr periods. All designs have been compared with experiments on 8 subjects (Group A) who slept at a conventional time (2400-0800) throughout a 3-wk stay in the isolation unit.

In the first design (see Figure 7.1 A) 4 subjects (Group B) took a single 8-hr sleep per day, but at a different time each day. The ordering of these times was randomized. In the second experimental design (see Figure 7.IB) 5 female subjects (Group C) took their sleep in two 4-hr periods per day. The time of one of these, labeled with a triangle in the figure, was such that its midpoint was at the same time as that of the 8-hr sleep on the corresponding day for subjects of Group B. The other 4-hr sleep was always begun 12 hr earlier.

In the remaining experimental designs, the customary 8 hr of sleep per day were again divided into two 4-hr periods. By contrast, however, one of these —the "anchor sleep"—was taken at a constant time on each day: 2400-0400, 0400-0800, 0800-1200, or 1200-1600. The second 4-hr sleep taken in these experiments was at a different time on each successive day. For subjects of Group D, whose anchor sleep was 2400-0400 (see Figure 7. IC), the irregularly taken 4-hr sleep was at the same time as that taken on

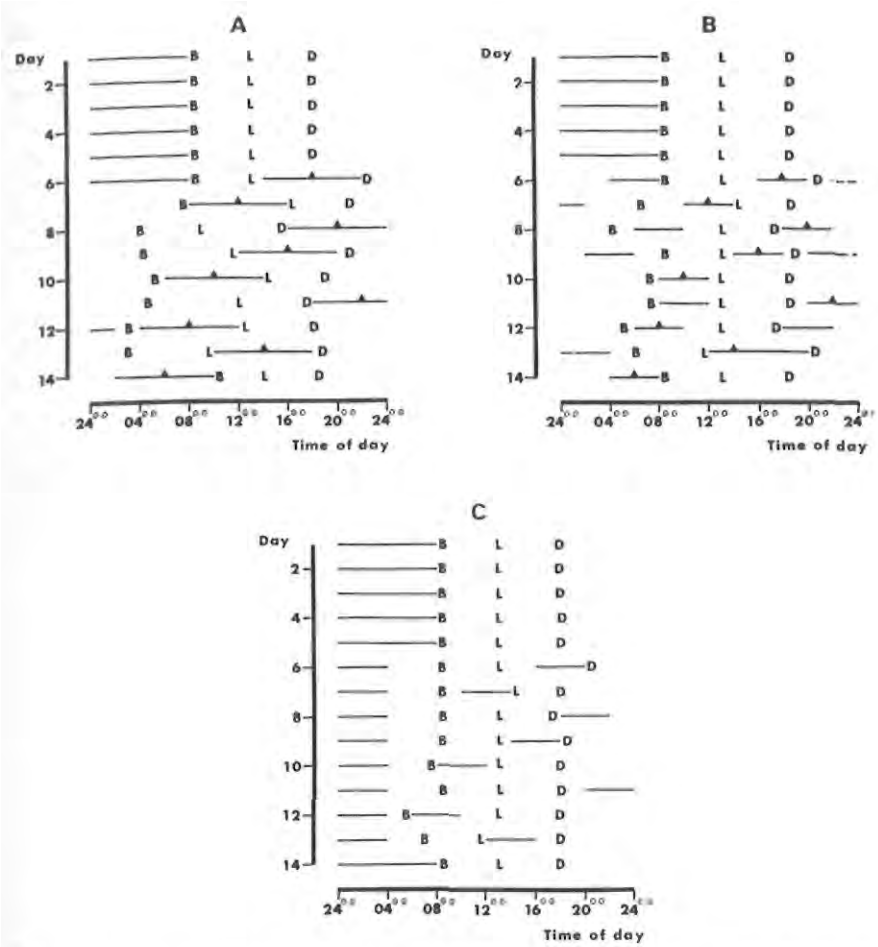

FIGURE 7.1. Experimental protocols for *(A)* Group B; *(B)* Group C; *(C)* Group D. Successive days in isolation unit from above downward. Mealtimes indicated as B (breakfast), L (lunch), or D (dinner). Horizontal bars represent times the subjects were allowed to sleep; the midpoints of irregularly placed sleeps are shown in parts *(A)* and *(B)*.

the corresponding day by Group C and labeled with a triangle in Figure 7.IB. For the other three designs, these irregularly taken sleep periods were 4 hr (anchor sleep 0400-0800, Group E), 8 hr (anchor sleep 0800-1200, Group F), or 12 hr (anchor sleep 1200-1600, Group G) later than on the corresponding day for the 2400-0400 anchor sleep experiment. To minimize the effects of sleep deprivation when the anchor sleep was taken at 0800-1200 or at 1200-1600, the irregularly taken sleep was advanced by one day and so was placed before the anchor sleep rather than after.

Examples of the behavior of the acrophases of 24-hr cosine curves fitted to data from subjects of Groups B and C are shown in Figure 7.2. In both cases it can be seen that during the experimental phase, when the subjects slept at irregular times, the acrophase was initially similar to that determined by fitting a 24-hr cosine curve to the entire control phase. Thereafter, the acrophases became progressively later, indicating that the period of the rhythms was greater than 24 hr; calculation showed the mean period of rhythms during the experimental phase was significantly ($p < .05$) greater than 24 hr (24.52 ± 0.19 hr, Group B; 24.68 ± 0.16 hr, Group C).

That is, despite the fact that meals were taken at regular times throughout, stable 24-hr rhythms were not obtained; rather, rhythms appeared to free-run with a period greater than 24 hr. The possibility that such a result was due to living in the isolation unit, in which conventional social Zeitgebers are impoverished, was eliminated by the results obtained from Group A who slept at normal times. During these experiments the average period of the body temperature rhythm did not differ significantly from 24 hr and there was an average phase delay of less than 2 hr between the beginning and end of the 21-day experiment (compare with Figure 7.2, for example).

Analysis of the data from Group D showed that the acrophases of the cosine curves fitted to the experimental phase remained at a fairly constant time throughout, and at one that was similar to that determined during the control phase (see Figure 7.3). The results from the 2 subjects for whom the

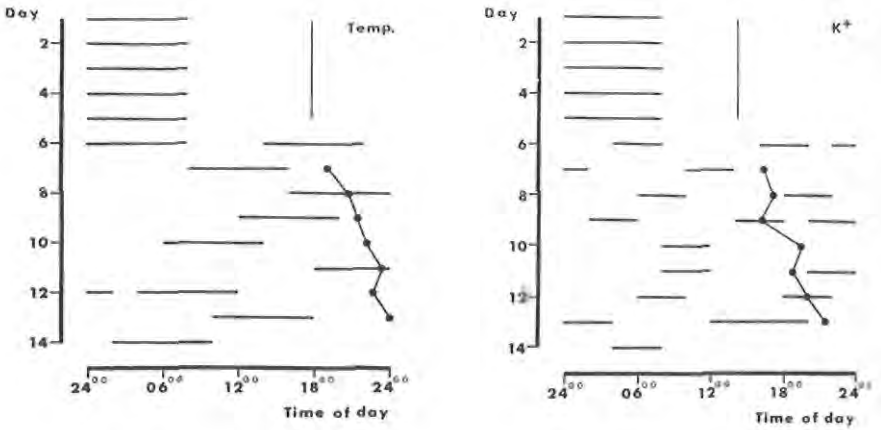

FIGURE 7.2. *Left:* Rectal temperature rhythm in a subject of Group B. *Right:* Urinary potassium rhythms in a subject of Group C. Acrophases of 24-hr cosine curves fitted to 72 hr of data pergressively incremented by 24 hr. Acrophase of 24-hr cosine curve fitted to all control phase is shown as a vertical line. Reprinted with permission of Gordon & Breach, Science Publishers, from Minors DS, Waterhouse JM (1981): Anchor sleep as a synchronizer of rhythms on abnormal routines. *Int J. Chronobiol* 7:165–188.

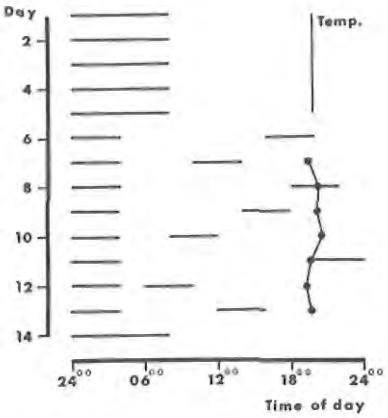

FIGURE 7.3. Rectal temperature rhythm in a subject of Group D. Data plotted as in Figure 7.2. Reprinted with permission of Gordon & Breach, Science Publishers, from Minors DS, Waterhouse JM (1981): Anchor sleep as a synchronizer of rhythms on abnormal routines. *Int J Chronobiol* 7:165–188.

ordering of the irregularly taken sleeps over successive days was reversed were indistinguishable from those of the rest of the group. This stability has been confirmed by determining the period of the rhythms during all but the first 4 days of the experimental phase. (The reason for omitting the first 4 days of the experimental phase was to remove the effects of any transient changes that might occur immediately after subjects started sleeping at irregular times.) The mean period of all rhythms from subjects of Group D was not significantly different from 24 hr.

Such results would lead one to speculate that taking an anchor sleep at times other than 2400-0400 would also result in stable 24-hr rhythms. For Group E, whose anchor sleep was 0400-0800, the acrophases were stable during the experimental phase even though they were sometimes a few hours later during the experimental phase than in the control phase. As previously, the mean period of the rhythms for all variables was determined over all but the first 4 days of the experimental phase and this was not significantly different from 24 hr. Figure 7.4 shows examples of the behavior of the acrophases fitted to data from Groups F and G. In both cases it can be seen that, during the latter part of the experimental phase, the acrophases were constant but that this stability was not gained immediately. In the initial part of the experimental phase the acrophases became progressively later so that, once stability was attained, they were delayed when compared with the initial control phase. To confirm stability during the last 96 hr of the experimental phase, the mean period of all rhythms was determined; in neither group was this mean significantly different from 24 hr.

In all the experimental designs, meals were eaten at times as near as

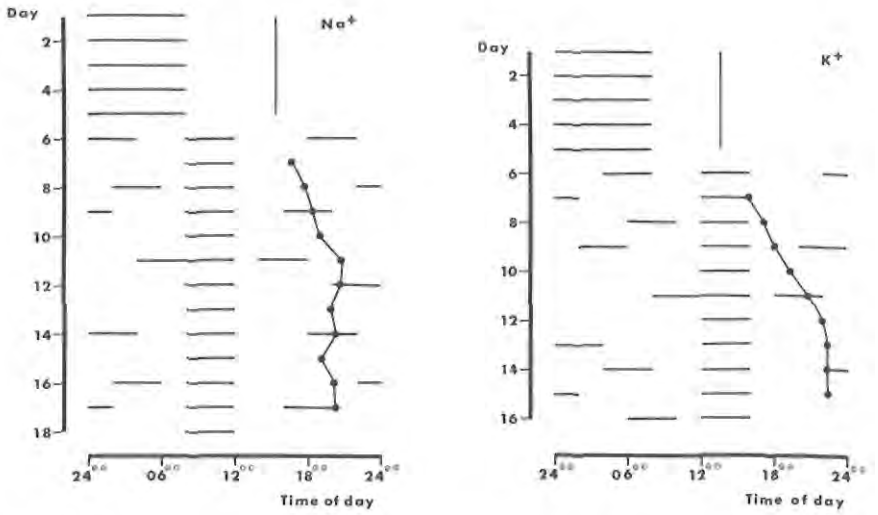

FIGURE 7.4. *Left:* Urinary sodium rhythm in a subject of Group F. *Right:* Urinary potassium rhythm in a subject of Group G. Data plotted as in Figure 7.2. Reprinted with permission of Gordon & Breach, Science Publishers, from Minors DS, Waterhouse JM (1981): Anchor sleep as a synchronizer of rhythms of abnormal routines *Int J Chronobiol* 7:165–188.

possible to those during the control phase. The possibility exists, therefore, that the regular mealtimes were responsible for the stable 24-hr rhythms found in Groups D-G. But there is evidence against such a view. First, stable 24-hr rhythms were not obtained in Groups B and C who also ate meals at the same time as during the control phase (see above). Second, for Groups F and G the acrophases of the 24-hr rhythms should not have been different from those determined during the initial control days if mealtimes had been a dominating influence. In contrast, a shift due to the anchor sleeps seems likely since, when the differences in acrophase from that during the control phase were calculated, they were similar to the change in midsleep times (about 8 hr in Group G and 6 hr in Group F).

A difficulty of interpretation of these data exists, however, since it is known that sleep also exerts a direct, "masking" effect upon many rhythms, generally decreasing the value for a variable (Aschoff, 1988; Minors and Waterhouse, 1989; Waterhouse and Minors, 1988). Thus the stability might have resulted from the *regular masking effect* of the 4-hr sleep and the internal oscillator need not have been synchronized to a 24-hr day, but rather have continued to free-run. The further observation that the stabilized rhythms showed a constant relationship to midsleep when anchor sleep was taken at different times might be explained either by a masking effect of sleep or by sleep acting as a Zeitgeber. However, this is unlikely to be a full explanation of the results since, when anchor sleep was taken at

times far removed from normal sleep times (Groups F and G), the new phase of the circadian rhythms was not achieved immediately (see Figure 7.4). This lack of an immediate phase response strongly suggests the slower process of adjustment of an internal oscillator rather than the immediate effect of masking.

Phase 2: The "Constant Routine" Experiments

A better indication of what is happening to the internal oscillator during such experiments requires us to investigate the endogenous component of circadian rhythms in more detail.

One way in which the endogenous component can be studied with less interference is to attempt to minimize exogenous rhythmicity. This can be achieved by requiring subjects to adhere to a "constant routine" (Mills et al., 1978; Minors and Waterhouse, 1984); that is, for a period of 24 hr, subjects are required to stay awake in surroundings of constant temperature, lighting, humidity, and social contact, and to take regular, identical snacks throughout.

The first protocol (Group H) was a control experiment in which subjects slept and ate at normal times (sleep 2400-0800) except on two occasions (in the middle and 5 days later at the end of the experiment) when this routine was interrupted at 0400 for a 24-hr constant routine. All other protocols (Groups I-L) were divided into two phases: an initial control phase of 5 days during which subjects slept at a conventional time (2400-0800), followed by the experimental phase during which sleep was taken in two 4-hr periods as in the experiments described in Phase 1. At the end of the control phase and again at the end of the experimental phase subjects underwent constant routines. Details of the times of anchor sleeps and the starts of the constant routines are given in Table 7.1.

The data from the two constant routines were analyzed by cross-correlation. Data from the first (control) constant routine were shifted by hourly increments (0-23) to produce the data set D1. For each of these shifts the control constant routine data were also shifted by hourly increments (0-23) to produce data set D2. The two data sets, Dl and D2, were then added together in each of the 11 ratios: 10:0, 9:1, 8:2, . . . , 1:9, 0:10 to produce sets of "trial data." In practice, the duplicate permutations — for example, Dl shifted 5 hr/D2 shifted 10 hr compared with Dl shifted 10 hr/D2 shifted 5 hr —were analyzed only once; even so, over 3000 sets of trial data were produced. Each set of trial data was then compared with the experimental data set from the second (experimental) constant routine. The set of trial data that gave the highest correlation coefficient was taken to be a description of the phase changes to the control constant routine that had taken place during the experimental phase of the protocol.

When the acrophases obtained from days when sleep was allowed during the experimental phase were considered ("nychthemeral data" measured in the presence of masking), results from Group I similar to those found with Group D (above) were obtained. That is, rhythms were stable (their period not significantly different from 24 hr) and their phase was similar to that during the control days.

Figure 7.5 shows a representative example of the urinary potassium data from the two constant routines of a subject from Group I. It can be seen that, during both constant routines, the times at which potassium excretion reaches a maximum and a minimum are similar. However, high rates of

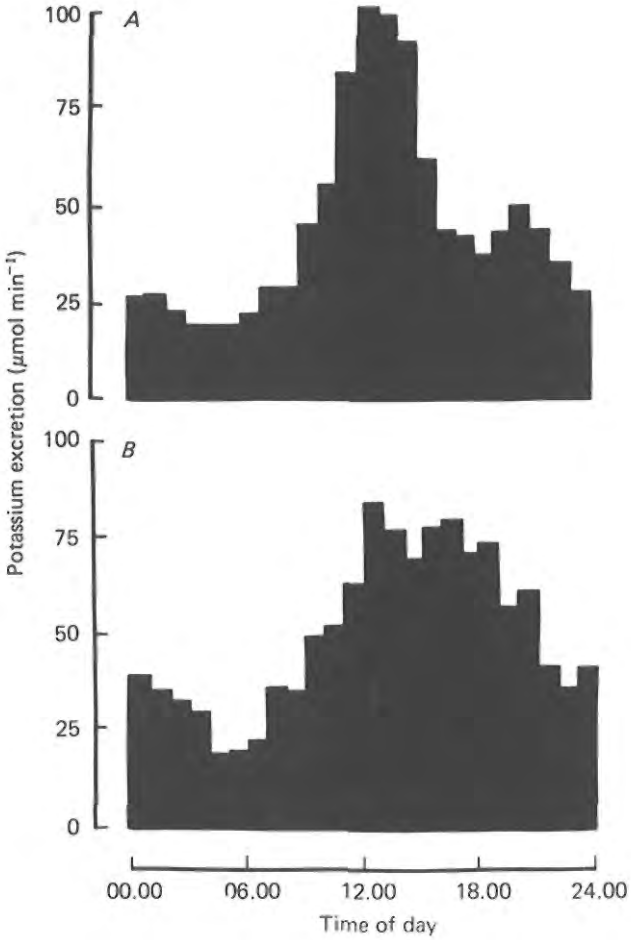

FIGURE 7.5. Urinary potassium excretion rates in a subject from Group I during the two constant routines: *(A)* during the control constant routine; *(B)* during the constant routine following the days when anchor sleep had been taken. Reprinted with permission of the authors, from Minors and Waterhouse (1983).

excretion are more protracted in the second constant routine than in the first. Analysis by cross-correlation of the data shown in Figure 7.5 yielded the highest correlation when the data from the second constant routine were correlated with "trial-data" formed from six parts of unshifted control data mixed with four parts of the same data phase-delayed by 5 hr. This analysis was performed on all variables and subjects for Group I and the shifts have been combined to give the frequency distribution shown in Figure 7.6. (Thus the example of Figure 7.5 would contribute "6" to the "zero shift" entry and "4" to the "5 hr delayed" entry of Figure 7.6.) When the distribution of shifts is considered, it can be seen that phase advances were infrequent; by contrast, the "best-fitting" mixture often contained a component that was unshifted together with a component that was phase-delayed by about 4 hr. A similar result was obtained when the distribution for each variable was displayed separately.

This splitting of rhythms into two components was not an artifact produced by (say) the stay in the isolation unit or by the two constant routines since it was not found in Group H, who spent a similar duration in

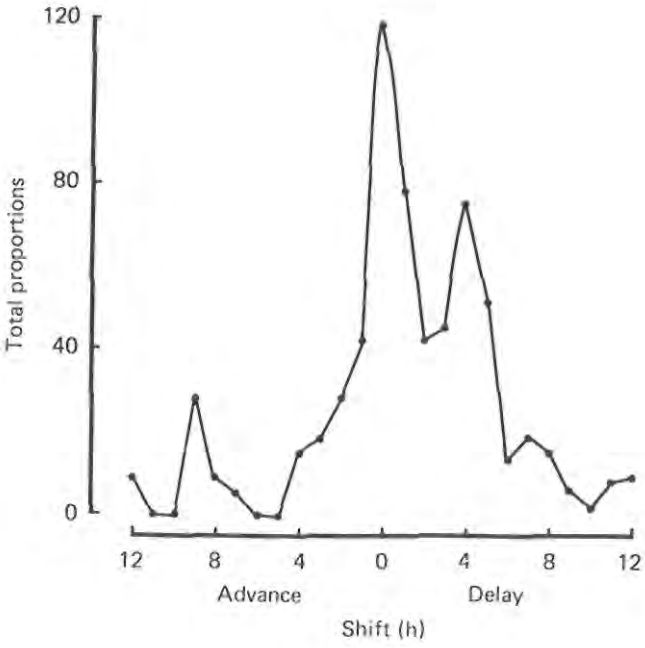

FIGURE 7.6. Proportions and phase-shifts of the data from the control constant routine that yielded the highest correlation coefficient when compared with the second constant routine (for further details see text). The figure shows summed proportions for all rhythms measured in Group I. Reprinted with permission of the authors, from Minors and Waterhouse (1983).

the isolation unit but who slept 2400-0800 each day. In this group, stability of acrophases was observed during the experimental days (as with Group A) and, when the constant routine data were analyzed as before, a component delayed by 4 hr was absent, the frequency distribution of shifts showing a single peak centered on zero shift.

Thus the two components in the data of Group I require another explanation. The unshifted component might be explained by some component of the human circadian system being entrained by the anchor sleeps. An alternative or additional explanation, however, is that it might be due to the two constant routines beginning at the same time (0400) and that there is a residual masking effect associated with the initial change from sleeping and fasting to starting the constant routine (this will be commented on more fully later). As regards the delayed component, possibly the anchor sleeps were unable to entrain these rhythms so that they free-ran with a period in excess of 24 hr. Alternatively, there might be some delay in circadian rhythms produced by cumulative sleep loss. This could arise in a protocol involving split sleeps, particularly if some sleeps were taken at the "wrong" circadian time. It is *not* due to sleep loss resulting from the constant routines as is indicated by the results from Group H.

To try to establish which of these options were appropriate we studied groups in which the times of anchor sleeps and the starting times of the constant routines were varied (see Table 7.1). We chose an earlier time for the anchor sleep (2000-2400) since we had already shown that anchor sleeps at times later than normal sleep times (Groups F and G) resulted in phase-delayed rhythms (at least measured under nychthemeral circumstances) and so a delay would not enable a distinction to be made between an entrained or free-running rhythm. In addition, by varying the time at which the two constant routines were started, we would investigate any effect that residual masking might have. A summary of these aspects of the protocols, together with the implications of possible experimental outcomes, is given in Table 7.2.

When the acrophases of the nychthemeral rhythms (measured when sleep was allowed) were considered, the shifts produced by the 2000-2400 anchor sleeps seemed to depend in part upon the variable under consideration. The rhythms of urinary water, calcium, phosphate, and urate excretion showed advances of about 2 hr, which suggests that they were considerably affected by the sleep-wake times and less by mealtimes (the timing of which did not change). By contrast, the acrophases of deep-body temperature and urinary potassium, sodium, and chloride excretion changed less, or even became delayed. The most parsimonious explanation of this is that the internal clock has not been phase-advanced (possibly it is even slightly delayed) and that masking effects due to the sleep-wake cycle increase with the sequence: body temperature/urinary potassium < urinary sodium/chloride < urinary flow/phosphate/urate/calcium.

The data from the constant routines in these experiments were cross-

TABLE 7.2. Summary of predicted phase-shifts if they were produced by residual masking (the starting times of the constant routines), the anchor sleep times, or an unentrained rhythm

	Constant routine			Anchor sleep		
	Start times					
Group	1st	2nd	Residual masking	Time	If rhythm entrained[a]	If rhythm unentrained
H	0400	0400	0 shift	Control	No change	Later
I	0400	0400	0 shift	2400–0400	0,2,4 hr earlier	Later
J	0400	0400	0 shift	2000–2400	4,6,8 hr earlier	Later
K	0400	2400	4 hr earlier	2000–2400	4,6,8 hr earlier	Later
L	0800	2400	8 hr earlier	2000–2400	4,6,8 hr earlier	Later

[a]Whether effect is due to sleep onset, midsleep, or waking, respectively.

correlated and the results combined as described previously. The results from such an analysis applied to all subjects and variables (together with those from Group I for comparison) are shown in Figure 7.7. This figure also shows phase changes that would be predicted for the residual masking effect of the constant routines, S; that had been found nychthemerally, N; and that would be predicted if they had been determined wholly by the time of anchor sleep, A.

The following general findings emerge when the results from Groups I-L are considered:

1. A shift that can be related to a particular phase of the anchor sleep (A) is not clear.
2. A shift close to, or coincident with, that observed nychthemerally (N) was present. (We note at this point that the standard errors of the mean value for N for Groups K and L were comparatively large.)
3. A delayed component (2-6 hr) was present.
4. A shift coincident with the effect of times of start of constant routines (S) was present.
5. The relative size of the components described in (3) and (4) depended on the variable under consideration; the relative importance of (4) increased with an increasing masking effect of the sleep-wake cycle in the sequence indicated above.

Component S indicates that our constant routine protocols have a residual masking effect as has been commented upon by Czeisler et al. (1989). Based on the present results, our data lead us to agree and enable us to suggest possible causes: onset of feeding and activity. These are the same as those that apply under nychthemeral conditions but, of course, they are much reduced in effect during constant routines. We do not believe that a residual masking effect negates the value of our protocol, due to the method of mathematical analysis that we use. It is a method that compares the data

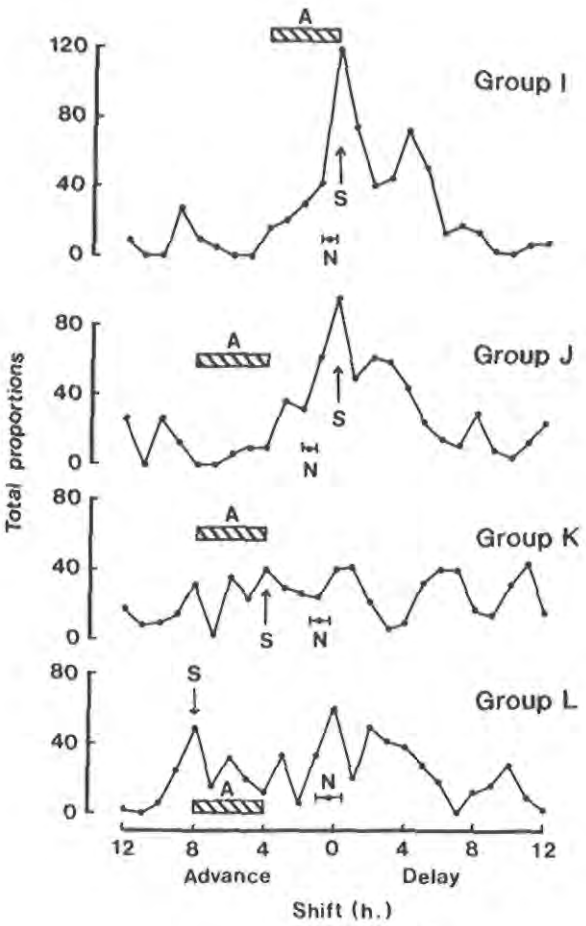

FIGURE 7.7. Data presented as described in the legend to Figure 7.6 for Groups I–L. The hatched bars labeled A indicate phase-shifts expected if the phase-shift was due to a shift in sleep times during the anchor sleep; S, phase-shift expected from a direct effect of the waking time at the start of the two constant routines (for further details, see Table 7.2). N, mean shift of acrophase (±1 SE of mean) between control days and last 96 hr when sleeps were being taken. Data of Minors and Waterhouse (1983).

sets in terms of *two* components. In effect, we are comparing two constant routine data sets in terms of the shifts due to component S *as well as the endogenous component.* This endogenous component appears to be phase-delayed by an amount comparable to that found in Groups B and C (see Figure 7.2).

Even though the results show considerable irregularity, a component shifted by an amount comparable to that found nychthemerally was often

present also. It seems possible, therefore, that there are three components to deal with: not only those due to factor S and a free-running component but also one (partially adjusted by the anchor sleep) that is observed nychthemerally, N, as well as during the constant routines. Any cross-correlation analysis would enable two of the components to be measured (presumably the two strongest, which will depend upon the subject, variable, and details of the protocol), and all components would appear in a frequency distribution assembled from several subject-variables (Figures 7.6 and 7.7, for example).

Phase 3: A Two-Component Cross-Correlation Method for Data Analysis

The problem with constant routines is that sleep loss and the general restriction of normal activities preclude their frequent use. Cosinor analysis can be performed on data collected under nychthemeral conditions even though the result might be affected by masking. In an attempt to combine the advantages of both of these methods, in collaboration with S. Folkard (Sheffield) we have developed a simple technique that can be applied to body temperature data and that enables us to estimate the phase of the endogenous component of a circadian rhythm measured under nychthemeral circumstances.

The method, which has been described more fully elsewhere (Minors and Waterhouse, 1988; Folkard et al., 1991), is based upon the assumption that a rhythm can be described as the sum of endogenous and exogenous components. In outline it involves the following stages:

1. Values for the endogenous component are taken from published literature (Aschoff and Wever, 1980). They are results obtained under conditions of constant bed rest and modified where necessary to take into account the effect of sleep (see Folkard, 1989). (They are very similar to results obtained by us during control constant routines.)
2. The exogenous component either (a) can be taken from the literature [by comparing the endogenous rhythm with that found under normal, nychthemeral conditions —see Aschoff and Wever (1980), above]; or (b) can be calculated for each individual by subtracting the endogenous component from the average of the control nychthemeral data.
3. When the experimental days have the same sleep-wake pattern as the control days [as after a time-zone transition (Folkard et al., 1990)], then the exogenous component can simply be shifted by the appropriate number of hours. In the present experiments, the sleep-wake pattern is altered and so a different procedure is required. The average exogenous effect over all waking (activity) hours and the average effect for all sleeping hours are calculated and it is these values that can be used when the sleep-wake

pattern for any experimental day is known. We also assume that the exogenous effect takes 1 hr to develop. Thus, for a subject sleeping from 2000-2400 and 0400-0800, the hourly exogenous components from 2400-1000 would be:

S, A, A, A, A, S, S, S, S, A, A

where S is the exogenous effect appropriate for sleep; and A is the exogenous effect appropriate for waking.

4. The exogenous component is added to the endogenous component, this latter being shifted in hourly increments, 0-23. Each combination is then compared with the experimental data and the highest correlation coefficient is taken to represent the shift of the endogenous component between the control and experimental days. (In many respects this analysis is the same as that of the constant routine data.)

Both methods described in 2a and 2b above have been used by us to estimate the exogenous component. The shifts of the endogenous component calculated by using the two methods were almost identical; therefore, the average of the shifts calculated by the two methods has been used in the following reanalysis of the results obtained from Groups A-L on successive days during the experimental phases.

There were no significant shifts in the endogenous component during the experiments for Groups A or H. Subjects from Groups B and C showed a progressive delay that could be used to estimate the period of the endogenous component. It was 24.48 hr ± 0.17 *(n* = 9), and compares closely with the cosinor result (see above: Phase 1: The "Anchor Sleep" Experiments).

When the subjects who had taken anchor sleeps were considered, individual results similar to those shown in Figures 7.3 and 7.4 were present. That is, a stability of phasing of the (endogenous component of the) temperature rhythm appeared to be achieved. For Groups F and G such stability was with a substantially delayed phase and after the passage of a few days (as with Figure 7.4). For each subject, the average shift of the endogenous component over the last 4 days was calculated and the mean values are shown in Table 7.3. Table 7.3 also shows, when appropriate, the paired differences between the shift of the endogenous component measured on the last nychthemeral experimental day and the shift assessed from constant routines. Inspection of the standard errors of the mean shifts during the last 4 days indicates that the variance about the mean (SE^2 x *ri)* is largest for Groups J-L; the considerable irregularity in the results from these groups has been mentioned already in connection with the constant routine results and the mean acrophase shift, N, measured nychthemerally (see Figure 7.7). More information on this is given in Figure 7.8 in which the distribution of shifts of the endogenous component (each assessed as the average of the last 4 experimental days) is compared for Groups D and I (Figure 7.8A) and Groups J-L (Figure 7.8B). The mean ± SE of these averages (see Table 7.3) is included also. Whereas the results from Groups

TABLE 7.3. Shift of endogenous component in anchor sleep experiments as assessed by the two-component cross-correlation model and constant routines

Group(s)	Anchor sleep	Mean shift (last 4 days)[a]	Difference from constant routines[b]
J-L	2000–2400	−1.32 ± 0.68 (20)	0.72 ± 0.78 (18)
I	2400–0400	−0.04 ± 0.61 (8)	2.17 ± 1.08 (8)*
D	2400–0400	−1.36 ± 0.30 (7)	
D + I	2400–0400	−0.66 ± 0.39 (15)	
E	0400–0800	−0.09 ± 0.27 (4)	
F	0800–1200	−5.38 ± 0.79 (4)	
G	1200–1600	−9.32 ± 0.42 (2)	

[a]Assessed by two-component cross-correlation model. Delays are negative.
[b]Paired differences between shift on day 8 (measured by two-component cross-correlation model) and shift assessed by constant routines.
*P = .10 that difference equals zero (Student's t tests, two-tailed).

D and I form a reasonably normal distribution, so that the mean ± SE are useful parameters, this is not so for Groups J-L. For these individuals there seems to be a distribution of values around zero shift *(less* delayed than for Groups D and I) with an extended "tail" covering delays of 2-9 hr.

The present cross-correlation method has been used with success in time-zone transition studies (Folkard et al., 1990). Thus, it gave results not significantly different from assessments made by constant routines and yet significantly different from a method such as cosinor analysis that does not correct for masking effects. The present method differs in that it uses "average" corrections to describe the exogenous effects due to sleep and waking or activity, yet it retains the basic concept of describing the rhythm as the sum of endogenous and exogenous components.

The concept of "anchored rhythms," with a delayed phase if the anchor sleep is taken at an abnormally late time —a concept that was originally found by us under nychthemeral conditions (Phase 1) and for which there was some evidence from constant routine studies (Phase 2) —is supported by this newer analysis. Also, the size of phase shift observed in Groups F and G is similar to that measured in the experiments of Phase 1.

The method has also shed light on the observation that the data from Groups J-L were rather irregular (as judged by the larger standard errors in Table 7.3 and the results in Figures 7.7 and 7.8). It appears that subjects could be divided into those that were anchored by the 2000-2400 sleeps — with phase shifts centered around zero shift — and those that were not and so showed delayed rhythms. Even though the constant routine data are irregular, they also can be interpreted in this way, with a whole range of shifts, from advances to considerable delays (inserted on Figure 7.8B), being shown. The similarity in results provided by the two-component cross-correlation model and constant routines is stressed by the fact that the

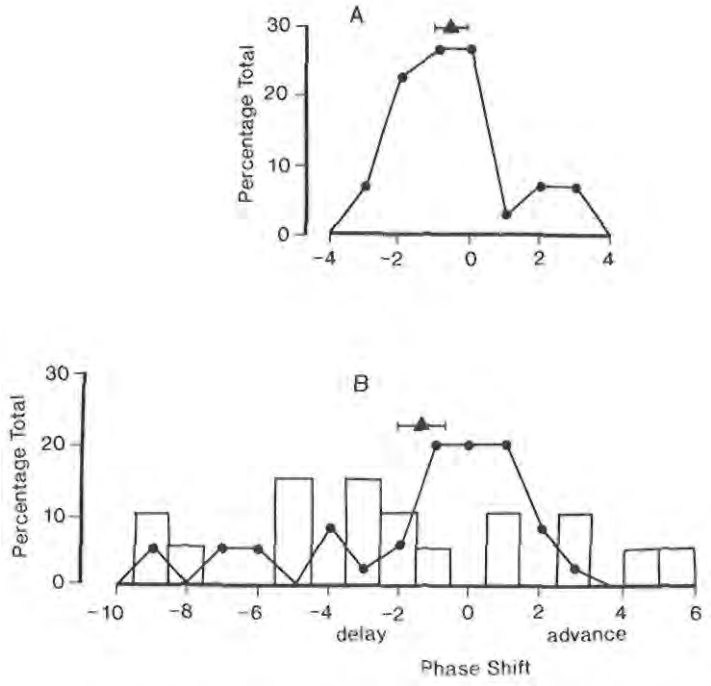

FIGURE 7.8. Distribution of shifts (calculated as the average of the last 4 nychthemeral days) as assessed by the two-component cross-correlation model. Mean ± SE of this is shown also. *(A)* Groups D and I; *(B)* Groups J–L. For these latter groups the distribution of phase-shifts assessed by constant routines is shown in histogram format.

paired differences between the constant routines and the two-component model were not significantly different. For Group I, the few constant routines that were assessed were mainly delays, in contrast to the results from cosinor analysis and the two-component model, so the paired differences between the two-component model and constant routines verged on statistical significance. Further data are required to resolve if the constant routines and two-component cross-correlation method give different results with this protocol.

General Conclusions

We consider that the analyses indicate the following conclusions with regard to our anchor sleep studies.

1. Subjects on normal sleep-wake schedules in our isolation unit show stable circadian rhythms.

2. On irregular sleep-wake schedules, rhythms show a period in excess of 24 hr. We attribute this to a lack of Zeitgebers.

3. Four hours of sleep taken at the same time each day are generally sufficient to "anchor" circadian rhythms to a 24-hr period, even when the other 4 hr are taken irregularly. The phase at which the rhythms are stabilized is related quite closely to the anchor sleep time (including the effects of inactivity, darkness, posture) rather than to mealtimes. This anchoring effect does not appear to be solely a masking effect of anchor sleeps but also involves stability of the endogenous component of the circadian rhythm.

4. The partial exception to this anchoring effect is the case of anchor sleeps taken slighly *earlier* than normal (2000-2400), when a substantial proportion of individuals appear not to be stabilized. (We assume that they would be stabilized after "losing" about 18 hr in the case of regular 2000-2400 sleeps.)

5. Constant routine studies, **but** not the two-component cross-correlation model, suggest that a nonanchored, delayed component of rhythms may be present with regular sleeps at 2400-0400. The cause of this is unknown. Further work would be required to establish if it indicated the presence of a second but unentrained oscillator; an effect of cumulative sleep loss in such a protocol; the tendency for rhythms to show ultradian components in such circumstances; or some other reason(s).

Implications of Anchor Sleeps

For shift workers, there is the problem that circadian rhythms are slow to adjust to the night shift, and the worker has often rotated to another shift before adjustment is complete (Akerstedt, 1985). For "permanent" night workers also there is a problem because, during rest days, workers tend to readjust their habits to a diurnal life-style and the loss of adjustment to night work is faster than the gaining of it in the first place. In all these circumstances, an inappropriate phasing of circadian rhythms will lead to suboptimal performance at work and greater difficulty in achieving uninterrupted sleep at home.

The present work implies that the phasing of rhythms might be maintained at a chosen timing by the use of anchor sleep. Thus, regularly taking a 4-hr sleep during the late morning —and the rest of sleep at a less regular time—would seem to be a way for night workers to maintain adjustment to night work even during rest days. We note that in some workplaces a regular short sleep is taken (officially!) during the night shift (Sudo, 1980). Not only should this alleviate fatigue but it might act as an anchor sleep (though it would stabilize rhythms so that night work would be associated with the *troughs* of most rhythms).

The continual adjustment of rhythms that accompanies those whose

hours of work are always changing is believed to contribute to the malaise experienced by some shift workers. Such a problem could be particularly marked for aircrews on duty tours involving long-distance flights since such personnel have to contend with time-zone transitions as well as irregular hours of work (Preston, 1970, 1973). Inspection of sleep logs indicates that their sleep is often fractionated and it is quite common for one of these sleeps to be taken during the hours of night (as measured on home time) if this is possible (see also Graeber et al., 1986). How effective this use of anchor sleeps is in stabilizing circadian rhythms with respect to home time is not known, but it might be an example of a "self-selection" of those individuals who can deal most effectively with the problems posed by their life-style. The potential usefulness of anchor sleeps is underlined by the fact that consideration is being given (Connell, personal communication; Graeber, 1990) to scheduling duty periods so that there is the possibility of taking anchor sleeps during irregular sleep-activity rosters.

Finally, the concept of anchor sleep has also been used to design polyphasic sleep schedules for quasi-continuous operations (e.g., emergencies, rescue operations). In such situations, nocturnal uninterrupted sleep of normal duration is hardly possible due to high demand on individuals and irregular hours of work. Stampi et al. (1990) investigated the effectiveness of schedules involving nocturnal anchor sleep at regular times (4 hr, 0100-0500) plus 4 hr of diurnal sleep divided into short naps of different durations (80, 50, and 20 min). The subjects appeared to adapt particularly well to such polyphasic sleep schedules, especially to those involving 20-min naps. Overall, performance was not significantly reduced relative to baseline levels. Anchor sleep served the function of allowing nocturnal recuperation of slow wave sleep (SWS), which was hardly present during diurnal 20-min naps. In fact, the total 24-hr amounts of SWS were virtually identical to the total SWS time in the subjects' 8-hr baseline sleep.

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8

Beyond Circadian Regulation: Ultradian Components of Sleep-Wake Cycles

PERETZ LAVIE

Abundant evidence indicates that endogenous cycles of arousal include both circadian and ultradian components (see Lavie, 1985, 1989a,b). The circadian and ultradian cycles interact with homeostatic regulatory processes to determine the tendency to fall asleep at different times of the day. Studies conducted on humans isolated in time-free environments have greatly advanced our knowledge and understanding of the circadian regulating system. These studies have led to several theories explaining the sleep-wake cycle and its interaction with the environment, which were exclusively based on circadian oscillators and homeostatic principles (Wever, 1975; Daan et al., 1984; Kronauer et al., 1982).

There is considerably less agreement regarding the importance of ultradian cycles in sleep-wake regulation. None of the models proposed so far for sleep-wake regulation have incorporated, or even taken into account, the existence of noncircadian cycles. There are several reasons for this neglect. First, there appears to be more than a single frequency of ultradian cycles in arousal. Experimental findings have shown ultradian cycles with periodicities centered at 1.5-2 hr, 3-5 hr, and possibly even around 12 hr (Broughton, 1975; Zulley and Campbell, 1985; Lavie and Scherson, 1981; Lavie, 1982). It is not yet known if these reflect independent oscillatory processes, related processes, or perhaps a single oscillatory process whose frequency is modified by other variables. In many cases the ultradian cycles have been shown to be unstable, and of relatively low amplitude, particularly when compared to the robust changes in arousal associated with the circadian sleep-wake cycle. [See Lavie (1989a,b) for further discussion.]

Furthermore, one of the problematic issues concerning the possible role of ultradian cycles in sleep-wake regulation is the relevance of the measured parameters to sleep-wake cycles. Can we say, for instance, that ultradian

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cycles in EEG alpha activity or theta activity are related to sleep propensity? Cycles in alpha activity, or any other EEG frequency for this matter, may reflect processes completely unrelated to activation or inactivation of somnogenic neural structures. They may be related, for example, to the activity of neural structures responsible for fine-tuning arousal levels during waking.

To overcome this difficulty, we investigated the possible role of ultradian cycles in sleep-wake regulation by employing an ultrashort sleep-wake cycle. With this method, subjects are required to attempt to fall asleep at frequent intervals across the 24 hr in order to construct a sleep propensity function (SPF). To avoid the confounding effect of sleep accumulation, no more than a few minutes of sleep were permitted each time.

The present chapter describes this method, and summarizes the results of a large number of studies that pointed to the importance of ultradian cycles in the control of sleep-wake regulation.

The 7/13 Sleep-Wake Paradigm

The ultrashort sleep-wake cycle paradigm was described in great detail previously (Lavie, 1986). Briefly, it requires subjects either to fall asleep or to resist sleep repeatedly at frequent intervals throughout the day. Different studies utilizing this paradigm varied from each other by the amount of prior wakefulness before the start of the procedure, and by the starting time of the ultrashort cycle. In the basic experiment, subjects came to the laboratory at 2100 after having a normal day without naps. They were fitted with electrodes to record EEG, EOG, and EMG, and spent the night asleep in the laboratory until approximately 0640; in the sleep-deprivation condition, they spent the same time period awake under supervision. In both experimental conditions, at 0700, subjects began a schedule of 7 min sleep, 13 min awake, for 24 hr. Although in our early studies (Lavie and Scherson, 1981; Lavie and Zomer, 1984) a 5/15 min sleep-wake ratio was utilized, this was later modified to 7/13 min to approximate more closely the 1:2 normal sleep-wake ratio.

Electrophysiological recordings were carried out during the 7-min sleep attempts to determine sleep stages. At the end of each 7-min *attempting-sleep* trial, whether asleep or awake, subjects were taken out of the bedroom for 13 min. In the middle of the 13 min, subjects were tested on cognitive and/or psychomotor tasks. Approximately equal-size meals of light snacks and soft drinks were available every 2 hr throughout the experimental regimen.

In most of the studies, subjects returned for a second experimental period during which the temporal structure of subjects' ability to ***resist*** *sleep* was investigated. As in the attempting-sleep condition, subjects came to the

laboratory at 2100, spent the night awake, and at 0700 began an ultradian schedule of 7 min awake in bed with eyes closed, 13 min awake outside the bedroom, for at least 24 hr. The specific instructions to the subjects were to lie in bed with eyes closed, and try to resist sleep for 7 min. Electrophysiological recordings were performed during the 7-min resisting-sleep trials as before. Here too at the end of the 7-min trials, whether asleep or awake, subjects were taken out of the bedroom, and tested on the same psychomotor tests. The order of the two experimental conditions of attempting and resisting sleep was counterbalanced across subjects.

To motivate subjects to conform to the specific experimental demands, monetary bonuses were paid to the best performing subjects in each condition.

The Sleep Propensity Function

"Gates" of Sleep after a Normal Sleep

After a night of normal sleep, sleep propensity varied in a predictable way across the 24 hr. It was low during the morning-midday period, increased somewhat during the afternoon, decreased again during the evening, and then gradually increased during the night. It should be noted, however, that in individual subjects nocturnal sleepiness did not increase in a smooth, monotonous manner, but in a series of steps superimposed on the ascending slope. These steps, however, were organized in an orderly manner. Figure 8.1 presents the averaged data of 8 subjects, tested for 48 hr under both experimental conditions after a night of normal sleep in the laboratory (Zvuloni, unpublished data). The figure shows that the ascending nocturnal slopes of the nocturnal crest in sleepiness in fact comprised discrete steps spaced approximately 2 hr apart. Figure 8.2A presents the portions of the histograms corresponding to the ascending slopes of the SPFs (10 hr, 1900-0500) during the first and second days of the study, after subtraction of the linear trends, and Figure 8.2B presents the cross-correlation functions between the attempting-sleep (AS) and resisting-sleep (RS) conditions calculated from these data. These clearly show 2-hr rhythms in sleep propensity. The 2-hr cycles were common to both testing conditions and, most interestingly, they were also in-phase as indicated by the maximum correlation at lag zero. The 2-hr rhythms were superimposed on the ascending slopes of the nocturnal increase in sleepiness. Since the same 2-hr cyclicity was found during the diurnal portion of the data, and in data for individual subjects, these strongly support the notion that the sleep propensity function (SPF) comprises both ultradian and circadian components.

The stepwise increase in nocturnal sleepiness agrees with the subjective

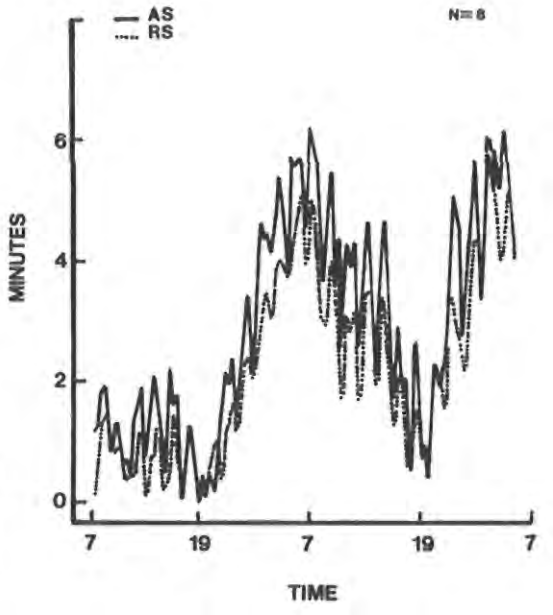

FIGURE 8.1. The mean data for total sleep of 8 subjects tested for 48 hr with the 7/13 sleep-waking paradigm under the resisting- and attempting-sleep conditions (RS, AS).

experience of evening sleep "spells" that are temporarily relieved by redundant "catnaps." It also agrees with the subjective experience of "missing the sleep gate," that is, the subjective introspection of some patients that sleep must come at a certain precise time which, if missed, leads to difficulties in initiating sleep until the subsequent gate is "open." This complaint is sometimes dramatically presented by patients suffering from phase-delay or phase-advance syndrome (Czeisler et al., 1981). These patients cannot fall asleep outside their specific individual sleep "gates" which greatly deviate with respect to the environmental Zeitgebers.

"Gates" of Sleep after Sleep Deprivation

The temporal structure of sleep propensity generally remained unchanged after sleep deprivation, although the amount of sleep per trial was greatly increased (see Figure 8.3). But instead of two sleepiness peaks, after sleep deprivation we generally find three pronounced peaks: morning, midafternoon, and nocturnal. The results of a study utilizing the ultrashort sleep-wake cycle for 48 hr (the data of which is presented in Figure 8.1) indicated that the morning peak in sleepiness seen in most of the 24-hr studies reflects the "tail" of the nocturnal crest in sleepiness. Another difference between the basal structure of sleepiness and that obtained under sleep deprivation is reflected in the shape of the nocturnal increase in sleepiness. In contrast with the gradual stepwise increase in nocturnal sleepiness under the normal sleep condition, in many cases the nocturnal

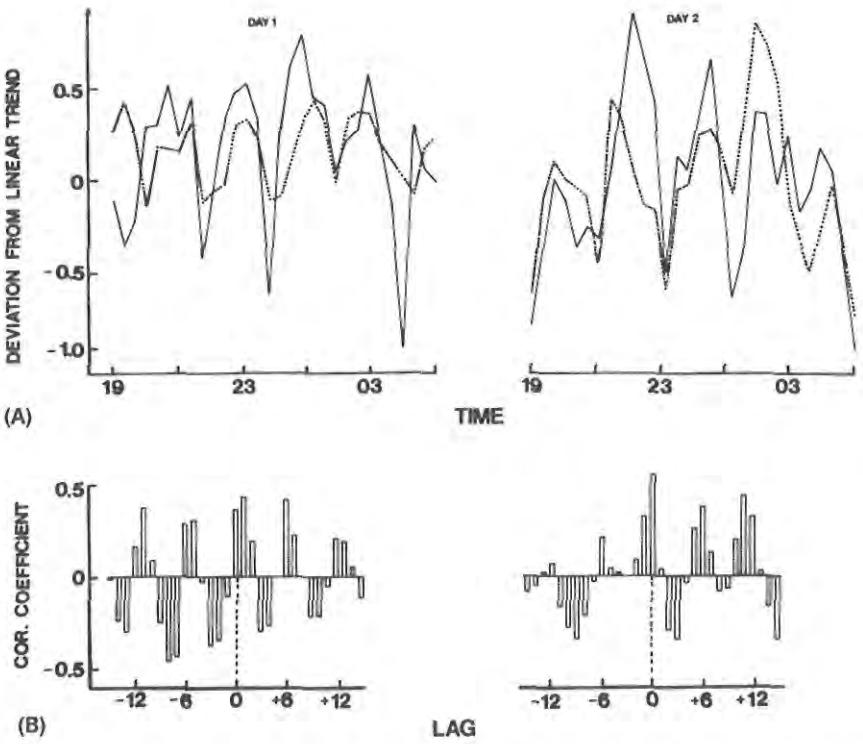

FIGURE 8.2. (A) Total sleep for the period 1900–0500 for the first and second days of the study after detrending; (B) cross-correlation functions between the detrended data of the AS and RS conditions presented in 8.2A.

increase under sleep deprivation was rather steep, appearing in some subjects as an "all-or-none" phenomenon. This is shown in Figure 8.4 which depicts the sleep histograms of the same subject, tested under the AS and RS conditions for 24 hr starting at 0700 after a 24-hr sleep deprivation. Irrespective of the experimental condition, nocturnal sleep steeply increased at 2300 and remained elevated for 4.5 hr thereafter. This sharp transition trial was termed the nocturnal sleep "gate" (Lavie, 1986) and its timing was shown to be a stable individual characteristic (see below). Comparison of sleep propensity curves of subjects tested under both sleep-deprivation and normal sleep conditions suggests that the well-defined gate under sleep deprivation coincided with the timing of the first steplike increase in sleepiness under normal sleep conditions. This is exemplified in Figure 8.5 which presents the first and second 24-hr testing periods of a subject tested for continuous 48 hr under both RS and AS conditions. While in the first 24-hr period this subject was tested after a monitored sleep in the

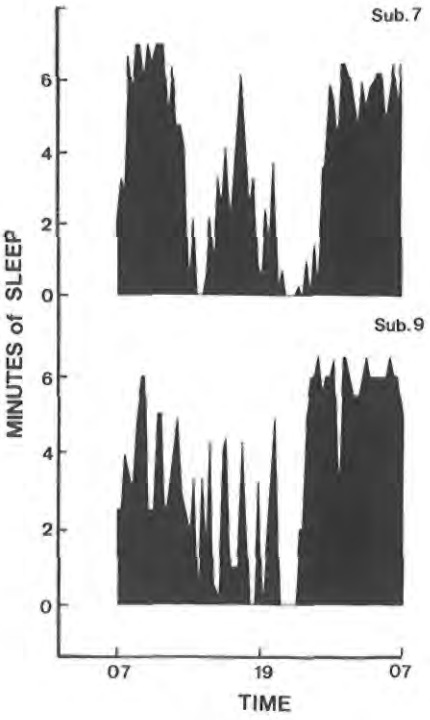

FIGURE 8.3. Total sleep for 2 subjects tested under AS conditions for 24 hr after a night of sleep deprivation. Note the sharp increase in nocturnal sleepiness in both subjects and the prominent midafternoon peak for Subject 7.

laboratory, during the second 24-hr period he accumulated a considerable amount of sleep loss. In each condition, the first and second 24-hr periods are presented one above the other to emphasize the remarkable correspondence of the nocturnal gates during the first and second days of the study.

Further results from our laboratory demonstrated that the nocturnal "sleep gates" remained stable even when 2-hr naps were interjected at different times during the 7/13 RS schedule (Lavie and Weler, 1989). In that study, 9 subjects were tested twice on the 7/13 RS paradigm, after one night of total sleep deprivation. At either 1500 or 1900, the ultradian sleep-wake cycle was interrupted and subjects were allowed uninterrupted 2-hr sleep periods. At the end of these periods, that is, at 1713 and 2113, the ultradian schedule was resumed until 0400. Subjects did not know in advance either the timing of the naps or their planned length.

Although the 2-hr naps had differential effects on postnap sleepiness levels, the mean times of the nocturnal sleep gates were very close: 2340 and 2400 for the early and late naps, respectively. The Spearman rank order correlation between the times of the gates in the two conditions was 0.68 ($p < .025$). This is the same order of magnitude as the correlation obtained between the times of the gates without naps (Lavie, 1986).

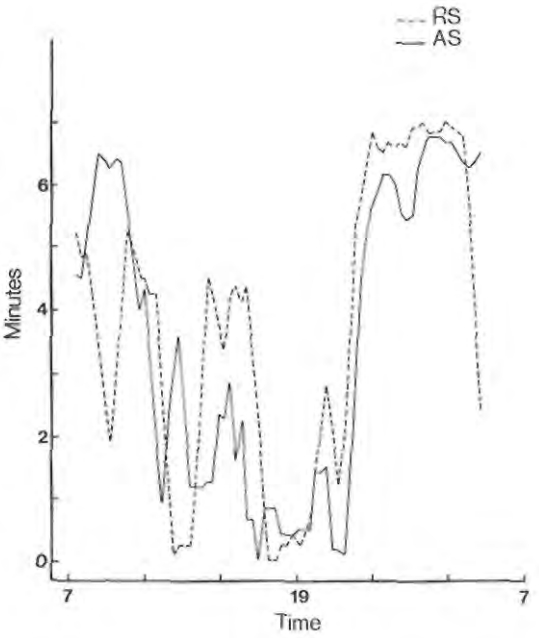

FIGURE 8.4. Total sleep of a subject tested with the 7/13 sleep-waking cycle after 24 hr of sleep deprivation under AS and RS conditions. Note the remarkable similarity in the timing of the sleep gate.

The "Forbidden Zone" for Sleep

The period just before the nocturnal sleep "gate" was characterized by a pronounced decline in the tendency to fall asleep, or, alternatively, by an impressive increase in the ability to resist sleep. The occurrence of the nadir in sleepiness just before the nocturnal sleep gate, which was also reported by Strogatz (1986) in subjects isolated from time cues, was found to be a very robust phenomenon. It was found in each of the studies employing the ultrashort sleep-wake cycle. In studies continued for 36 hr or for 48 hr, the "forbidden zone" for sleep was clearly visible during both the first and second day of the study (see Figure 8.1).

The existence of an early-evening nadir in sleepiness, and its robustness, may indicate that during that period there is spontaneous activation of arousal-related neural networks that either interferes with the process of falling asleep or greatly facilitates the ability to resist sleep. Therefore, I have termed this period the "forbidden zone" for sleep (Lavie, 1986). It also should be mentioned that, as previously discussed, Lavie and Weler (1989) reported that naps taken during that period had less immediate restorative value than naps taken during the midafternoon sleepiness peak.

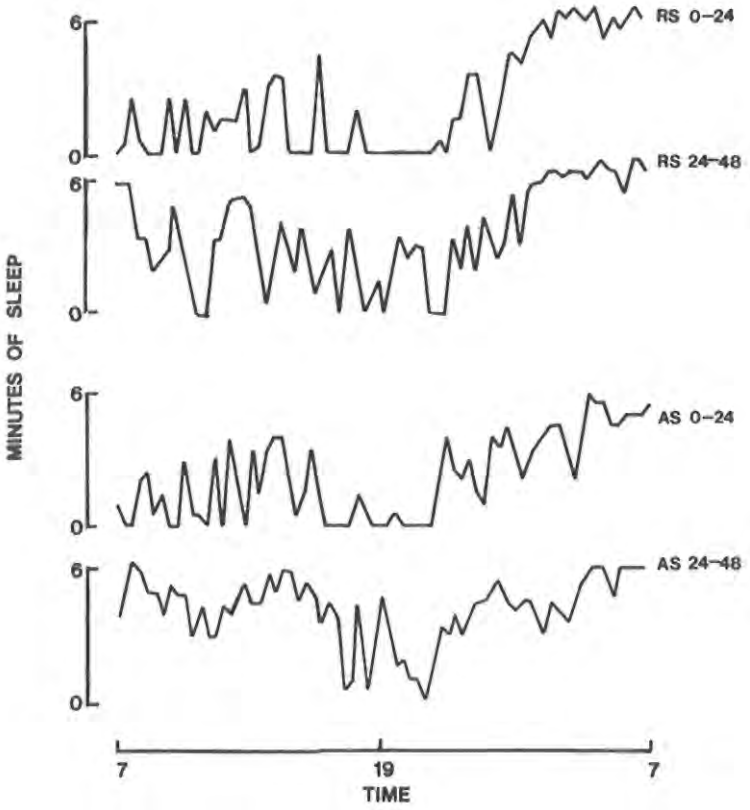

FIGURE 8.5. Total sleep for the first (0–24) and second (24–48) 24-hr periods of a subject tested for 48 consecutive hours under AS and RS conditions. Note that the sleep gate was "opened" at exactly the same time in spite of the progressive accumulation of sleep debt.

"Sleepy" and "Alert" Subjects

Generally, the terms "sleepy" and "alert" are used with pathological connotation. We refer to a person as "sleepy" when he or she complains of excessive daytime sleepiness. "Alert" patients usually complain of insomnia. The data accumulated with the 7/13 sleep-wake cycle suggest that normal persons can also be characterized as "sleepy" or "alert." There were reliable individual differences in the amounts of sleep obtained in the 7/13 paradigm. The within-subjects consistency can be appreciated from examining Figure 8.6, which depicts the sleep histograms of 2 subjects tested under the AS and RS conditions for 36 hr. To emphasize the shape of the SPFs, they were smoothed by a 3-point running median procedure (Tukey, 1977). It is evident that subjects showed an almost identical pattern of

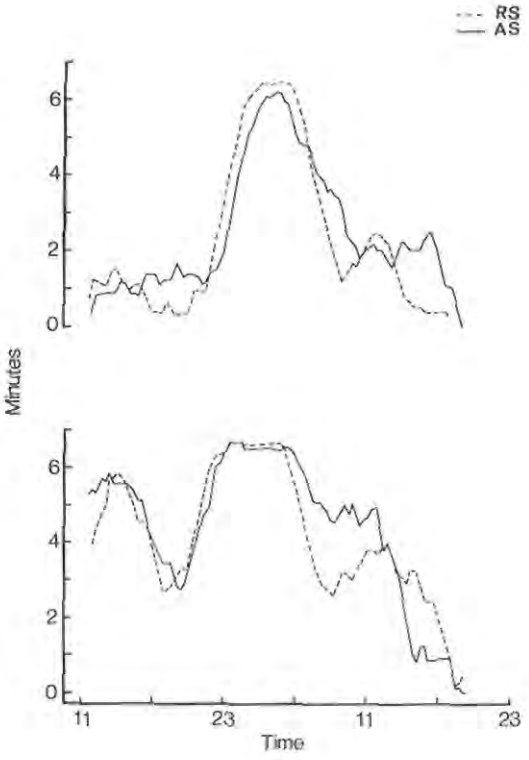

FIGURE 8.6. Total sleep of "sleepy" (lower panel) and "alert" (upper panel) subjects tested under AS and RS conditions for 36 hr after 28 hr of sleep deprivation.

sleepiness under the two conditions, both with respect to the times of the peaks and the nadirs in sleepiness, and with respect to the overall amounts of sleep.

Three types of findings support the interpretation that individual differences in the amounts of sleep reflect basic individual differences along the "sleepy-alert" continuum rather than "state"-related differences reflecting differential sleep restrictions. First, there was a significant positive correlation between the amounts of sleep obtained in the AS and RS conditions (Lavie, 1986). Second, there was a great consistency in the amounts of sleep obtained in the sleep-deprivation and in the normal sleep conditions. The Pearson product moment correlation between total sleep in subjects tested twice for 24 hr, once under AS condition without prior sleep deprivation, and then under the RS condition after sleep deprivation, was 0.65 ($p < .04$) (Lavie and Segal, 1989). Furthermore, the correlation between total sleep obtained during the first 24 hr and the second 24 hr in 8 subjects tested for 48 hr was 0.82 ($p < .01$) for the AS and 0.81 ($p < .01$) for the RS conditions. The correlation between the total amount of sleep obtained during the entire 48-hr experimental period in the two conditions was 0.73 ($p < .03$).

Third, the amount of diurnal sleep obtained in the 7/13 sleep-wake cycle was also significantly correlated with parameters of nocturnal sleep. Total sleep during the ultrashort schedule was positively correlated both with total sleep and with sleep efficiency, and negatively correlated with sleep latency during the preceding nocturnal sleep (Lavie and Segal, 1989).

Therefore, we believe that based on their obtained amounts of sleep in the ultrashort sleep-wake cycle, subjects can be divided into two somnotypes: "sleepy" and "alert." "Sleepy" subjects can easily fall asleep when instructed to do so, but also fall asleep when instructed to remain awake. "Alert" subjects, on the other hand, cannot easily fall asleep but can easily resist sleep. Preliminary results showed that "sleepy" and "alert" individuals responded differently to the benzodiazepine receptor antagonist RO 15-1788 (Lavie, 1989c), suggesting some underlying biochemical differences between the two types.

"Morning" and "Evening" Persons

Individual differences in the timing of sleep are well documented. The "morningness-eveningness" chronotypology, originally proposed by Kleitman (1963), has gained experimental support and popularity in recent years. There is general agreement that evening persons ("owls") reach their peak body temperature and diurnal efficiency 2-3 hr earlier than morning persons ("larks"), and that they have a more flexible sleep-wake cycle than their morning counterparts (Horne and Ostberg, 1977; Webb and Bonnet, 1977; Foret et al., 1982). Since none of the studies to date have investigated the 24-hr structure of arousal in the two chronotypes by electrophysiological methods, we utilized the 7/13 sleep-wake ultradian schedule to investigate the 24-hr structure of sleep propensity in morning and evening persons (Lavie and Segal, 1989).

Eleven subjects, 6 "morning" and 5 "evening" persons, were selected for the study based on their responses to a modified version of the Horne and Ostberg (1977) questionnaire.

The study included two parts: first, the 7/13 sleep-wake paradigm under the AS condition after a monitored night's sleep in the laboratory, and second, the 7/13 RS paradigm after a night of sleep deprivation.

After a night's sleep both groups revealed the typical pattern of bimodal distribution of sleepiness—midafternoon and nocturnal peaks, separated by a nadir in sleepiness. The main difference between the two groups was with respect to the rising of nocturnal sleepiness. It was lagged in the evening group by about 2 hr starting at 2140 in comparison with 1920 in the morning group. This resulted in a significant difference in the amount of total sleep between the two groups. "Morning" persons had 45% more total sleep per trial (1.45 min/trial vs. 1.03 min/trial), and a more than twofold higher amount of stage 2 (0.67 min/trial vs. 0.26 min/trial) sleep.

Under the sleep-deprivation condition, the evening group had a less-

pronounced nocturnal peak, and had no clear midafternoon sleepiness peak. There was a significant interaction between the type and time of day with respect to total sleep. Evening persons had more sleep during the morning period, particularly during the time period 1100-1400, while morning persons had more sleep during the night. Another distinct difference between the two groups was in the occurrence of sleep gates. Well-defined sleep gates, all within the range of 2120-2320 with a median time of 2220, were identified for all morning persons. In contrast, only 3 of the 5 evening subjects showed a sleep gate, all of which occurred later than the latest gate of the morning group (2400).

These results provide firm experimental support to some of the frequent claims regarding the differences between the two chronotypes which were based on subjective data. The findings that all the morning types had distinct sleep gates within a narrow time range, and that they had less sleep during the day after a night of sleep deprivation, support the suggestion that they have a more rigid sleep-wake cycle. This might cause them to be less suitable for shift work. Evening persons, on the other hand, appeared to be much more flexible in their sleep times which would be advantageous with respect to the adaptation required in rotating shift work. Thus, evening persons would have less difficulties in sleeping during the day after a night shift.

Pathological Somnolence: The Case of Narcolepsy

Diurnal sleep patterns of narcoleptic patients have been utilized by several authors as an experimental model for investigating sleep-wake regulations, particularly with respect to the operating characteristics of the REM oscillator. Some offer evidence for the existence of daytime ultradian cyclicity in the occurrence of REM episodes in narcoleptics (e.g., Schulz, 1985) (see also Billiard et al., and Schulz et al., this volume). Since this finding has important implications regarding the nature of the REM oscillator, we employed the ultrashort sleep-wake cycle in order to determine if the diurnal structure of sleep propensity in narcoleptics is different from that of normals (Lavie, 1991).

Six patients with narcolepsy-cataplexy participated in the study. Patients were studied under the RS condition from 0700 until 1900, after a nocturnal sleep in the laboratory. Experiments were conducted after a 10-day wash-out period from all antinarcoleptic medications.

As could be expected, patients found it extremely difficult to resist sleep. Total sleep averaged 3.4 min/trial, which is almost three times more sleep than in normals tested under the AS condition during the same time period (1.22 min/trial). Figure 8.7 presents the mean sleep stages histograms for the 6 narcoleptics and 8 normals. The latter attempted to fall asleep under the 7/13 paradigm during the same time period after a normal sleep in the laboratory. The two striking differences between the two histograms are (1) the elevated amounts of sleep and (2) the preponderance of REM sleep, in

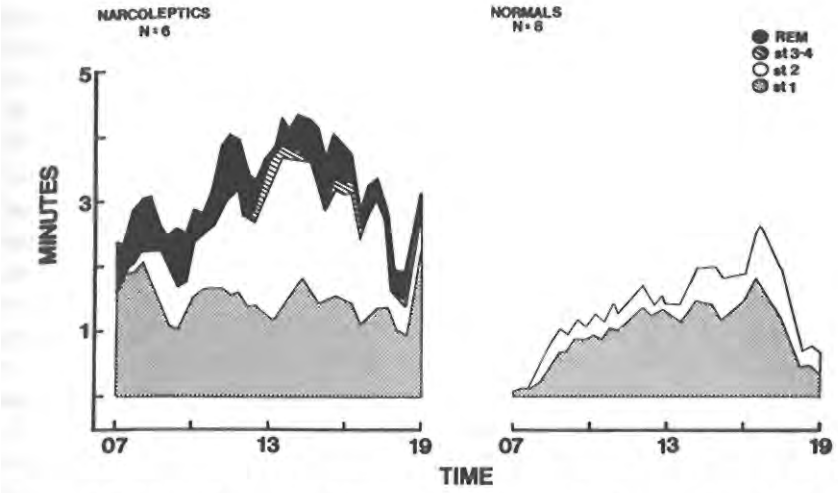

FIGURE 8.7. Mean sleep stage histograms for 6 narcoleptics tested under the RS condition, and 8 normals tested under the AS condition, from 0700 until 1900.

the narcoleptics. It is also interesting to note that the midafternoon peak in sleepiness in narcoleptics was advanced with respect to the timing of the midafternoon sleepiness peak in normals. But the most striking difference was with respect to REM sleep. REM occurred in narcoleptics in an episodic "on-off manner which in some patients was remarkably periodic. Across all patients the distribution of inter-REM intervals showed a peak at 80 min which accounted for 26% of all inter-REM intervals. The predominance of 80-min cycles was also corroborated by periodogram analyses. Previously, I showed a similar periodicity in the appearance of REM during the 7/13 sleep-wake paradigm in normals (Lavie, 1987). In variance with the results of the narcoleptic patients, normals showed ultradian periodicity in REM only during the nocturnal period, and after an obligatory accumulation of a critical amount of NREM sleep.

It can be concluded that based on the results with the 7/13 sleep-wake paradigm, the narcoleptic patients have abnormalities of both the REM and the NREM oscillators. The results firmly support the claim that in narcoleptics the REM oscillator does not cease to function during brief periods of waking. But the results also suggest that the midafternoon peak in sleepiness occurs earlier in narcoleptics than in normals.

Theoretical Implications and Conclusions

Subjective experience reveals that sleepiness at any given time is a function of the length of prior wakefulness and the time of day. At 0400, most people feel sleepy regardless of the length of prior wakefulness. Sleepiness

is considerably augmented at this time, however, if there is an accumulated sleep debt. These principles have been elegantly incorporated in a sleep-wake regulation theory by Daan et al. (1984). Combining homeostatic principles and a circadian oscillator, Daan, Beersma, and Borbely assumed that a sleep-promoting *substance S* is built up during active waking, and decreases during sleep. Sleep onset is triggered when S approaches an upper threshold, and waking occurs when S reaches a lower threshold. These thresholds, it is suggested, are controlled by a circadian oscillator. Process S was considered to be a monotonic continuous process, which was approximated by fitting a curve to the minutes of slow wave sleep or to the integrated power of the EEG, during the first REM-NREM cycle of sleep periods preceded by varying amounts of prior wakefulness.

Some aspects of the temporal structure of sleepiness revealed by the ultrashort sleep-wake cycle are incompatible with theories combining only homeostatic principles and a single circadian oscillator. The data accumulated by the 7/13 ultrashort sleep-wake schedule show that SPF during the day is not a linear or exponentially behaving process. At two distinct time periods, midafternoon and early evening, there are pronounced changes in the SPF which appear at least partially independent of prior wakefulness. Furthermore, under sleep pressure, the change from the pre-sleep-gate nadir in sleepiness to the nocturnal gate, appeared to be an abrupt, "all-or-none" phenomenon.

It should be added that other sleep-wake models cannot easily account for the present findings. After a detailed examination of all the prevailing models of sleep-wake behavior, Strogatz (1986) summarized by saying: "The existence of the nap phase, the evening wake-maintenance zone and anomalous circadian phase during entrainment, are all beyond the reach of current models" (p. 210).

Is there a need for a different sleep-wake model? While the answer to this question at this stage should be no, the present data indicate that some modifications of the prevailing models are certainly needed. Based on the present data, a third principle, that of ultradian variations, should be taken into account in addition to circadian and homeostatic regulating processes. This is certainly suggested by the stability of the temporal structure of sleepiness under all experimental conditions, and by the close correspondence between the results of the AS and RS conditions. Thus, at certain privileged circadian phases, a "clocklike" ultradian mechanism temporarily overrides the activity of homeostatic processes, and increases or decreases sleep propensity independently. This can be accomplished by an ultradian modulator of the sleep threshold.

Furthermore, it appears that besides the slow ultradian rhythms that may account for the midafternoon gate in sleepiness and the forbidden zone for sleep, fast ultradian variations on the order of 2 hr are also involved. The existence of the fast ultradian rhythms (circa 1.5-2 hr/cycle) are suggested by the steplike nocturnal increase in nocturnal sleepiness and by the findings

of 1.5-2 hr/cycle rhythms in sleepability during the day (Lavie and Scherson, 1981; Zvuloni, unpublished data). As I have discussed previously (Lavie, 1985), the fast ultradian variations in the SPF during the course of the day provide multiple transition points from wakefulness to sleep. Should the need arise, any one of these narrow sleepiness gates can facilitate the transition from wakefulness to sleep. This is analogous in some respects to one aspect of the periodic REM occurrence in sleep. Several lines of evidence suggest that REM periods can be seen as privileged "natural" end points of sleep, evolved to ensure a smooth transition from sleep to wakefulness. In isolation, free-running subjects tend to wake up from sleep preferentially from REM sleep (Weitzman et al., 1980). Similarly, subjects requested to wake up from sleep at a predetermined time without the aid of an alarm clock tended to awake from REM sleep (Lavie et al., 1979). Awakening from REM sleep was reported to be associated with higher levels of alertness and better performance of tasks attributed to the right hemisphere (Gordon et al., 1982). Since the right hemisphere is responsible for orientation in space, right-hemisphere activation upon awakening has an obvious adaptive value.

It can therefore be concluded that the fast ultradian rhythms during both sleep and wakefulness provide great flexibility to the sleep-wake regulatory system. The relatively smaller variations in sleepability during wakefulness, on the one hand, and variations in wakeability during sleep, on the other hand, provide multiple transition points along the sleep-wake continuum during which smooth and efficient transitions from one state to the other can be ensured.

Acknowledgments

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9

Adult Napping and Its Effects on Ability to Function

DAVID F. DINGES

Daytime napping by otherwise healthy human adults has been a controversial topic within sleep research, owing in part to the lack of integrated information on napping. Recently, the topic was extensively reviewed (Dinges and Broughton, 1989a) and considerable evidence offered to support the conclusion that napping can often be an adaptive option, reflecting a chronobiologically regulated sleep tendency that is amplified by sleep pressure and gated by environmental opportunity (Dinges and Broughton, 1989b). This chapter reviews work we have conducted over the past 10 years on napping patterns and their effects in healthy young adults.

Patterns of Napping Behavior

Many of the early studies of human adult sleep behavior did not mention napping. Since approximately 1970, however, nearly two dozen reports of sleep-wake behavior have included assessments of napping in over 10,000 adults in nine countries. We have reviewed these data elsewhere (Dinges, 1989) and noted the following patterns:

Although there are individual differences, on average 61% of adults nap at least once a week (across studies the mean number of naps per week was 1.78). Napping more than once within a day was extremely rare. The duration of naps observed across studies was remarkably consistent, averaging 73 min; no study found nap durations of < 15 min or > 120 min to be common. It would appear that the ultrashort sleep or catnap (<15 min) is either not reported in surveys and sleep logs, or is not commonly practiced by most persons who do nap. In general, the incidence of napping

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increased through adulthood in both siesta and nonsiesta cultures. Most studies did not record differences between males and females at any age in either the incidence or duration of naps. Daytime work demands appeared to account for the reduced levels of napping in nonsiesta cultures more so than did either reports of inability to nap or no need to nap. The percentage of persons napping frequently (i.e., 4 or more per week) was highest (60-80%) in studies of cultures near the equator that permit a daily siesta period. But not every equatorial culture allows siesta time, and among those that do, napping is not universally the most prevalent activity during siesta. Involuntary napping, which refers to falling asleep when intending to remain awake, has been observed among night-shift workers and patients with sleep disorders, but it is not known what proportion of napping in studies by otherwise healthy adults falls into this category. Postprandial sleepiness does not appear to be the cause of most napping by adults. Although field data on the detailed contexts in which naps are taken (body posture, light, and noise levels) are not available, there is evidence that naps taken on chairs and in nonoptimal environments result in more sleep fragmentation and less deep (SWS and REM) sleep and are judged to be less satisfying and less refreshing.

Individual Differences in Napping Behavior

It is often assumed that underlying the distinction between nappers and nonnappers is a difference in personality, mental health, sleep hygiene/sleep disorders, or some other facet of functioning. Yet, virtually no reliable data indicates that nappers are more likely to be pathological in any way than are nonnappers (Dinges, 1989). In a study of over 1000 college student nappers and nonnappers, for example, there was an identical proportion of subjects (11% in each group) who reported sleep complaints (Dinges et al., 1982). In short, among otherwise healthy adults, napping is neither necessarily a reflection of sleep problems nor a palliative for them. There are, however, reliable differences in napping patterns that have intriguing implications for the use of daytime nap strategies.

APPETITIVE AND REPLACEMENT NAPPING PATTERNS

Documentation of reliable individual differences in napping behavior and the factors that relate to these differences has been a major goal of our research, which accounts for most of what is known in this area. To conduct this work we have focused on college students, who display a wide range of napping behaviors.

In our early survey and interview studies of over 3000 students, we distinguished between two types of nappers among the approximately 55-60% of college students who reported napping "sometimes to often" (Evans and Orne, 1975; Evans et al., 1977; Dinges et al., 1980). Of the 55%

regular nappers, we consistently found that 75% reported napping *only when tired.* These nappers were designated as *replacement* or compensatory nappers. The remaining 25% reported napping *even when not tired,* and hence were designated *appetitive* nappers. Although these distinctions were based on attributions subjects gave for napping, long-term study of these groups revealed that the self-reports were grounded in basic behavioral differences.

Table 9.1 displays data on the nap pattern characteristics of 12 replacement and 11 appetitive nappers as obtained from a 35-day sleep log completed by subjects during a normal month of school. Subjects selected for study were healthy, without sleep complaints, and representative of their napper category. Appetitive nappers napped significantly more often than did replacement nappers, which is what would be expected if replacement nappers napped only when tired while appetitive nappers napped without regard to fatigue. The two groups did not appear to differ, however, in the timing or durations of their naps. The table presents medians and nonparametric tests, but similar results are obtained for means and parametric statistics.

Although there was a trend for appetitive nappers to take their naps somewhat earlier in the afternoon than replacement nappers, we were surprised to find that there were no reliable statistical differences in nap timing between these two groups. We had expected that replacement nappers would show more variable nap times, if they were napping whenever they felt tired, whereas appetitive nappers were expected to show a less variable schedule due to a planned daily afternoon nap. Figure 9.1 displays the temporal distribution of all logged sleep onsets and offsets on nap days for 11 appetitive and 11 replacement nappers. Naps are evident by the small (1-2 hr) phase shift between sleep onset and offset distributions in the center of the figure. Regardless of the attributions subjects make to explain why they nap, nap placement appears to be midway in the wakefulness period, onsetting 7-8 hr after nocturnal sleep offset and ending

TABLE 9.1. Napping behaviors (medians) taken from 35-day sleep logs of 11 "appetitive" (A) nappers and 12 "replacement" (R) nappers

	A	R	Mann-Whitney[a]
Total number of naps taken	181	131	
Number of naps per subject	17	11	$U = 28, p < .025$
Number of naps per week/subject	3.2	2.6	$U = 34, p < .05$
Naps (%) between 1200 and 1800	72%	72%	
Time of nap (hr)	1554	1625	$U = 34.5$, ns
Nap duration (min)	60	90	$U = 51$, ns
Time from morning awakening to nap onset (hr:min)	7:08	7:57	$U = 50$, ns

[a]Two-tailed tests.

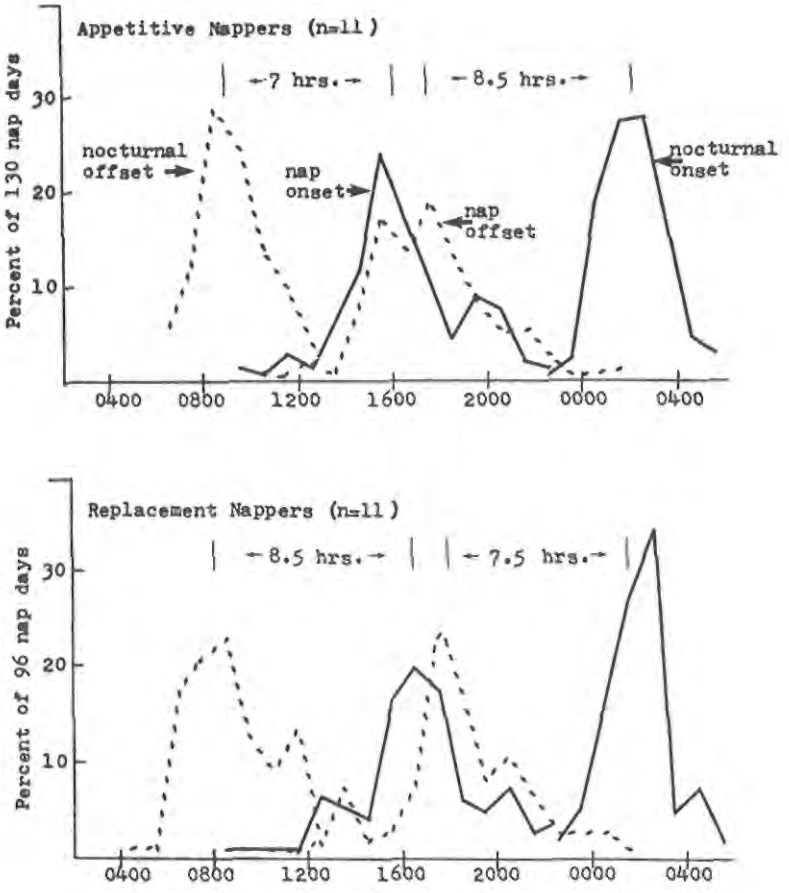

FIGURE 9.1. Frequency distributions of all sleep onset times (solid line) and sleep offset times (broken line) logged on nap days only, during a 35-day period by 11 appetitive and 11 replacement nappers. Vertical lines denote the median times of each distribution.

7.5-8.5 hr before nocturnal sleep onset. When data are combined across these two nap groups, the pre- and postnap wakefulness periods are virtually 8 hr (see Dinges, 1989, Figure 2).

It is noteworthy that the two sleep-onset zones correspond closely to the "primary and secondary sleep gates" observed by Lavie (1986) in ultradian studies of sleep-wake structure. Moreover, Figure 8.1 reveals two time zones during which sleep rarely occurred in either napper group, the first around 1200-1300 and the second around 2200-2300. These are similar to the "forbidden zones for sleep" of Lavie (1986) and the "wake-maintenance zones" of Strogatz (1986).

In an effort to determine whether the timing of the naps in midafternoon, as reflected in the sleep-log data of appetitive and replacement nappers, represented a zone of sleepiness in all healthy young adults, we surveyed 528 habitual nappers and 428 nonnappers. Among the questions asked was one that required subjects to check times (on a 24-hr scale) that they were "most alert and awake," and another that requested times they "worked most efficiently." Figure 9.2 displays the results obtained. Regardless of whether subjects habitually napped, and regardless of the category of napper, fewer than 5% of subjects selected the hours between 1530 and 1730 as times they

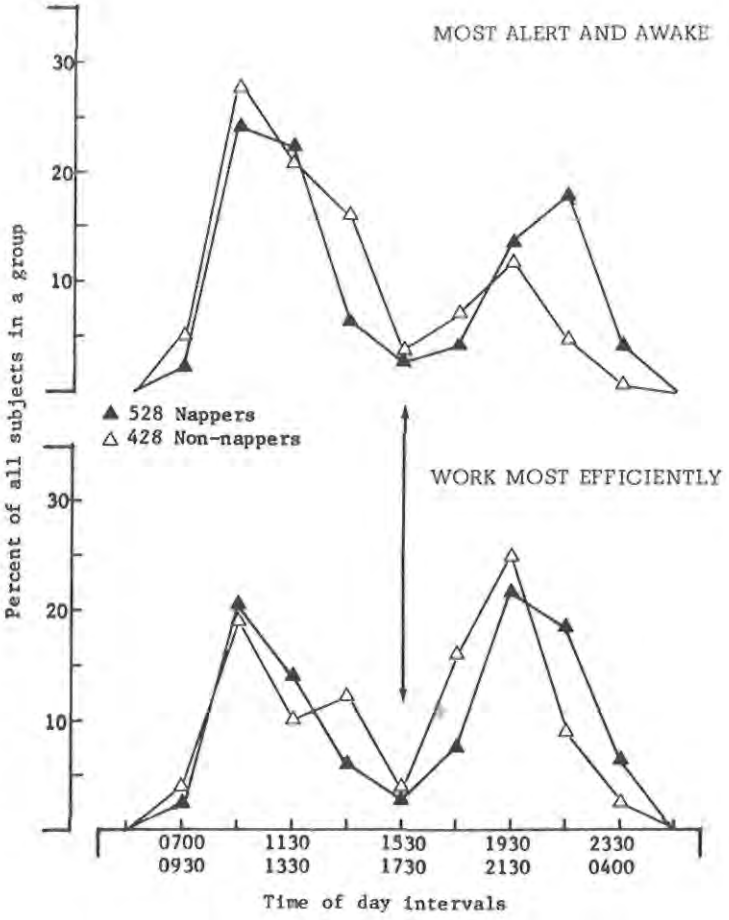

FIGURE 9.2. Proportion of survey respondents from 528 nappers and 428 nonnappers indicating the time of day they were most alert and awake and when they worked most efficiently. Responses between 0930 and 2330 were grouped into 2-hr periods. The vertical arrow identifies the period from 1530 to 1730 as the daytime period least selected by subjects.

were alert or worked most efficiently. Mornings and evenings were clearly preferred to midafternoon. Survey data gathered in other studies of students, on times of greatest daytime sleepiness (without mention of napping), conform fully to this picture (e.g., Dinges, 1989, Figure 3).

It would appear that daytime napping by college students in the midafternoon is more or less temporally bound to a "nap" phase in the circadian cycle, as Broughton (1975) originally suggested. If midsleep is used as a marker, naps occurred nearly 180° (24 hr = 360°) after nocturnal sleep and, together with night sleep, formed a biphasic sleep-wake cycle in both appetitive and replacement nappers. This midafternoon zone of sleepiness was also reflected in the times identified by students as when they were least alert and least efficient at work—even students who did not nap identified the midafternoon zone as one of sleepiness. We believed that the basis of this pronounced nap-zone sleepiness in college students resided in their sleep needs. We therefore set about determining whether there were different sleep needs in appetitive and replacement nappers.

SLEEP NEED AND APPETITIVE VERSUS REPLACEMENT NAPPING

Replacement nappers were thought to nap due to increased sleep need (only when tired), whereas appetitive nappers were thought to nap for reasons unrelated to sleep need (even when not tired)—for example, as a result of a biphasic sleep cycle. To evaluate this issue, we compared nocturnal sleep amounts reported in sleep logs for nights prior to nap days and nights prior to nonnap days. In addition, we asked subjects to indicate how much sleep they felt they needed, and we calculated from sleep logs the total sleep per 24-hr obtained on nap days. Figure 9.3 presents the results of these comparisons for replacement nappers, while Figure 9.4 presents comparable data for appetitive nappers.

Appetitive nappers not only napped more than replacement nappers (Table 9.1), but they also averaged significantly more nocturnal sleep on nights before nap days than replacement nappers and a higher estimated sleep need. They did not differ from replacement nappers in the amount of sleep reported on nights before nonnap days. Thus, appetitive nappers averaged the same amount of sleep on nights prior to nap and nonnap days, while replacement nappers consistently averaged less sleep on nights prior to nap days relative to nights prior to nonnap days ($p < .001$).

Figure 9.3 shows that replacement nappers sleep least on nights prior to nap days—an observation that lends credence to their claim of napping "only when tired." Their sleep amounts on nights before nonnap days and total sleep time (TST) per 24 hr on nap days are both comparable to their estimated sleep need. Since the average nocturnal sleep amount is just over 6 hr on nights before nap days, it is reasonable to conclude that replacement nappers nap in response to a sleep "debt" that results from their periodically obtaining too little nocturnal sleep.

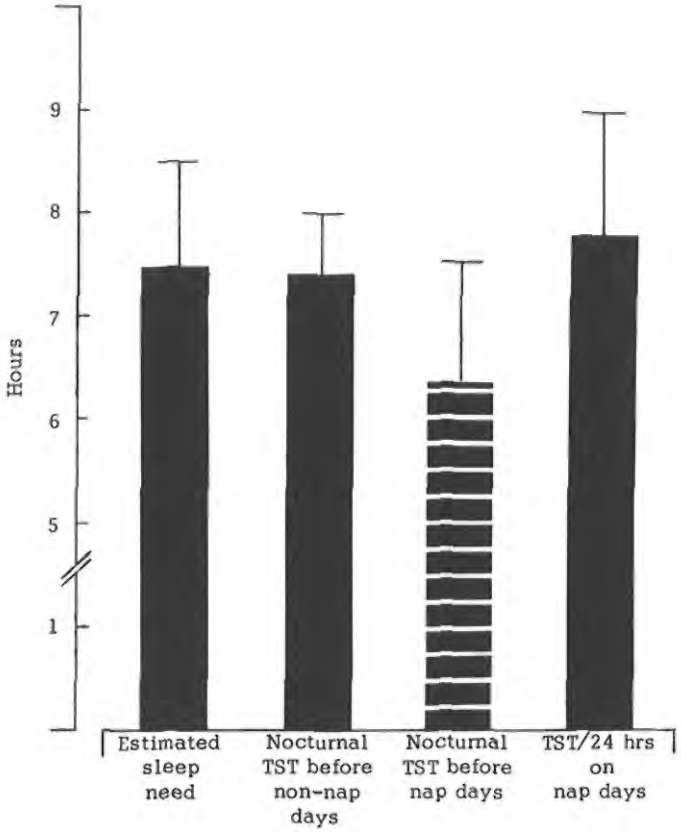

FIGURE 9.3. Histograms of mean (SD) values for (1) estimated sleep need (obtained by survey), (2) nocturnal sleep amounts reported in sleep logs for nights prior to nap days and (3) nights prior to nonnap days, and (4) the total sleep time per 24-hr period (calculated by adding nap sleep to nocturnal sleep the night before a nap day). Data are from 11 replacement nappers. Interrupted histograms are significantly different (at least $p < .05$) from solid histograms by paired comparisons.

Appetitive nappers, on the other hand, appear only to achieve their estimated sleep need on days they nap (TST/24 hr), consistently falling short of it with nocturnal sleep (see Figure 9.4). Does this suggest that they need more sleep and hence nap to satisfy this need, or simply that they have a skill at being able to sleep? Certainly, appetitive nappers appear to be adept at getting sleep; they not only nap frequently without shortened sleep the night before, but they also report being able to fall asleep almost anywhere, and they do not have to be tired to nap. It is noteworthy that no behavioral or psychological parameter other than sleep need—not diet, sex, exercise, personality structure, or acquired skills—was associated with

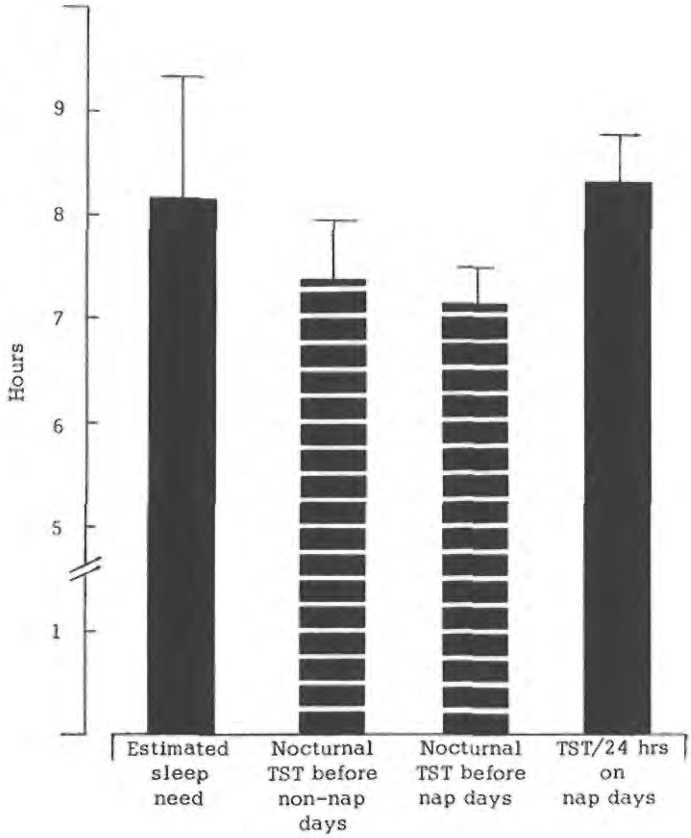

FIGURE 9.4. Data derived and compared in same manner as in Figure 9.3. These results are from 11 appetitive nappers.

napping behavior in appetitive or replacement nappers. Moreover, there was no evidence that naps disrupted nocturnal sleep in these subjects. To determine the purpose served by naps in appetitive and replacement nappers, we conducted a series of laboratory studies.

Individual Differences in Nap Sleep Infrastructure

Laboratory studies of napping conducted from 1963 to date have included polysomnographic monitoring of sleep stages, resulting in a wealth of data on sleep infrastructure of naps (Dinges, 1989). Results have been remarkably consistent. Daytime naps are not miniatures of nocturnal sleep compressed in time. Rather, nap sleep infrastructure depends on the time of day the nap is taken and the duration of the nap. The most robust finding is that REM sleep shows a pronounced circadian pattern. Latency to REM

sleep is shorter and REM sleep amount is greater when naps are taken in the morning between 0800 and 1200 relative to the afternoon or evening hours (Broughton, 1989; Dinges, 1989). Assuming comparable nap durations, afternoon and evening naps have more SWS than do morning naps, with evening naps having the most SWS and the least REM sleep. Afternoon naps have sleep infrastructures unlike both the beginning and end portions of nocturnal sleep.

Beyond these generalizations, only a few studies have investigated the role of individual differences in nap sleep infrastructure. In a seminal study from our laboratory, Evans et al. (1977) reported that during afternoon naps appetitive nappers had significantly higher amounts of stage 1 sleep than either replacement nappers or nonnappers (actually the nonnappers represented a special group of nonnappers who reported that naps produced unpleasant aftereffects — they were only 4% of all subjects). The increased stage 1 sleep in appetitive nappers seemed consistent with the view that their high proclivity for napping was not in response to sleep need as much as it was a tendency to sleep for psychological reasons. Thus, Evans et al. (1977) concluded that napping seemed to serve different functions for appetitive and replacement nappers.

In a replication and extension of the work by Evans et al. (1977) we (Dinges et al., 1980) confirmed that appetitive nappers averaged more stage 1 sleep (minutes and %), as well as more stage changes, than the special group of nonnappers. However, in the replication, replacement nappers were not different from appetitive nappers in this regard. A careful evaluation of the data from both studies revealed that it was the special group of nonnappers who had unusually consolidated afternoon sleep. In addition, these nonnappers (who reported that naps produced unpleasant physical and/or mental aftereffects) were significantly ($p < .05$) more likely than nappers to be bothered by background sounds when attempting to concentrate, to have difficulty falling asleep in an alerting environment, to drink coffee, and to have a blood relative with insomnia (Dinges et al., 1980). Thus, they appeared to be a subgroup of subjects who have difficulty regulating their daytime sleep-wake activity.

Another major study we conducted on nap sleep infrastructure involved the study of these three groups of subjects while attempting to take afternoon naps in sleep-conducive versus alerting environments (Dinges et al., 1980, 1981). The issue of nap sleep quantity and quality in alerting environments bears directly on the usefulness of naps in sustained work scenarios, where often the napping environment is not particularly sleep-conducive. Consistent with an earlier Japanese study (Ichihara et al., 1979), we observed significant sleep fragmentation and diminished self-reported satisfaction with naps taken on semireclining chairs in lighted rooms, suggesting that naps composed of increased light sleep stages are not judged to be better than those with deep consolidated sleep. Although a nonoptimal environment reduced positive feelings about the naps, nappers (but

not nonnappers) continued to report subjective benefits from naps, even in the alerting environment. In addition, we were surprised to find that virtually all nappers, and even most of the special group of nonnappers, could sleep in the alerting environment.

Control of Napping: Evidence for a Preparatory Response

In our early work on napper differences (Evans and Orne, 1975), there was a tendency for nappers to have lower oral temperatures than nonnappers at electrode hookup immediately prior to laboratory naps. We wondered whether such a difference could be a physiological manifestation of an ability to engage in daytime sleep, or a preparatory response to a daytime nap. To determine the reliability of this difference and its specificity to daytime nap periods, we repeatedly recorded oral temperature before and after laboratory naps and wake control periods in the afternoon to determine at which point in time this difference emerged.

Examination of prenap sublingual temperatures revealed that the special group of nonnappers (those who reported naps produced unpleasant physical or mental aftereffects) averaged significantly higher temperatures prior to nap onsets than did either replacement or appetitive nappers (see Dinges et al., 1980, Figure 4). In addition, although nonnappers did not differ in sublingual temperature between nap and nonnap days, nappers had significantly *lower* prenap oral temperatures on lab nap days relative to lab nonnap (wake control) days.

Figure 9.5 displays the oral temperature results for readings made approximately 1 hr before an afternoon laboratory nap. Analysis of variance revealed that the main effect for days (nap vs. wake control) was statistically significant for both replacement and appetitive napper groups, but not for nonnappers ($p > .2$). Thus, nappers averaged significantly lower body temperatures before the nap periods than before wake control periods. At 1 hr prenap (precontrol period) the effect was larger for replacement nappers than for appetitive nappers, but at the second temperature reading (5 min prenap and 5 min prewake control period) the effect was greater for appetitive nappers. These prenap temperature differences were accompanied by increases in self-reported sleepiness and calmness on nap days relative to wake control days (Dinges et al., 1980). It was as though nappers were psychologically and physiologically preparing themselves to nap by lowering their activation.

Although more research is needed on the meaning and basis of these preparatory responses and their relationship to the infrastructure and effects of naps, our data suggest that an ability to prepare for a daytime nap by lowering activation may be a central component of being able to control nap sleep onset at a variety of times and in varying environments. We suspect that the vast majority of adults, including most nonnappers, possess this capacity, but do not use it often enough (i.e., nap) to make it seem

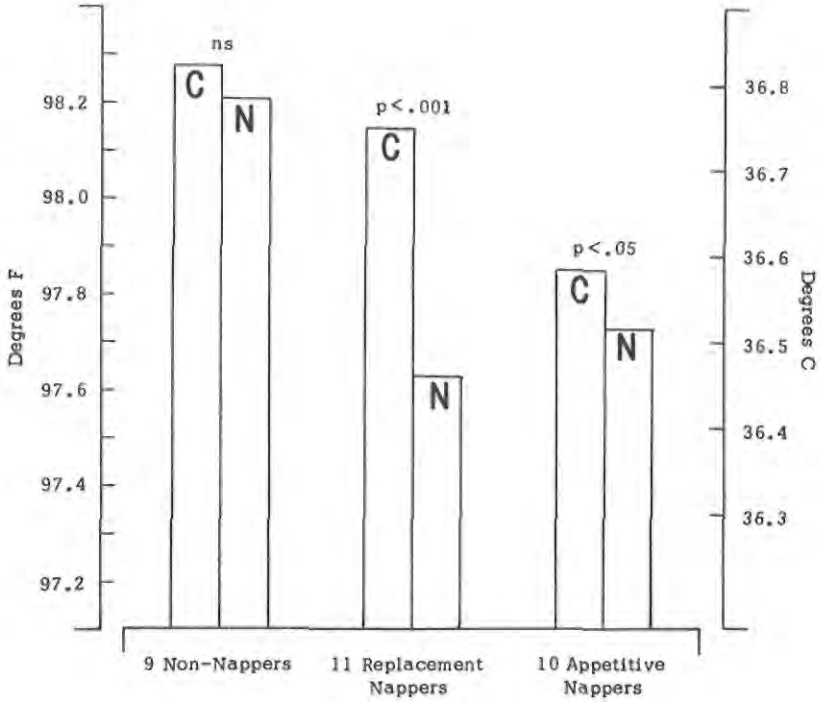

FIGURE 9.5. Histograms of mean sublingual temperature for the first recording made on two wake control (no-nap) laboratory days (C) and two laboratory nap days (N) for 9 nonnappers who reported naps produced unpleasant aftereffects, for 11 replacement nappers, and for 10 appetitive nappers. In all cases the recordings were taken approximately 1 hr prior to the 60-min wake control or nap periods.

voluntary. The timing of naps and their often compensatory nature suggest that an impetus, such as being in a circadian zone of sleepiness and/or increased sleep pressure brought on by reduced nocturnal sleep, may provide most individuals with the psychological justification and biological pressure toward decreased activation that appear to be essential for a nap to be taken voluntarily.

Finally, in terms of the appetitive versus replacement napper typologies we have used, it is important to note that appetitive nappers can and do replacement nap, and most nonnappers can nap if time is available or if they feel the need (Dinges et al., 1980). Consequently, our napper typologies probably do not represent stable personality traits as much as behavioral strategies along a continuum of control over daytime sleep potential. It would seem to be more appropriate, therefore, to speak of replacement and appetitive *napping,* rather than nappers.

Effects of Daytime Naps

In recent years, with the advent of sleep disorders medicine, napping has been regarded in a negative light for two reasons: it can be a sign of a disorder of excessive sleepiness (e.g., persons with sleep apnea or narcolepsy); and it can be a sign of poor sleep hygiene (e.g., disturbing subsequent nocturnal sleep). While both perspectives have merit, they are largely irrelevant issues when considering naps in otherwise healthy human adults. The second concern, in particular, is frequently misused to discourage persons from taking daytime naps. But there is no strong empirical support for proscribing against daytime napping based on the effects it has on mood, performance, and subsequent nocturnal sleep.

In a recent review of the effects of naps in healthy adults, we observed (Dinges, 1989) that "Data on the effects of naps reveal that they are neither consistently beneficial nor detrimental. Rather, both positive and negative effects can result, depending upon such factors as the extent to which the subject habitually naps, the nap duration, time of day of napping, and abruptness of awakening" (p. 193). The most frequently cited reason for why naps should not be taken concerns their putative effects on subsequent nocturnal sleep. There is, however, no evidence that naps adversely effect nocturnal sleep in all or even most persons who nap (Dinges, 1989). As noted earlier, neither survey studies nor extensive daily sleep-log data have provided any evidence that daytime napping is more often associated with nocturnal sleep difficulties and complaints than is nonnapping. Thus, prohibitions against daytime napping because of its alleged adverse effects on nocturnal sleep are not supported by data from healthy sleepers. Far more important are issues of nap effects on mood and performance. In this domain, there are two concerns: performance immediately upon awakening from a nap (i.e., during the period of sleep inertia), and performance and mood during the period of wakefulness subsequent to a nap.

Nap Sleep Inertia

"Sleep inertia" refers to the effects of sleep on performance and mood immediately upon awakening from sleep. During awakening from sleep, especially abrupt awakening from deep sleep, there usually is a period of transient but very profound confusion, disorientation, and impairment of performance—an impairment that has been documented for a wide array of behavioral tasks and that can be more severe than the impairment produced by sleep deprivation (Dinges, 1990).

In some of the only laboratory studies conducted of *nap* sleep inertia and the factors that produce it, we have shown that it occurs at awakening from afternoon naps in non-sleep-deprived healthy adults (Dinges et al., 1981). Its severity is greatly increased when the naps are taken by subjects who are

sleep-deprived (Dinges, 1990). The greater the depth of nap sleep, especially the amount and intensity of NREM sleep stages, the more severe the nap sleep inertia (Dinges et al., 1985). Since sleep depth is a function of sleep pressure resulting from prior wakefulness and circadian phase, nap sleep inertia will vary in relation to these factors, as well as the duration of the nap (Dinges et al., 1981; Dinges, 1990). Interestingly, the magnitude of the performance impairment during severe nap sleep inertia is not evident in either the subject's self-ratings of sleepiness or in the electroencephalogram (Dinges, 1990).

As we concluded elsewhere (Dinges, 1989), because awakenings from daytime naps (even naps in alerting environments) result in a transient sleep inertia, napping can have adverse effects on behavior and mood. Is the dysphoria and grogginess that can accompany nap sleep inertia a major factor in the avoidance of naps by some persons? Our data would suggest otherwise, since among college adults, at least, unpleasant aftereffects are not the reason why most nonnappers indicate that they do not nap (Dinges et al., 1980). There is little evidence that sleep inertia is even the basis for the complaints of the small minority of nonnappers who report unpleasant mental and physical aftereffects from naps and whose data are presented in this chapter, since they report these consequences long after sleep inertia effects on performance have disappeared (Dinges et al., 1980). Because the duration of nap sleep inertia is usually not more than 15 min—giving way to performance and mood comparable to the prenap period—its adverse effects should not be considered a major impediment to the practice of napping by adults, unless they are engaged in activities that require maximum performance efficiency at a moment's notice.

Beneficial Effects of Naps on Mood and Performance

Nap sleep inertia gives way to mood and performance levels that match or exceed prenap levels. It is often assumed that a major reason why many persons nap is to enhance mood and/or performance, either by increasing activation after a nap, or by extending activation into the later evening to attenuate or delay the circadian decline in alertness. The latter effect, and its use in sustained operations, has been referred to by us as "prophylactic napping" (Dinges et al., 1987).

NAP EFFECTS IN NON-SLEEP-DEPRIVED SUBJECTS

Elsewhere we have reviewed the dozen or so laboratory studies on the effects of naps taken by adults on mood and performance, concluding that the results are remarkably consistent across studies (Dinges, 1989). In general, mood variables, such as self-reports of sleepiness, fatigue, and activation, have been found to improve following naps, especially if the subjects are nappers (Dinges et al., 1980) and they have not developed a

major sleep debt (Dinges et al., 1988). In some studies the positive effect of naps on alertness have been found to be only slightly greater than those observed for bed rest without sleep, while other investigations have documented significantly greater mood enhancement following naps compared both to bed rest and to control wake periods that do not involve bed rest. When increased subjective alertness has not been observed, the subjects under study were either those who typically have had adverse reactions to naps, or subjects who were undergoing (or about to undergo) nocturnal sleep restriction.

The effects of naps on performance of non-sleep-deprived subjects have also been extensively investigated and reviewed (see Dinges et al., 1980; Dinges, 1989). Taub's three investigations (Taub et al., 1976, 1978; Taub, 1982) are the most widely cited in this area, in part because they were published in seven articles (see also Taub, 1977, 1979; Taub et al., 1977; Taub and Tanguay, 1977), but they are by no means the only studies, or necessarily representative of the results from other investigators.

Taub has consistently reported performance improvements during the hour following naps in non-sleep-deprived, healthy young adults. This includes afternoon naps varying in duration from 0.5 to 2 hr (Taub et al., 1976; Taub, 1979), and naps taken in the morning and evening (Taub et al., 1978). Our studies (Dinges et al., 1980, 1981) and those of Bertelson (e.g., Daiss et al., 1986) of performance within an hour or two after naps have not observed improved performance relative to prenap performance levels and no-nap control conditions. Differences in procedures and demand characteristics among studies may account for these performance discrepancies. Whether or not performance improvements occur shortly after naps, it is important to keep in mind that performance before naps in all of these studies of non-sleep-deprived subjects was quite high, so it is not clear that significant improvement was possible beyond the prenap level. Positive effects on mood and subjective alertness/sleepiness are far easier to document following naps in non-sleep-deprived adults.

PROPHYLACTIC NAPPING AND SLEEP DEPRIVATION

Performance enhancement may not always be apparent shortly after a daytime nap, but it can be evident many hours later, especially if prolonged wakefulness follows the nap. This observation is the simplest description of *prophylactic* napping. Although prophylactic napping, a concept originally proposed by Orne (Dinges et al., 1987), was for some time not considered to be possible "because sleep could not be stored," much recent evidence now supports the usefulness of the approach. Briefly, prophylactic napping refers to the use of naps for improved activation in advance of sustained wakefulness.

A number of studies have shown enhanced performance between 1.5 and 10 hr after a nap in non-sleep-deprived subjects, relative to subjects not

permitted a nap (Gillberg, 1984; Godbout and Montplaisir, 1986), and similar positive results have been observed for using sleep latency tests (Gillberg, 1984; Carskadon and Dement, 1986). In the most extensive laboratory study of the effects of prophylactic naps in sleep-deprived adults, we also observed delayed positive effects on performance (Dinges et al., 1987, 1988). This research included five different 2-hr nap opportunities during 2.5 days without sleep. One of these opportunities involved a nap on the first afternoon, after only 6 hr of wakefulness (i.e., before 46 hr of sustained wakefulness). We observed that in subjects who took this prophylactic nap on the first afternoon there was improved reaction time on a sustained attention performance task, but it was not evident until 10 hr after the nap (i.e., during the first full day of sleep loss).

Once sleep-deprivation was present, that is, when the nap was taken after anywhere from 18-42 hr of sustained wakefulness, the positive effects of the nap on performance were evident within an hour after the nap and were sustained for between 6 and 30 hr postnap (Dinges et al., 1987). On the other hand, these naps taken during a period of sustained wakefulness had virtually no effect on mood parameters (Dinges et al., 1988), suggesting that the nap prophylactically prevented some of the performance deterioration that typically results from sustained wakefulness, but that subjects were not phenomenologically aware of this benefit. It appears therefore that afternoon naps, including those taken prophylactically before sustained wakefulness, have beneficial effects on performance and physiological sleep tendency up to 12 hr after they are taken, although the napper may not be fully aware of these benefits.

Finally, it should be noted that there is no consistent evidence that the effects of naps on mood and performance beyond the sleep inertia period are directly associated with a specific stage of sleep. In general, it appears that individual differences and factors that influence nap sleep infrastructure will both determine postnap benefits. If the nap sleep is not consolidated due, for example, to environmental disturbances, then SWS will be reduced and postnap benefits may be reduced. On the other hand, it remains uncertain to what extent particular sleep stages are necessary in order for a nap to be perceived as increasing subjective alertness and performance.

Conclusion

Naps, especially afternoon naps of approximately 1-hr duration, are a common feature of the sleep of healthy adults such as college students, who possess the flexibility in work-rest schedules that permits napping to take place. Two types of napping patterns are commonly seen. *Replacement napping* refers to naps taken in response to subjective fatigue, which

appears to have its basis in reduced nocturnal sleep. *Appetitive napping* refers to naps taken ostensibly without regard to fatigue. Although appetitive napping is less common than replacement napping, appetitive nappers nap more frequently of all groups. Regardless of the reasons subjects attribute for naps, they are consistently timed to occur in midafternoon, approximately midway in the wakefulness period, suggesting that they are regulated by an endogenously timed period of sleep facilitation in addition to being influenced by overall sleep need [total sleep time (TST)/24 hr]. Considerable evidence indicates that most persons can nap if asked to do so. Naps generally do not adversely affect nocturnal sleep. Although sleep inertia can occur upon awakening from naps, this inertia almost always gives way to beneficial effects on mood and performance. *Prophylactic napping,* which refers to naps taken in advance of sustained wakefulness, enhances performance but not necessarily subjective alertness. In general, the available data on nap patterns and nap effects in otherwise healthy adults suggests that napping is a normal, appropriate, and beneficial feature of adult sleep-wake patterns.

Acknowledgments

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III

Polyphasic and Ultrashort Sleep and Their Effects on Performance

10

The Effects of Polyphasic and Ultrashort Sleep Schedules

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Despite the considerable research conducted in the last three decades on relatively regular shift-work systems, the problems associated with unusual schedules, especially during conditions of intensive work, have received very limited attention. But such quasi-continuous work situations are becoming increasingly common in industrialized societies. Some of these extended work scenarios involve performance of essential services in industries such as health care, transport, and nuclear electrical power, while others involve high-responsibility tasks in extreme situations, such as rescue operations, space missions, and defense efforts. In some situations the demands of continuous performance cannot easily be met simply by sharing work through conventional shift scheduling. In such situations of extreme demand the usual adult human monophasic sleep pattern (one 6-8 hr sleep episode per 24 hr) can rarely be maintained, resulting in an accumulated sleep debt. This debt can cause a serious decrease in performance effectiveness, and compromise decision-making capabilities during critical operations.

To illustrate this problem, the case of space missions will be briefly examined. Unexpected high-intensity operational demands have been *the* major cause for acute problems of sleep loss and severe performance degradation in the last 30 years of space exploration (see Stampi, 1991, for a review). Serious accidents have occasionally occurred as a result of such deficits in functioning. While continued improvements in space hardware and in mission planning should reduce the immediate life-threatening risks to astronauts, the growing complexity of space missions and particularly of those involving extended stays in space suggest that emergencies will never disappear. The Space Station missions will extend staytimes in orbit to 6 mo

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or longer; Mars missions will last in excess of 2 yr. The size of the potential loss from human error will increase with larger crews.

Under emergency conditions, which could last for days or weeks, vital demands of continuous performance cannot be easily met by conventional shift-work schedules. Crews may be required to work around the clock up to the limit of their performance effectiveness, getting their proper rest only after a severe problem has been solved. Consequently, while the advantage of adopting regular, circadian-based, work-rest schedules has been clearly demonstrated for normal conditions, there will always be a need for altered sleep-wake cycles in order to deal with emergencies, and not just for those occurring in space.

Are there sleep strategies that permit an individual to function near maximal efficiency during intensive work? In this chapter, I will propose that adult humans may have the ability to adapt to polyphasic sleep schedules, and that these might represent feasible and promising solutions to maintain high levels of efficiency during continuous work or emergency situations. Findings that strongly suggest a propensity of humans for polyphasic sleep are discussed, and a detailed review of the literature on polyphasic and ultrashort sleep is presented. Before examining these aspects, a brief summary of the main issues related to sleep management during sustained tasks is presented.

Sleep Management during Continuous Work

Although continuous wakefulness has been maintained for as long as 264 hr, the detrimental effects on performance of total sleep deprivation are well known (Naitoh et al., 1971). The generally accepted upper limit for working intensively and continuously is between 2 and 3 days. In some situations the detrimental effects of sleep loss may appear within the first 24 hr of continuous work. Recently, several reports have extensively examined and discussed how sleep needs can be judiciously satisfied during continuous work (Naitoh et al., 1986; Stampi, 1989b; see also Naitoh, Chapter 13). A total of seven different types of strategies that, in theory, may enable satisfaction of a minimal sleep demand, emerged from these reports. These will be briefly summarized here.

1. *Storing sleep in advance of anticipated sleep loss.* Accumulating sleep for later use is an intriguing idea. If possible, individuals could merely prolong their habitual 8-hr sleep length by several hours before the start of a sustained work scenario. Unfortunately, no study has yet demonstrated that sleep can be voluntarily prolonged much beyond the usual 8-hr daily sleep length. Even studies of persons deprived of sleep for 8-12 days rarely report recovery sleep lengths of more than 13-15 hr. Of course, even a modest prolongation of nocturnal sleep may be of some use for extended

work of relatively short (24-36 hr) duration. But it has been reported that extending sleep beyond the habitual amount may produce unpleasant physical and mental consequences for both non-sleep-deprived (Taub and Berger, 1973) and partially sleep-deprived subjects (Herscovitch et al., 1980), as well as performance decrements similar to those experienced with similar amounts of sleep loss. However, field studies during prolonged work indicate that some individuals may be able to store sleep, although this capacity appears to be extremely rare (Stampi, 1989a). In sum, independent confirmation that a significant amount of sleep can be stored is not yet available.

2. *Enhancing the restorative value of sleep.* Sleep "efficiency" per unit time might be augmented by the use of pharmacologic or natural agents that induce or deepen sleep. Although there are well-known agents that may facilitate sleep onset or produce some benefits on sleep pathologies, no procedures that consistently reduce sleep need have thus far been documented.

3. *Extending wakefulness.* Sleep can be reduced within certain limits; similarly, wakefulness can be extended by the use of pharmacologic stimulants. However, pharmacologic agents to extend wakefulness could only be of relative utility for the short term, as the deleterious and/or rebound effects of various types of stimulants are well known.

4. *Continuous (monophasic) 5-hour sleep.* Research has demonstrated that if sleep is gradually reduced in amount, most performance can be maintained at normal levels with 60-70% of the usual amount of sleep (4.5-5.5 hr per day; e.g., Friedmann et al., 1977). Approximately 5 hr of nocturnal sleep certainly appear to be the best way to obtain minimal sleep. However, under most environments that demand sustained work, a period of 5 uninterrupted nocturnal sleep hours cannot be taken because it usually conflicts with work demands.

5. *Anchor sleep.* Minors and Waterhouse (Chapter 7) have observed that by dividing an 8-hr sleep into two 4-hr sleep periods, circadian rhythms become stabilized, as long as one of the sleep periods is anchored to a fixed time of day. Although anchor sleep may offer a degree of scheduling flexibility, and the idea is certainly worth exploring further (as will be discussed), the demands of some work schedules may not permit workers to have the luxury of sleeping for 4 unbroken hours during the fixed time period.

6. *Irregular napping.* The usual way of getting some sleep during extended work is to sleep whenever possible. There is growing evidence that when *some* sleep is permitted during sustained operations, even in the form of a single nap, an improvement in subsequent performance is often observed (e.g., Naitoh et al., 1982; Dinges et al., 1987). However, even a 1-hr nap may turn out to be too long in some sustained operations. Moreover, it is possible that irregular napping may be too disrupting to the sleep system.

7. *Polyphasic ultrashort sleep.* It has been hypothesized that by dividing a reduced amount of sleep into several ultrashort sleep periods throughout the 24 hr it would be possible to achieve considerable levels of sleep reduction (Stampi, 1989b). That is, by taking short naps at many times during the 24-hr period, the reduced waking interval between one nap and the following would allow sleep to be more efficient (see Stampi, Chapter 1, for further details on this hypothesis). Before examining the preliminary experimental evidence suggesting that it might be possible to reduce sleep duration by means of polyphasic sleep, I will briefly discuss whether adult humans may indeed adapt to polyphasic sleep schedules *without* necessarily reducing the normally available total sleep time.

Evidence of Adaptation of Adult Humans to Polyphasic Behavior

The fundamental issues and experimental evidence suggesting that humans may adapt well to polyphasic sleep (at least for limited periods) are discussed extensively in Chapter 1 and in many sections of this volume. The main issues will be summarized here:

1. Polyphasic sleep is the characteristic sleep pattern in nature: monophasic sleep is an exception reserved to the human and a handful of other mammalian species and some birds (Ball, Chapter 3).
2. Sleep fragmentation is observed in particular in certain mammals living in dangerous environments (Stampi, Chapter 1).
3. Polyphasic sleep is the typical sleep pattern during the first months of human life, during which a 3- to 4-hr cycle in sleep-wake behavior is normally displayed (Salzarulo and Fagioli, Chapter 4; Webb, Chapter 5).
4. In the elderly, nocturnal sleep tends to become more fragmented and daytime naps may become relatively frequent, even in healthy, alert individuals (Webb, Chapter 5).
5. Subjects studied in situations isolated from time cues (including "disentrained" and/or bed-rest conditions) show a marked tendency to have multiple naps in addition to the major sleep episode (Campbell, Chapter 6).
6. Studies conducted in cultures isolated from modern societies (Temiars and Ibans) show that they permanently adopt polyphasic sleep patterns (Petre Quadens, 1983; see also Stampi, Chapter 1).
7. Four-hour cycles in sleep propensity *(a* and *b)* or in SWS *(c)* have been recognized in *(a)* subjects asked to remain in constant bed rest (Nakagawa, 1980; Zulley, 1988), *(b)* following ultrashort sleep-wake paradigms (Lavie, Chapter 8), and *(c)* in the frequent sleep episodes of narcoleptic patients (Billiard et al., Chapter 15). This ultradian cycle is

superimposed on the circadian cycle and the midafternoon peak in sleep propensity (see also Dinges, Chapter 9).

8. Recent studies, focused primarily on modeling the effects of sleep apnea on performance and sleepiness, suggest that a fragmented nocturnal sleep consisting of many short sleep episodes (4 min-2.5 hr) can yield as much recuperation as much longer, continuous sleep (Bonnet, 1986; Magee et al., 1987; see also Naitoh, Chapter 13).
9. The remarkable recuperative value of brief naps, which normally occurs with or without prior sleep deprivation, has been documented in a large number of studies (Dinges, Chapter 9; Naitoh, Chapter 13; Angus et al., Chapter 14).
10. Finally, many experiments have provided direct evidence that adult humans have a surprising ability to adapt to different types —and different levels —of polyphasic sleep-wake behavior.

This chapter focuses primarily on a critical review of these studies. In analyzing the major outcome from these studies, particular attention is given to the question of whether polyphasic sleep schedules are feasible and practical strategies to maintain acceptable levels of performance. These concepts are then pushed one step further by presenting and discussing preliminary evidence that polyphasic sleep might, indeed, reduce total sleep requirements, at least in the short-to-medium term.

Studies of Adult Humans under Polyphasic and/or Ultrashort Sleep Schedules

The first study to evaluate whether "one might take his sleep in installments rather than a single stretch, as most of us do now" was conducted by R. W. Husband in 1935 (p. 792). Unfortunately, that pioneering study remained largely unnoticed and the hypotheses raised by Husband did not get much attention from investigators for the best part of the following 40 years. Interestingly, it is important to point out that —with several noticeable exceptions — renewed interest in polyphasic sleep patterns was not motivated primarily by the need to find alternative work-rest schedules for a judicious management of sleep under conditions of sustained performance. Rather, it was the discovery of REM sleep and of intrasleep cyclicity, the growing interest in the functional role of SWS, and the description of the entraining properties of "anchor" sleep on circadian rhythms, that stimulated several researchers to use polyphasic schedules as a tool for investigating the functions of sleep.

The laboratory and field studies reviewed in this chapter are divided into two groups. This is to provide a better understanding of the different scopes and designs adopted. Findings are discussed, with particular focus given to sleep structure and behavioral functioning under polyphasic schedules.

Many studies investigated the changes occurring in other functions, such as core body temperature and hormonal levels, which are briefly reported here.

The main distinction between the two groups (see Table 10.1) refers to the total amount of sleep subjects were allowed to take in a given 24-hr period, specifically, to whether designs involved sleep reduction or not. In the first group of studies, subjects were allowed a total time in bed (TBT) of 8 hr, whereas in the second group of studies available bed times ranged from as

TABLE 10.1. Studies adopting polyphasic sleep-wake schedules[a]

Reference	*N*	DURN (days)	SED (min)	WED (min)	TBT (hr)	EEG	PERF
Polyphasic Schedules without Reduction in TBT							
Alluisi and Chiles (1967)	17	15	120	240	8	N	Y
Curtis and Fogel (1972)	6	14	120	V	8	N	Y
Weitzman et al. (1974)	7	10	60	120	8	Y	N
Kelley et al., 1973; Dement et al., 1972	1	5.3	30	60	8	Y	N
Carskadon and Dement (1975,77)	10	5.3	30	60	8	Y	N
Webb and Agnew (1975)	2	6	180	360	8	Y	N
Webb and Agnew (1975)	4	14	240	480	8	Y	N
Stampi et al. (1992a)	4	14	20	40	8	Y	Y
Anchor Sleep (No Reduction in TBT)							
Hume and Mills (1977)	9	8	240	V	8	Y	N
Minors and Waterhouse (1981, 1983)	54	7–12	240	V	8	N	N
Stampi et al. (1990b)	4	4	20/240	74	8	Y	N
Stampi et al. (1990b)	4	4	50/240	160	8	Y	Y
Stampi et al. (1990b)	4	4	80/240	240	8	Y	Y
Polyphasic Schedules with Sleep Reduction							
Husband (1935)	1	30	180	180–900	6	N	Y
Hartley (1974)	12	4	80	300–480	4	N	Y
Moses et al. (1975); Lubin et al. (1976)	10	1.7	60	160	6.5	Y	Y
Carskadon and Dement (1975)	1	5.3	15	75	4	Y	N
Mullaney et al. (1983)	10	1.8	60	360	3.4	Y	Y
Haslam (1985)	6	5	60	300	4	N	Y
Stampi (1985, 1989a)(*)	99	10–49	$X = 121$	V	$X = 6.3$	N	Y
Tafti et al. (1990)(*)	1	6	15–20	240	2	Y	N
Stampi et al. (1990a)	1	19	15	225	1.5	Y	Y
Stampi and Davis, 1991	1	48	30	210	3	Y	Y
Stampi et al. (1992b)	1	19	180	1260	3	Y	Y

[a]Key to abbreviations and symbols: *N*, number of subjects; DURN, experiment duration (days); *SED*, sleep episode duration (min); *WED*, wake episode duration (min); *TBT*, total scheduled time in bed per 24 hr (hr); EEG, whether EEG was recorded (yes/no); PERF, whether performance was measured (yes/no); V, variable duration; *X*, mean; (*), field studies.

short a period as 1.5 hr up to 6.5 hr per day. As can be inferred, in the second group of studies the focus was often, but not exclusively, on the investigation of strategies for continuous work scenarios. Within the studies that did not diminish the subjects' daily bed time (first section), a subgroup of studies is included in which subjects were allowed daily regular 4-hr periods of "anchor" sleep, plus 4 hr of sleep taken either at irregular times or divided into shorter naps.

As this review makes clear, not only the scopes but also the methodologies adopted by investigators differed considerably between studies. For example, studies that conducted polysomnographic monitoring (EEG, EOG, EMG) would normally not submit their subjects to performance testing, and vice versa, with a few exceptions. As a result, some studies provide detailed descriptions of sleep structure under polyphasic sleep, but little data about whether subjects were functioning properly during the trials. Similarly, other studies that investigate performance thoroughly have not reported any information about sleep composition.

Another important consideration concerns adaptation to the schedules adopted. That is, what is the time necessary for an adult human to adapt to a polyphasic ultrashort sleep-wake pattern, if indeed this is possible? Since the imposed sleep regimens are quite different from the habitual nocturnal, 1-per-day sleep episode, it could be expected that subjective adaptation to such unusual schedules takes a relatively long time. As a comparable example, it is known that following sleep displacement during shift-work or jet-lag studies, full entrainment to a new schedule usually requires a minimum of 3 and an average of 7 days (cf. Folkard et al., 1985). The major limitation of several polyphasic sleep studies reported here has been the short period allowed for adaptation, that is, the short duration of the experiments. Only 8 of the 23 experiments reported here lasted for 2 wks or more, while some experiments where as short as 1.5 days.

As will be discussed, studies conducted by the author and one nondocumented experience reported in Chapter 11 suggest that adaptation to certain extreme polyphasic schedules may require periods of 2 wks or longer. In other words, it appears that during the first experimental period on such unusual schedules, sleep-related parameters [e.g., total sleep time (TST)], sleep stages, as well as biological rhythms, performance, and behavioral parameters, can be disrupted or abnormal, after which they tend to stabilize. Any true comparison with baseline levels can have a functional meaning only after allowing a period necessary for adaptation (of yet-unknown duration, if possible at all). Nevertheless, even the very short studies are reported here because they provide important insight into issues concerning polyphasic behavior. Yet, these and other important limitations should be kept in mind in the interpretation of the main findings of these studies.

Polyphasic Sleep without Reduction in Total Bed Time

The studies within this group generally adopted schedules with a sleep-wake ratio of 1:2, that is, one-third of the time in a sleep-wake cycle was allowed for sleep and two-thirds of the time for wakefulness, resulting in approximately 8 hr of every 24-hr period being devoted to total bed time (see Table 10.1).

The 4/2 Work-Rest Schedule

The first study in this group was part of an extensive research program investigating the effects of different work-rest schedules on sustained performance and circadian rhythms under simulations of defense operations (Alluisi and Chiles, 1967). Within this project, a total of 17 individuals were submitted to schedules consisting of 4 hr of sustained work and 2 hr of rest for periods of 15 days. Studies included investigation on other regimens, such as 4/4 and 6/2 work-rest schedules. In the first two of the 4/2 studies, operational air force personnel showed, in general, significant decrements over days. However, 2 of the 11 subjects used in these two experiments were able to maintain high-level performance throughout the 15 days. In a subsequent study with 6 highly motivated Air Force Academy cadets, all subjects did not show any decrements in most tasks. The general reaction of the subjects suggested to the authors of the study that performance would probably fall off if the experiment were prolonged to perhaps 30 days. However, this hypothesis was not verified since after the first 15-day period, subjects were shifted to a less-demanding 4/4 schedule for the remaining 15 days.

It was concluded that whereas with proper control of selection and motivational factors, crews can work effectively for at least 2 wks (and probably longer) using a schedule of 4 hr on duty and 2 hr off, and that crews can work even more effectively for periods of at least 1 mo (and quite probably for 2 or 3 mo) using a schedule of 4 hr on duty and 4 hr off. In addition, the latter schedule would apparently require less-demanding controls of selection and motivational factors. The investigators also concluded that duty periods of 4 hr or less are preferred, and that rest periods as short as 2 hr are acceptable to most subjects. They also found that 18 hr of work per day, using a 6-work/2-rest schedule is probably too extreme a condition for periods exceeding 4 or 5 days.

A very important element that emerges from these studies is that motivation is indeed a key factor in facilitating adaptation to polyphasic schedules. As will be discussed in this chapter, strongly motivated subjects are able to achieve surprising levels of adaptation to sometimes extreme polyphasic schedules.

The Polyphasic Random-Living Schedule

The basic question that motivated this study was whether regularity of synchronizer schedules is a feature necessary for normal functioning, or if these are merely a matter of convenience and habit (Curtis and Fogel, 1972). The authors argued that if some regularity of schedule is necessary for humans, then on a sustained and completely irregular schedule loss of efficiency might be cumulative rather than transient, and interference with learned responses might sum to produce measurable deficits in cognitive or personality function. In other words, the study was designed to determine whether random-living schedules are accompanied by changes in cognitive, personality, or physiological function. The random-living schedules in which 6 subjects were engaged for 2 wk were such that each 24-hr day contained four 2-hr periods in which sleep was allowed in a quiet, darkened room and eight 2-hr periods out of bed in natural or artificial daylight, with all periods occurring in totally random sequence. Three meals per day were also served at random intervals.

This is the only study that assessed individual ability to fall asleep for relatively *short* periods at *irregular* times. According to its authors, it showed no evidence that regularity is essential to normal mental functioning. No impairment of cognitive or personality functions was detected. A clear circadian rhythm was found in subjective sleepiness: all subjects reported feeling maximum fatigue when they were awake between 0500 and 0700, regardless of how recently or how long they had slept. The results also suggested that the considerable individual differences found in subjects' ability to adjust their sleep to a random schedule may have been determined in part by personality factors. One subject reported being almost always able to fall asleep promptly and remain asleep until awakened. The report was supported by the subject's usually "cheerful and alert manner." Another subject reported considerable difficulty in adapting to the schedule, lying awake an estimated 30-40% of the time in bed (polysomnography unfortunately was not done in this study), and frequently feeling tired and irritable. The remaining subjects formed a continuum between these two extremes. Increased ability to nap at irregular times correlated with higher scores on the California Psychological Inventory scales of Intellectual Efficiency, Sense of Well-Being, Dominance, Self-Acceptance, Sociability, Achievement via Conformance, and Psychological Mindedness, and with low scores on the Femininity scale. The authors concluded that there is no evidence that manipulation of environmental and sleep schedules into grossly distorted forms over a limited time span can contribute to neurotic or psychotic developments in healthy subjects.

This study brings up a second key factor that, in addition to personal motivation, may play an important role in allowing quick and successful adaptation to polyphasic behavior: the ability to fall asleep quickly at almost any time during the 24 hr. It is obvious that, while individuals with

relatively long sleep latencies may still get a good night's sleep, they will be at a serious disadvantage under polyphasic schedules. Indeed, individuals who are able to fall asleep quickly and capable of staying asleep for most of their short-duration bed times will be advantaged when they have to nap many times per day. The related question is whether this is an ability that can be learned with "practice" and, if so, which would be the best training. Studies of prolonged polyphasic schedules undertaken in the field (solo sailors, Stampi, 1985, 1989a; see also an individual experience report, Sbragia, Chapter 11) and in laboratory-controlled conditions (Stampi et al., 1990a; Stampi and Davis, 1991), suggest that, indeed, certain individuals may learn to fall asleep quickly under these conditions.

"Days" of 90 and 180 Minutes

Findings from the next three studies using ultrashort "days" are discussed together since they show several methodological similarities, even though their goals were remarkably different. In 1974, Weitzman and colleagues from the Montefiore Medical Center, New York City, published a study in which 7 young adults were subjected to a 3-hr sleep-wake schedule for a period of 10 days. They were allowed eight 1-hr sleep times, equally spaced throughout each 24-hr period (1 hr of sleep followed by 2 hr of wakefulness). The aims were to determine whether an imposed 3-hr sleep-wake cycle "day" would significantly alter the normal temporal pattern of Cortisol and growth hormone (GH) secretion, and entrain a 3-hr sleep-wake pattern independent of the 24-hr pattern.

As the study demonstrated, the former did not occur, since the secretory episodes of Cortisol and the body temperature curves showed a persistence of the circadian periodicity for all subjects. However, the amplitude of the temperature cycle was decreased, and a 3-hr Cortisol cycle superimposed on the 24-hr curve was detected. Low plasma concentrations of Cortisol were associated with the "dark" (sleep) period and high concentrations with the first hour after "lights on." Although the mean 24-hr output of GH was not different from baseline, the sharp peak in secretion found between 2300 and 0100 in baseline was not present during the experiment. The authors concluded that the persistence of the circadian rhythms of temperature and Cortisol in spite of the attempt to disrupt these functions for 10 days demonstrates the highly resistant nature of these systems.

The other two studies with short "days" were conducted at Stanford University and used very similar designs. Kelley et al. (1973; see also Dement et al., 1972) observed 1 subject on a 90-min schedule of sleep and wakefulness for nearly 6 days. The subject was placed on a schedule alternating 60 min of wakefulness and 30 min of sleep. The original intent was to determine whether the subject could adjust to a schedule that was comparable to the NREM-REM cycle or the putative Basic Rest-Activity Cycle (BRAC, Kleitman, 1961; see also Chapters 1 and 8). Kelley et al.

found that REM sleep showed an unusual pattern, that is, it occurred in close proximity to the onset of sleep. Carskadon and Dement (1975, 1977), in a series of two studies with a total of 10 subjects, repeated the experiment to determine whether this unusual occurrence of REM sleep was replicable.

A clear-cut 24-hr distribution of sleep was apparent in all three studies (Kelley et al., 1973; Weitzman et al., 1974; Carskadon and Dement 1975, 1977), with maximum sleep times occurring in the late morning (0600-1300). Neither study explained this apparent shift from baseline (i.e., 2300-0700) in the circadian propensity to sleep. Sleep efficiency [calculated as the sum of stages 2, 3, 4, and REM sleep divided by total time in bed (TST/TBT)] was generally maximal when the temperature was lowest, but overall TST was reduced compared with baseline. Sleep efficiency was around 50% in all studies. Interestingly, in spite of the fact that the subjects were experiencing a considerable sleep reduction, only a small amount of sleep was achieved between 1900 and 0100 hours. These were the first descriptions of this evening "forbidden zone" for sleep, subsequently observed in many studies (see Chapters 1 and 8).

As could be anticipated from the higher number of sleep onset opportunities, overall stage 1 *amounts* were increased relative to baseline; conversely, amounts of all other stages were significantly reduced, with greatest decrements observed for REM sleep. Yet, stage *percentages* were significantly different from baseline only for stages 1 (which increased) and REM (which decreased). Quite interestingly, though, these effects were more severe at the beginning of the study, and tended to disappear toward the end, as clearly shown in the study of longer duration (Weitzman et al., 1974). During the last 3 days of this 10-day polyphasic sleep schedule, the average percents of all sleep stages were identical to the baseline condition, in spite of the major reduction of total sleep. It could be hypothesized that if longer periods were allowed on such polyphasic schedules (e.g., 3 wk or more) the sleep-stage percentage measures (and probably also sleep efficiency) might gradually become similar to those of baseline nocturnal sleep. This is indeed what occurred during subsequent studies (Stampi et al., 1990a; Stampi and Davis, 1991) investigating the effects of prolonged polyphasic schedules (19 and 48 days, respectively). These findings, which will be discussed in more detail in the next section of this chapter and in Chapter 12, challenge the assumption that mainly SWS may have some sort of prominent role in the sleep system (e.g., SWS as the major component of "core" sleep; see Horne, 1988). The latter was hypothesized after observations that many studies involving partial sleep deprivation show reductions in all but SWS stage amounts. This may be true in the short term, but in long-term experiments it appears that *all* sleep stages (including the often-neglected stage 2) have equal probability of occurrence.

In the "short-day" studies, sleep stages did not progress in the usual (nocturnal) manner when REM sleep was present. If results are combined across studies, REM sleep occurred in approximately 30% of all naps (26%

of 910 naps in the Stanford studies, and 35% of 560 naps in the Montefiore study). When REM sleep was present, it frequently occurred in close proximity to nap onset [sleep onset REM periods (SOREMPs)]. For example, in the Stanford studies, mean REM latencies were of 8 min, against an average of 45 min found in baseline recordings. These studies confirmed that SOREMPs can be easily induced in normal subjects, and they were the first to raise doubts about the significance of the diagnostic nap recording of a SOREMP in narcolepsy-cataplexy. As Carskadon and Dement (1975) point out, at the very least the apparent ease of producing SOREMPs in normals would indicate that careful attention must be given to the whole day's schedule and the time of the diagnostic nap.

REM sleep *amount* revealed a clear circadian rhythm, such that most of it (e.g., 74% in the Stanford study) occurred in the rising phase of the body temperature curve (i.e., 0200-1000). However, REM sleep *onset* had no consistent relationship to time of day. Rather, it appeared to be related to the amount of sleep time since the last REM onset.

REM sleep and SWS appeared to be mutually exclusive, that is, they rarely occurred together. In the Stanford study, REM and SWS coexisted in only 27 (3%) of 910 naps, and of these, the normal relationship of SWS giving way to REM occurred only 8 times. Weitzman et al. (1974) reported a sequence reversal of SWS and REM in 50% of 89 naps in which these stages occurred together (from the total of 560 naps). In the Montefiore study SWS and REM sleep occurred together more often (16%) than in the Stanford study (3%), probably due to the longer nap durations (1 hr) in the former study. In general, REM sleep tended to occur (128 times) in sleep periods that alternated with those containing SWS and rarely appeared (19 times) in consecutive sleep episodes (Carskadon and Dement, 1975). SWS, on the other hand, recurred with both consecutive (153 times) and alternate (185 times) sleep periods in the same study. SWS had no clear circadian rhythm and was evenly distributed across the 24 hr. Combining results across studies and including data from the Moses et al. (1975) study to be described in the next section, subjects were awakened from REM in 18% of the naps and from SWS in 44% of the naps.

A striking increase in recovery TST was found, higher than that usually found in sleep deprivation studies of comparable duration. Such increases were more prominent for REM and SWS. It is difficult to determine whether the increase in recovery REM sleep reflected a deprivation-rebound effect or was due to the extended recovery sleep. The disruption of the 24-hr sleep-wake cycle produced by the fragmented sleep schedules was hypothesized as the reason for this exaggerated recovery TST increase: during the recovery nights, the subjects probably delayed awakening time due to the phase shift in TST peak (from early morning to mid- to late morning) which occurred during the experiments. Most likely, these experiments, lasting from 6 to 10 days, were long enough to produce a disruption in the sleep system, but probably still too short to permit full entrainment of subjects to

the new schedules. In a reanalysis of the Stanford studies data, Strogatz (1986) found that, in fact, circadian rhythms of both TST and body temperature were free running with a period of 25.25 hr, as a result of the schedule, even though no effort was made to shield subjects from time cues.

In the Stanford study, sleepiness ratings were collected and they paralleled the phase shift found in TST. Subjects were sleepiest in the late morning hours and lower ratings were observed in the evening hours (the "forbidden zone" for sleep). Moreover, pre- and postnap sleepiness ratings appeared to demonstrate regular patterns. Naps with longer SWS periods were generally followed by more self-reported sleepiness, whereas greater prenap sleepiness predicted greater amounts of REM sleep in the subsequent nap.

Although on the first experimental day napping did not alleviate subjective sleepiness ratings, throughout the next 4 days the sleepiness measures decreased, and mood improved considerably, with all approaching baseline levels. In all studies there was a relative lack of complaints usually associated with other studies of comparable sleep loss. Taken together, these findings suggest that the subjects were gradually adapting to the polyphasic schedule. However, the studies were too short to confirm this apparent trend.

Sleep on Other Than 24-Hour Regimens

The next set of experiments (Webb and Agnew, 1975, 1977) was concerned with the effects of other than 24-hr schedules on sleep efficiency and sleep stages. Regimens shorter and longer than 24 hr were investigated. In each schedule the ratio of sleep to wakefulness was held constant in a 1:2 relationship. The regimens studied were 9-, 12-, 18-, 30-, and 36-hr sleep-wake cycles. In the two regimens relevant to this review, 2 subjects were submitted to a 3/6 sleep-wake schedule (a 9-hr "day") for 6 days, and 4 subjects to a 4-8 schedule (a 12-hr "day") for 14 days. Sleep efficiencies obtained on the two schedules were slightly less (80 and 84%, respectively) than those obtained under the baseline 24-hr days (93.4%), yet considerably higher than in the Stanford and Montefiore studies. The sleep losses resulted in part from the sum of virtually normal mean sleep latencies, and in part from a slight increase in wakefulness after sleep onset. Stage 2 was reduced and stage 4 increased, but the amount of REM was maintained within the modified regimens. REM percents were greater and stage 1 + 2 percents smaller in the 9-hr-day study as compared to the 3-hr-day and the 90-min-day studies described previously. Similarly to the latter and other studies, REM amounts showed clear circadian rhythms with greater amounts from midnight to early morning hours. In sum, as Webb and Agnew (1977) conclude, under these altered regimens (though not as altered as in the three previous studies) "the basic structure of sleep persists" (p. 448). In addition, "the general stability of the EEG sleep pattern is all the

more remarkable when a more refined analysis indicates that the variables of prior wakefulness, sleep length, and time of sleep are operating predictably on the infrastructure of sleep" (p. 448).

The 1-Hour "Day"

In the last study discussed in this section (Stampi et al., 1992a), 4 subjects have been submitted to a 1-hr "day" for a period of 2 wk. Subjects appeared to have adapted well to the highly fragmented sleep regimen involving 20-min sleep and 40-min wakefulness. Surprisingly enough, their most common complaints were not related to the short duration of sleep episodes they were allowed to take. Rather, subjects were often disappointed by the short waking periods, which did not allow them to engage in activities longer than 40 min at any one time. Contrary to previous studies, this study included repeated performance testing in a variety of tasks *and* physiological recordings (EEG, EOG, EMG). Results from this study are currently being analyzed and will be reported elsewhere.

The 20-Minute "Day"

Although their experiments lasted for relatively short periods (up to 48 hr), the findings from studies conducted by Lavie (Chapter 8) and colleagues on ultrashort 5/15 min and 7/13 min sleep-wake schedules are relevant to this section. They have confirmed that there are ultradian, semicircadian, and circadian "gates" of sleep and wakefulness, facilitating transition between states. When sleep pressure was high (e.g., at night), subjects were able to fall asleep within less than 1 min after lights out and take advantage of the better part of such reduced bed times. These studies are discussed in greater detail in Chapter 8.

"Anchor" Sleep (Without Reduction of Time in Bed)

The Concept **of "Anchor"** Sleep

In a series of studies that examined the effects of irregular and abnormal sleep-wake schedules upon the circadian timing system (Hume and Mills, 1977; Minors, and Waterhouse, 1981, 1983; see also Chapter 7), a total of 63 subjects were submitted to schedules consisting of 4 hr of sleep taken at the same time every day, plus 4 hr of sleep taken at irregular times, for periods ranging between 7 and 12 days. Previous studies had shown that irregular sleeping routines, whether as a single randomly timed 8-hr sleep or as two randomly arranged 4-hr sleep periods, were associated with free-running

circadian rhythms with periods greater than 24 hr. The Hume and Mills (1977) and the Minors and Waterhouse (1981, 1983) studies have demonstrated that if one of the 4-hr sleep periods—which they called "anchor" sleep—was taken at the same time each day, the circadian rhythms became stabilized with periods indistinguishable from 24 hr, even when the other 4 hr were taken irregularly. Circadian rhythmicity was assessed by several methods including "constant routines" conditions, that is, when rhythmic influences in the environment and sleep-waking pattern had been minimized.

Unfortunately, only the Hume and Mills (1977) study recorded EEG, EOG, and EMG on their 9 subjects, and none of the anchor-sleep studies reported any information on performance or sleepiness levels under such conditions. As occurred in the studies previously described in this chapter, the amount of REM showed a clear circadian rhythm, with maximum shortly before noon. The amount of SWS also showed a circadian rhythm, with maximum in the afternoon, but with lower amplitude compared to baseline. The amounts of SWS were largely dependent upon the duration of prior wakefulness.

Semipolyphasic Sleep Schedules

Stimulated, among other factors, by the potential of anchor sleep for improving stability in circadian rhythms, and hence presumably better adjustment to irregular or polyphasic sleep schedules, Stampi et al. (1990b) pushed the anchor-sleep concept a little further. Subjects were submitted to 4 hr of nocturnal (0100–0500) anchor sleep as in the original anchor-sleep studies, but the remaining 4 hr of daytime sleep were divided into multiple 20-, 50-, or 80-min naps. According to the definitions proposed in Chapter 1, these are typical examples of *semipolyphasic* schedules. Among the objects of the study were the investigation of the sleep architecture, recuperative value on performance, and sleep inertia effects of naps of different durations. Sleep inertia is a potentially important limiting factor under polyphasic schedules, as will be discussed. Nap durations were selected based on findings from field studies (Stampi, 1985, 1989a) in which individuals engaging in very extreme quasi-continuous operations appeared to prefer, and to adapt best, to polyphasic sleep patterns consisting of naps ranging from 10–20 min to 1 hr (these studies will be described extensively in the next section).

Four subjects participated in the study consisting of three different semipolyphasic sleep *conditions* of 4 days each, each condition separated by a 2-wk "wash-out" interval. In the first condition *(Poly-80)* they had 4-hr anchor sleep (0100–0500) plus three 80-min naps per day (see Figure 10.1); in the second condition *(Poly-50)* they had anchor sleep plus five 50-min naps per day; and in the third condition *(Poly-20)* anchor sleep was followed by twelve 20-min naps. Continuous EEG, EOG, EMG, and

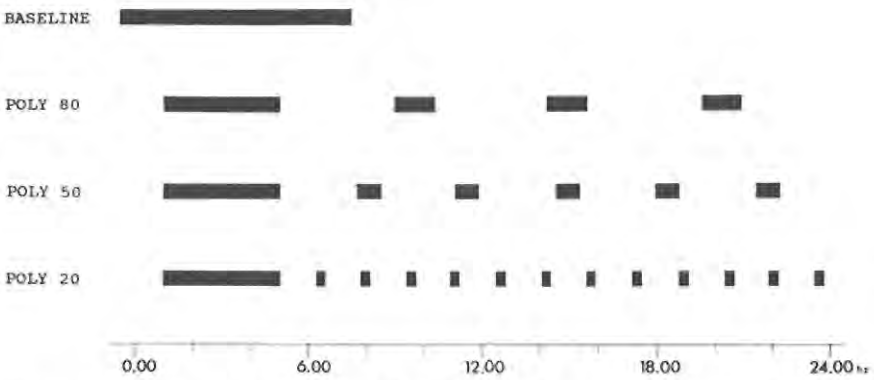

FIGURE 10.1. Sleep-wake schedules followed during the semipolyphasic study: 4 hr of anchor sleep plus three 80-min naps (Poly-80), five 50-min naps (Poly-50), or twelve 20-min naps (Poly-20); black bars: sleep periods.

tympanic temperature recordings were performed, and performance levels on several tasks were measured immediately after arousal from each nap (B), and also 20 min (C) and 54 min (D) after arousal. These values (B, C, D) were compared to the referential performance level measured 20 min before the start of each nap (A). This was done with the purpose of investigating variations in sleep inertia across time.

Analysis of combined nap and anchor sleep showed, as anticipated, that mean daily total sleep times were reduced in all conditions. Sleep efficiencies (TST/TBT) were directly related to nap duration: 85.2, 77.7, and 69.8% for Poly-80, -50, and -20, respectively, compared to an unusually high 97.4% sleep efficiency observed in baseline (see Figure 10.2). It should be noted that efficiencies of anchor sleeps alone were high and remarkably similar to baseline; the overall reduction in TSTs was caused by curtailed

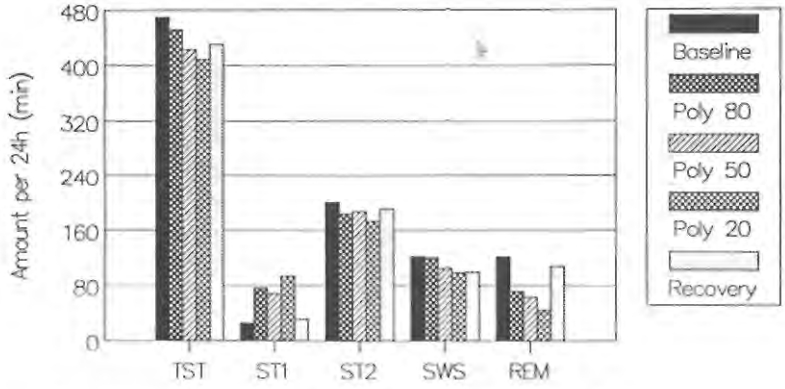

FIGURE 10.2. Sleep architecture during the semipolyphasic schedules.

nap efficiencies (78.3, 64.7, and 44.2%, respectively). However, subjects were unable to fall asleep in only 4 (2.7%) out of the total of 150 naps, all such 4 episodes being 20-min naps. Mean nap sleep latencies were of 5.3 min (to stage 1) and 10.9 min (to stage 2). Sleep propensity showed a midafternoon peak as well as a "forbidden zone to sleep" in the evening: sleep latencies were least at about 1530 and greatest between 1830 and 2200. Interestingly, all four episodes with no sleep fell within the "forbidden-zone."

Consistent in part with what is generally found in short-duration studies of sleep reduction, REM sleep was significantly reduced, but not SWS or stage 2 sleep amounts (see Figure 10.2). REM reduction was greatest in the short-nap-duration conditions. Mean daily REM sleep amounts were reduced to 58.6, 51.9, and 36.8% of baseline levels for Poly-80, -50, and -20, respectively. The total daily amounts of SWS and stage 2 sleep were remarkably similar to baseline. Despite SWS being virtually absent (4%) during the 20-min naps, the amounts "lost" during the naps were regained during the 4-hr anchor nocturnal sleeps. Overall nap stage 1 sleep was considerably increased, presumably due to the numerous sleep onset opportunities that were available. Stage 1 accounted for 43% of sleep time in the 20-min naps, the remaining portion consisting mostly of stage 2 sleep (49%).

Mean recovery sleep TSTs were not inversely related to polyphasic sleep TSTs, as one would expect if the subjects had accumulated any sleep debt. Rather, recovery TSTs were proportional to TSTs found during the polyphasic conditions (mean recovery TSTs were 7.9, 7.3, and 6.1 hr during the Poly-80, -50, and -20 conditions, respectively). This indicates that after 4 days in the polyphasic schedule subjects were presumably and gradually adapting to a reduced daily amount of sleep. Recovery sleep architecture following Poly-80 was virtually identical to baseline; SWS was reduced to 64% of baseline in recovery sleep following Poly-50, and REM sleep was 58.8% of baseline after Poly-20. No significant differences were found in the other recovery sleep components.

The polyphasic sleep patterns did not appear to reduce the subjects' *overall* performance effectiveness (excluding sleep inertia effects—see below). In fact, mean performance in each of the three polyphasic sleep conditions was not significantly lower than in baseline. Performance measured immediately after arousal from naps, however, did show considerable differences between each of the napping conditions. Performance decrements (relative to prenap levels) in the Memory and Search Test (MAST, Folkard et al., 1976) measured immediately upon arousal were positively correlated with nap durations. Decrements were of −14, −8, and −3% for Poly-80, −50, and −20, respectively. However, 54 min after arousal performance was improved relative to prenap, with mean gains of +3, +6, and +7%, respectively. Performance assessed by the Descending Subtraction Test (DST, Evans and Orne, 1976) showed postarousal mean decrements far more pronounced [−25, −36, and −21% for Poly-80,

−50, and −20, respectively (see Figure 10.3)], while 54 min after arousal performance was recuperated but not as much as observed for the MAST (+1.2, +0.4, and +2.6%, respectively).

Confirming previous findings (Dinges et al., 1988), the DST was more sensitive to sleep inertia effects than the MAST. Postnap sleep inertia durations (i.e., the time taken for performance to return to prenap levels) were considerably shorter for the MAST (about 10 min in all three conditions). Sleep inertia (measured by the sensitive DST) was greatest following 50-min naps (mean durations: 37.5 min) and least following 20-min naps (25.3 min), with intermediate levels (32.5 min) after 80-min naps. Sleep stage at the wake-up alarm was one of the key predictors of the amount of performance decrement upon arousal (see Figure 10.4). Greatest decrements were observed upon awakening from SWS (−40.5%), while awakenings from stage 2 produced the least decrements (−17.5%). Interestingly, the latter was remarkably similar to decrements observed when performance was measured following naps in which subjects *were not asleep* immediately before the alarm (−16% decrement). Awakenings from REM sleep produced a −21.6% mean decrement.

In general, the subjects appeared to adapt well to all three polyphasic sleep schedules. In fact, as soon as the effects of sleep inertia were over, there was usually an improvement in the postnap performance levels (relative to prenap), and overall performance was not reduced relative to baseline. Surprisingly, subjects appeared to adapt best to the apparently most "extreme" 20-min schedule, which stimulated subsequent studies using a 20-min sleep/40-min wake schedule for a longer period (Stampi et al., 1992a). Such good adaptation and positive nap recuperative effects oc-

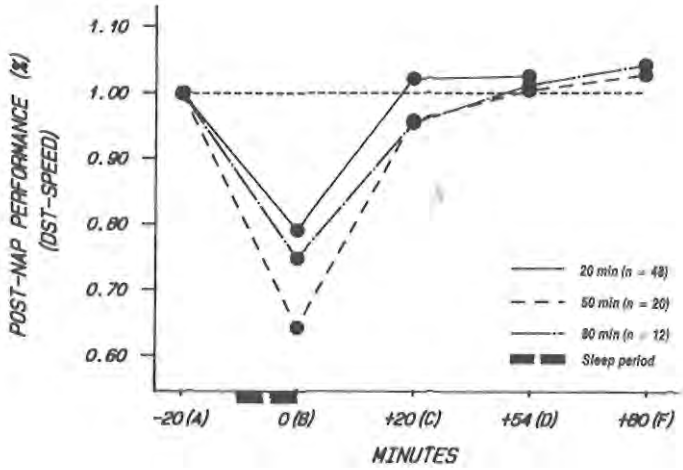

FIGURE 10.3. Sleep inertia and nap recuperative value (on DST performance) as a function of 20-, 50-, or 80-min nap durations.

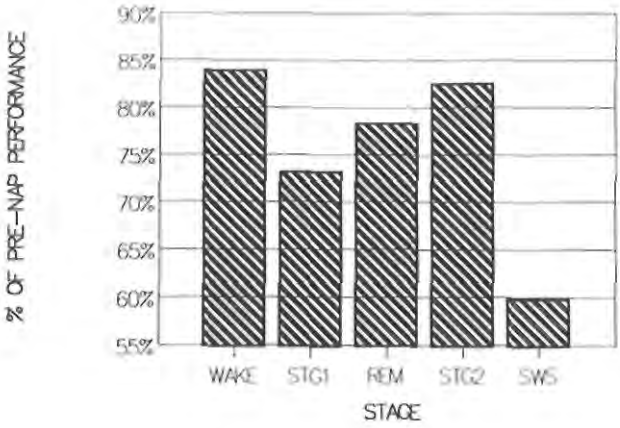

FIGURE 10.4. Performance upon arousal from naps (relative to prenap levels) as a function of sleep stage at arousal.

curred despite their 20-min nap architecture containing very limited amounts of SWS. It should be noted, however, that the 4-hr anchor sleeps played an important role in allowing a short-term recuperation of the presumably "obligatory" component (i.e., SWS) of "core sleep" (Horne, 1988), as discussed previously. In fact, the total daily amount of SWS (naps plus anchor) in Poly-20 was virtually identical to the total SWS time in the subjects' 8-hr baseline sleep. This study further reconfirmed the general tendency observed in ultrashort sleep experiments. That is, changes in sleep-stage composition seem to be related more to the reduction in TST than to polyphasic sleep per se. In the short term (but *not* in the long term), multiple naps (or reduced TST) appear to penalize REM sleep, but SWS will not be denied, regardless of the schedule.

The performance evaluation method used in this study allows speculation on possible nap optimization strategies that could be used to optimize performance levels during polyphasic sleep schedules. For example, which "ideal" napping durations (and timings) could be recommended to the 4 subjects engaged in this study, based on the data collected? Figure 10.5 shows the mean postnap sleep inertia levels in each of the three napping conditions as a function of time of day (midsleep time). Unquestionably, 50-min naps appeared to generate more sleep inertia. Sleep stage at arousal (namely, SWS) was the major factor responsible for this finding (see Figure 10.4). Fifty-minute naps were long enough to allow subjects to initiate SWS, but not too long to permit the ultradian sleep cycle to "return" to stage 1, stage 2, or REM. Indeed, in the 50-min naps, subjects were usually awakened from SWS, as Figure 10.6 indicates. This figure illustrates sleep infrastructure in three typical 24-hr periods for one subjects, with one period from each napping condition. In contrast to what occurred in the

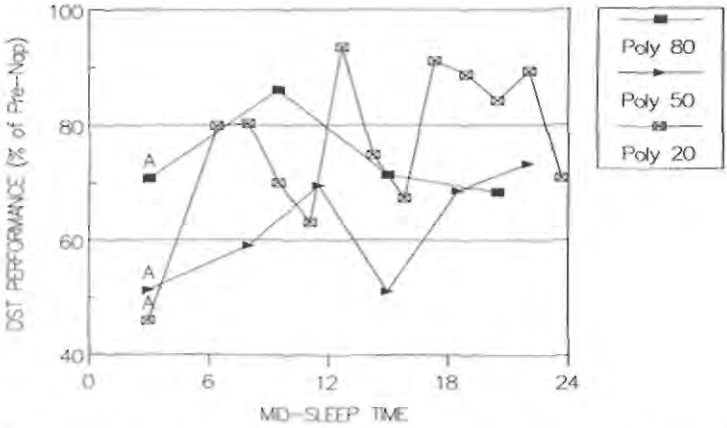

FIGURE 10.5. Performance upon arousal from anchor sleep and 20-, 50-, and 80-min naps (relative to presleep levels) as a function of time of day.

50-min naps, when subjects were engaged in 80-min naps they had enough time to return to "lighter" sleep stages (1, 2, or REM), whereas the 20-min naps did not provide enough time to proceed into SWS (in this study).

Figure 10.5 suggests that if our subjects took 8 hr of sleep per day consisting of a 4-hr anchor sleep plus six 20-min naps (at 0750, 1230, 1710, 1845, 2020, 2155) and one 80-min nap at 0900, they would, in theory, minimize their sleep inertia. That is, immediately upon arousal they would show performance decrements not below −20% of prenap performance levels, and shortly after arousal performance would be increased relative to prenap as a result of the nap. This is, of course, just hypothetical and has not been tested yet on these subjects. However, this is one among several examples of how an optimal nap-selection methodology might be developed with the techniques thus far described. Many other variants or combinations could be inferred from this data, and in any case these hypothetical "optimized" schedules should be subject to verification within these and other subjects.

Polyphasic Schedules with Sleep Reduction

This section includes studies in which different types of polyphasic patterns involving various levels of sleep reduction were investigated. Contrary to most studies discussed in the previous section, most studies included here were undertaken with the scope of studying work-rest schedules in conditions of high-demand or quasi-continuous work. Allowed total sleep times ranged from as short as 1.5 hr up to 6.5 hr per day.

Quasi-continuous work schedules may have different meanings according

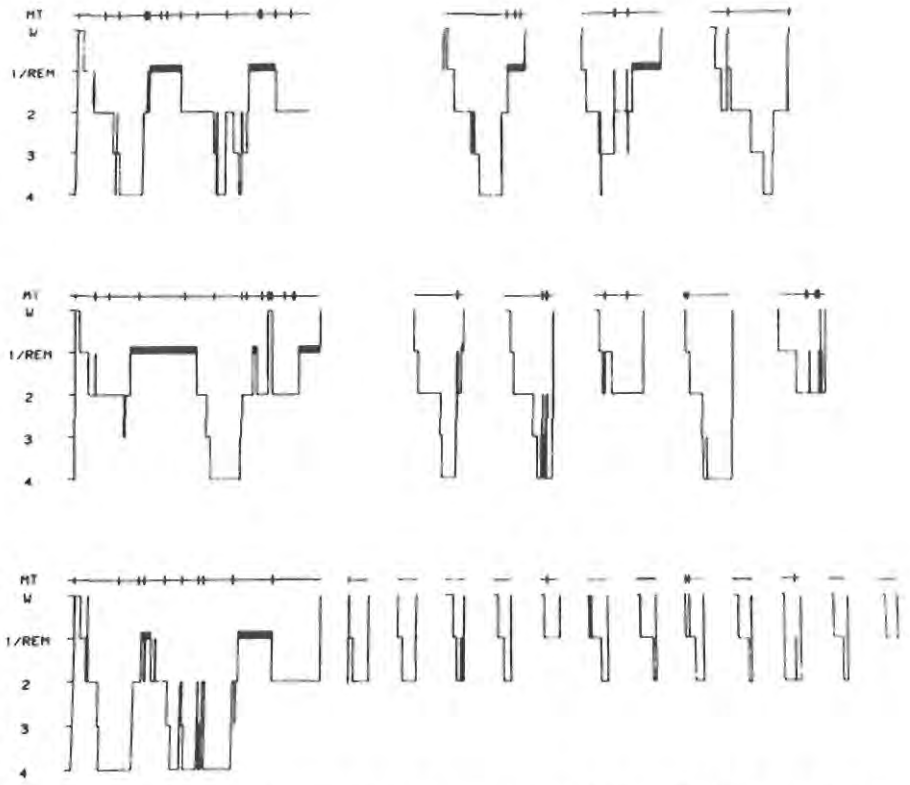

FIGURE 10.6. Sleep architecture during three typical 24-hr periods from one subject. *Top:* Poly-80 condition; *middle:* Poly-50 condition; *bottom:* Poly-20 condition.

to different investigators. For example, Englund and Krueger (1985) have defined two types of scenarios. According to them, *extended operations* consist of continuous work performed within a shift system but characterized by prolonged duty periods. Workers know that periods of rest, sleep, or relief from their duties will be forthcoming. Conversely, under *sustained operations,* there is continuous performance without rest or sleep, where the worker is expected to keep working as long as physically possible.

Continuous versus "Interrupted" Sleep

The first documented attempt toward comparative evaluation of one continuous versus several short sleep episodes was that of Husband (1935), as mentioned earlier. According to Husband, "one might sleep two to three hours, stay awake for a number of hours, and then sleep two or three hours more. This procedure would, in theory, take advantage of two stretches of deep slumber." After one month of uninterrupted 8-hr nightly sleep,

Husband's subject kept a very regular routine of 3-hr sleep (2300-0200), 3-hr waking (spent in various activities), and a second 3-hr sleep (0500-0800). Thus, the schedule was a compromise between a slight sleep reduction and fragmented sleep. A series of performance, physical, and psychological tests showed no signs of deterioration during or after the 1-month experimental period. Results led Husband to conclude that "there are no consistent differences between interrupted and continuous sleep; [however] it is recognized that the use of only one subject makes the results only suggestive" (1935, p. 796).

Four Hours Sleep versus Three 80-Minute Naps

The Husband study had a moderate level of sleep reduction (to 6 hr sleep per day). The first study to evaluate alternative schedules under conditions of considerable reduction in sleep (4 hr per day total) was conducted 40 years later (Hartley, 1974). The study compared performance of 12 subjects following each of three different schedules: over a period of 4 consecutive days in each condition, subjects were allowed one continuous sleep period of 8 hr (baseline), and one reduced sleep period of 4 hr, *or a* fragmented sleep period of three naps of 80 min each (4 hr total per day). Performance testing on a 70-min visual vigilance task was held once a day in the evening.

Hartley was the first to show that subjects are able to function reasonably effectively for 4 consecutive days with three 80-min naps per day only. Naps were allowed at 2310, 0530, and 1225. The study revealed that although performance of the nap group was slightly poorer than that of the baseline group permitted *twice as much* (8 hr) nocturnal sleep, it was much better than the performance of the third group allowed 4 hr of continuous sleep (0100-0500). That is, the same amount of sleep fragmented into three brief episodes throughout the 24 hr produced higher performance relative to monophasic sleep of comparable duration.

The Hartley (1974) study provided evidence that multiple naps were effective in offsetting performance decrements due to sleep loss and facilitated vigilance relative to a reduced nocturnal sleep period. Unfortunately, there was no information in this study" regarding sleep composition (EEG, EOG, and EMG measures were not recorded), nor concerning the variation of performance over the day, nor the recuperative value of each nap. In fact, performance measures were taken only once a day. Some of the subsequent studies discussed in this section tried to overcome such limitations.

The 60/160 Minute Sleep-Wake Schedule

Shortly after the publication of the Hartley (1974) study, another study that was designed with an applied point of view tried to answer a question that summarizes many of the issues raised in Chapter 1, in this chapter, and in

the whole volume: "given that there is some emergency which imposes at least one night of sleep loss on a subject, what is the best regime to follow before and during sleep loss which minimizes sleep loss impairment?" (Lubin et al., 1976, p. 335). In this study, performance and sleep architecture (reported by Moses et al., 1975) were examined over 40 hr, in subjects assigned to one of three groups: a no-sleep with exercise group, a bed-rest group, and a nap group. The 40-hr period was divided into 220-min cycles in such a way that 60 min were spent exercising, resting, or napping, and 160 min with performance and mood testing. Ten subjects were assigned to the napping group, and they took a total of ten 1-hr naps over the 40-hr period.

Lubin et al. (1976) found that when subjects are deprived of their normal nocturnal monophasic sleep, the fragmented sleep schedule diminished the sleep loss effects better than bed rest or exercise. Relative to a baseline non-sleep-deprived condition, the nap group had no impairment on six of the eight performance and mood measures taken. This is particularly interesting, since the napping subjects slept for only 61% of the available sleep time (about 4.8 hr of sleep per 24). Subjective ratings of sleepiness were increased relative to baseline similarly to what occurred on the first day of the Carskadon and Dement (1977) study (90-min "day"). It is likely that, if the study had continued for more days, sleepiness would probably have decreased to baseline, as occurred in the 90-min-day study over the next 4 days. The exercise group showed the most damage in all measures, indicating that exercise may potentiate the sleep loss effect. The bed-rest group was performing at an intermediate level between the two groups, and it was concluded that forced bed rest is not a short-term substitute for sleep.

Similarly to what occurred in polyphasic studies described in the previous section, nap sleep infrastructure (data from 8 subjects, Moses et al., 1975) showed that naps were not replicas of the first hour of the 8-hr nocturnal sleep, except when a nap coincided with the habitual retiring time. Only 25% of the 80 naps contained both SWS and REM; of these, only 2 naps showed the usual temporal sequence of REM following SWS. Twenty-one of the 34 naps containing REM were SOREMPs. REM amounts showed a circadian rhythm with peaks between 0200 and 1000, and sleep efficiency was maximal when temperature was lowest. Recovery sleep for the nap group did not show the usual changes associated with sleep loss, which were found in the exercise group.

Moses et al. (1978a), using the data of Lubin et al. (1976), examined the relationship between performance, self-reported sleepiness, oral temperature, and parameters of sleep infrastructure for the nap group. Within-subjects correlations indicated a significant relationship between total sleep time, oral temperature, and the number of missed signals on the auditory vigilance task. That is, they found lower accuracy on the vigilance task following longer nap duration with lower temperatures. Higher levels of postnap sleepiness were also associated with longer nap sleep. Since sleepiness was assessed within 5 min of awakening, these results suggest that

an increase in sleep inertia occurred following longer sleep. However, Moses et al. (1978a) concluded that performance, sleepiness, and temperature varied synchronously solely because of their common association with time of day. With circadian effects removed, these variables showed no consistent relationship. Thus, according to these findings, predictions about the level of sleepiness and performance cannot be made using temperature readings at a particular time of day.

Moses et al. (1978b) further compared sleep efficiency of the three different sleep-wake schedules reported previously: the 60/160 min (Lubin et al., 1976; Moses et al., 1975), the 60/120 min (Weitzman et al., 1974), and the 30/60 min (Carskadon and Dement, 1975, 1977). Nap sleep efficiency referred to the ratio of time spent in stages 2, 3, 4, and REM to TBT. The 90-95% sleep efficiency usually found in nocturnal monophasic sleep was reduced to less than 60% in these three polyphasic sleep studies. However, the longer duration study (10 days, Weitzman et al., 1974) indicated that sleep efficiencies were gradually increasing. This trend toward gradual adaptation to polyphasic patterns was confirmed by subsequent polyphasic studies of much longer duration (Stampi et al., 1990a; Stampi and Davis, 1991). That is, a considerable amount of time appears to be necessary before subjects can ("learn to") take full advantage of the limited times available for sleep during polyphasic schedules. All such short-day studies showed that fragmented sleep does not appear to alter circadian rhythms (Moses et al., 1978a).

In an additional reanalysis of data from these three studies (Lubin et al., 1976; Moses et al., 1975; Weitzman et al., 1974; Carskadon and Dement, 1975, 1977), Moses et al. (1978b) studied the length and rhythmicity of the REM cycles. Autocorrelation and r^2 analysis (a measure of the strength of the REM cycle periodicity) was applied to "compressed sleep" (i.e., all sleep minus the wake time between and within sleep periods) of the baseline sleep, the nap sleep during the experimental protocols, and the recovery sleep. Compared with baseline, there were no significant differences in nap REM cycle length (approx. 100 min) in two experiments (60/120 min and 60/160 min). The third study (30/60 min) had significantly shorter cycles (60 min). It appeared that this shortened REM cycle length was owing to the significantly shorter REM episodes in this study. Despite this difference, all three studies obtained the same amount of REM sleep relative to TST.

Relative to baseline sleep, however, nap r^2 values were significantly lower in the 60/120 and in the 30/60 schedules, indicating an increased variability in the timing of REM sleep during naps. In other words, the strength of the rhythm was considerably reduced; however, nap r^2 values were significantly higher than those obtained from a random distribution of sleep stages. An interesting finding was that both nap sleep efficiency and nap r^2 values were significantly correlated across the subjects, demonstrating that those who adapted well to the schedule (i.e., individuals who fell asleep easily and remained asleep during the scheduled sleep periods) also had more rhythmic REM sleep.

Moses et al. (1978b) found that although the *amount* of REM in all three studies was greatest between 0200-1000, REM *onset* had no consistent relationship to time of day. Rather, REM onset appeared to be related to the amount of sleep time since the last REM onset. They concluded, therefore, that these data offer further support for the view that the REM cycle is a sleep-dependent rhythm (that is, it appears only when the subject is sleeping) and not an expression of an ongoing Basic Rest-Activity Cycle (Kleitman, 1961). In other words, the timing of REM onset may be determined by a sleep-dependent ultradian clock: the clock may stop upon awakening and resume at the next sleep onset.

Moses et al. (1978b) also found that sleep-stage percents were remarkably similar in all three groups. They concluded that the reactions of the sleep system to a nap schedule, concerning sleep-stage percents and cyclicity, appeared to be relatively predictable. Johnson et al. (1977) compared the sleep data from subjects who adapted well to a gradual reduction of their nocturnal TST from 8 to 6 hr, showing no impairment in performance, with data from their previous nap study (Moses et al., 1975). They found that the time spent per 24-hr period in the various sleep stages was similar, no matter whether TST was reduced by shortening the single period *or* by fragmenting sleep into naps. Thus, concerning overall sleep-stage percentages and cyclicity measures, the sleep system appeared to react to these experimentally produced sleep reduction schedules in a relatively predictable manner, no matter what type of nap schedule was adopted.

The 15/75 Minute Sleep-Wake Schedule

One of the subjects in the Carskadon and Dement (1975) study repeated the same protocol 5 months after the first study, with the exception that this time he slept only 15 min during each of the 90-min periods (4 hr of sleep per day). Sleep-stage changes were similar to what had occurred in the 30/60 schedule. However, he had REM sleep more often (29 REM periods in 86 naps), and with shorter latencies from sleep onset (mean latency = 4.1 min). REM percentages were unchanged from baseline, but SWS amount was much lower than in the 30/60 schedule. Sleep occupied virtually 100% of the 15-min allotment on all sleep periods, with the exception of the 2230 and 0000 nap times (the "forbidden zone," again). The investigators reported that it was very difficult to arouse the subject at the end of the sleep periods. Measures of sleepiness and mood apparently showed subjective discomfort throughout the experimental period.

One 6-Hour Sleep versus Six 1-Hour Naps

In a study involving rigorous sustained performance testing, Mullaney et al. (1983) studied three groups of 10 subjects each who performed four tasks continuously over a 42-hr period. One group did not obtain any sleep over the 42 hr while the other two groups were allowed either 6 hr of sleep

following 18 hr awake, or six naps of 1 hr each spread over the 42 hr (one sleep episode every 7 hr). Objective sleep measures showed numerous occurrences of microsleeps (periods of drowsiness polygraphically defined as stage 1), which peaked at 0600-0800. Even though the napping group slept less than the monophasic sleep group, with sleep efficiencies of 72 and 92%, respectively, the two groups did not differ significantly in amounts of SWS.

There were no statistically significant differences between the three groups in pattern memory (accuracy) over the first 34 hr of the study. The only statistically significant result indicated that between 35 and 41 hr into the study, both types of sleep were better than no sleep at all. During the last 18 hr of the study, one 6-hr continuous sleep period appeared better than six 1-hr naps in offsetting sleep deprivation effects on the memory and addition tasks. Generally, both the fragmented and continuous sleep patterns were effective in counteracting impairments due to sustained work. For the nap group, sleep inertia was evident on all measures within 10 to 30 min after awakening. Performance generally improved after sleep inertia effects dissipated, indicating the beneficial effects of sleep. Since performance data measured immediately after arousal from naps were also included in the analysis, this may explain why in the last 18 hr of the 42-hr period, the fragmented sleep group performed not as well as the monophasic sleep group. That is, detrimental effects generated by sleep inertia occurred six times for the napping group (i.e., after each nap), as opposed to occurring only once for the monophasic sleep group. In addition, in the monophasic group, sleep time was coincident with the subjects' habitual sleep time, which allowed subjects to accumulate more sleep in one 6-hr period than in six 1-hr naps. In sum, the design of this study does not allow for a simple interpretation of the findings when compared between the two groups.

One 4-Hour Sleep versus Four 1-Hour Naps

In an attempt to answer a question frequently asked by military personnel on whether one period of uninterrupted reduced sleep is better or worse than several naps, Haslam (1985) studied the effects of 4 hr of sleep per day taken either as one continuous block (0200-0600) or as four 1-hr installments (taken at 0500, 1100, 1700, and 2300). Both groups of 6 subjects, all of whom were trained infantrymen, underwent a 23-hr continuous wake period prior to the 4-day quasi-continuous work period. Unfortunately, no physiological information on sleep is provided, since EEG was not recorded.

Sleep logs showed that the napping subjects had less subjective sleep than the monophasic sleep subjects, as could be expected. Despite these differences in TST, performance measured on a battery of cognitive tasks and mood did not show any differences between the two groups. That is, polyphasic sleep was as beneficial as an equivalent amount of uninterrupted

sleep. During the middle of the trial, mood and performance deteriorated in part in both groups. However, on the last day of the experiment, cognitive tests and mood scores were not significantly different from baseline values for either group, suggesting, once again, that an adaptation period is required before individuals can adjust to either reduced sleep or to polyphasic sleep schedules. The study reconfirmed the utility of 4-hr sleep, either in one uninterrupted block or in four scheduled 1-hr naps per 24 hr.

Interestingly, one decade earlier Hartley (1974) had shown that three 80-min naps were even better than 4-hr uninterrupted sleep, and Stampi et al. (1990b) showed that 80-min naps were more recuperative than 50-min naps, as discussed previously. This suggests that minor changes in nap duration may indeed be one of the key factors in modulating maintenance of reasonable levels of functioning under polyphasic-reduced sleep schedules. This assumption gained clear support and further understanding in the set of studies that will be described next.

Spontaneous Polyphasic Behavior

All studies examined thus far have been concerned with *imposing* a variety of different polyphasic sleep scenarios under laboratory conditions. That is, investigators have tried to understand the limits of the sleep-wake system, and its laws, by studying human reactions to schedules designed and based upon a number of different perspectives or hypotheses. Unquestionably, such an approach was very fruitful in providing the considerable knowledge discussed in the previous sections. However, until 1985, no one had attempted to answer a set of apparently very simple questions: What happens in the "real world" when individuals are exposed to prolonged periods of sustained work? What type of schedules would they *spontaneously* select or be forced to adopt? Would individuals tend to sleep less, totally avoid sleep, or switch to a napping regimen? If a polyphasic sleep pattern happens to be chosen, will it benefit the subject's performance? For how long can an individual function on this type of polyphasic schedule?

Given that in the real world there are plenty of quasi-continuous work situations, the next set of studies was undertaken with the purpose of trying to fill this gap and let nature provide the answers (Stampi, 1985, 1989a). The goals were to observe what work-rest strategies a group of motivated individuals would *spontaneously* adopt when they are exposed to prolonged high-demand, quasi-continuous work scenarios; that is, when the very basic biological need for sleep is *competing* with a powerful need to stay awake as much as possible (induced by the condition). The assumption behind these studies was that motivation, survival pressure, and other factors would force the individuals to "invent" or experiment with a number of different strategies. Providing that a suitable real-world situation could be identified, it would shed light on which are the most successful among such strategies.

After surveying a wide variety of continuous work scenarios, what appeared to be an almost ideal field model of sustained performance was identified: solo sailing races across the oceans. During a solo race the yachtsman is exposed for days, even weeks, to recurrent, unpredictable, and sometimes extenuating demands to be awake, so as to steer and adjust the yacht to varying and occasionally remarkable conditions of sea and wind, and to survey the tactics of competitors in order to optimize performance and sustain maximum speed. While he is asleep, the yacht may go off course, either yacht speed or control (by way of the self-steering devices) may not be optimal, and risks of collision with other ships increase. In normal conditions of visibility a yachtsman must check the horizon every 15 min to safely prevent collisions. Motivation to win is very high and is incited by valuable prizes and sponsors' rewards. In sum, some of the usual difficulties encountered in laboratory settings in trying to replicate quasi-continuous operations (e.g., lack of realism, excessively boring or excessively demanding tasks, complacent or unmotivated subjects) were not present here.

In the first (1960) Observer Single-handed Transatlantic Race (OSTAR), Sir Francis Chichester took 38 days to cross the Atlantic Ocean, but by 1988 the fastest yachtsman took only 10 days. Today, the limiting factor is no longer the *boat* (the latest achievements in technology are continually applied to yacht design to increase speed and performance) but the *sailor* and his capacity to wisely administer both mental and physical energy expenditure. Race performance depends therefore on an optimal balance between cognitive and motor skills. Interestingly enough, the 1980 OSTAR winner was a 67-yr-old yachtsman, suggesting that experience plays a key role for success. A detailed technical, medical, and psychological analysis of a solo race is reported by Bennet (1973).

The studies were conducted using a representative sample of 99 yachtsmen. Studies were initiated during the OSTAR of 1980 (3000 nautical miles, 54 yachtsmen studied), and were later completed by a study during the 1982 Round Britain Race (RBR) (1900 miles, 29 subjects), and the 1983 Mini-Transat (MT) (4300 miles, 16 subjects). While the OSTAR and MT were single-handed events, RBR was raced by crews of 2. The OSTAR was won in 17 days, the MT in 31 days, and the RBR in 8 days; the last competitors to finish the races took 49, 40, and 20 days, respectively. Competitor's ages ranged between 20 and 67 yr (means 38.7, 29.7, and 35.6, respectively), and only 4 among the 99 subjects were females. The studies were conducted by analyzing data from the competitor's sleep logs, and by interviewing them extensively before and after the races. Overall indexes of performance for each competitor were essentially based upon the goal of the competition, that is, the very meaningful measure for the sailors themselves: race results. Specifically, they were the mean speed of each yacht and the calculated mean handicap speed [i.e., (mean speed)/(square

root of the waterline length)], since the theoretical maximum speed of a vessel is a function of the square root of her waterline length.

For all three races, mean daily TST was 6.33 hr (SD = 1.7), significantly but not markedly shorter than the sailors' average TST at home (7.5 hr). The most frequently adopted TSTs were at about 5 hr (20% of OSTAR sailors) and 8 hr (28%) per day. Mean sleep episode duration (SED) was 2.01 hr for OSTAR, 0.61 hr for MT, and 2.7 hr for RBR. Overall mean SED was 2.02 hr (SD = 1.7), individual mean SEDs ranged from 0.33 to 7 hr. Therefore, although most sailors only slightly reduced daily TST, many divided it up into several short episodes. Sleep fragmentation was far more evident in the MT race (mean SEDs of all competitors ranged from 20 to 60 min), most likely because the reduced dimensions of the yachts in this race permit faster and hence more frequent maneuvers.

The yachtsmen appeared to be more concerned with the duration than with the timing of sleep episodes. With the exception of some individuals who were able to adapt to highly variable and irregular SEDs, most tried to sleep for the number of minutes (or hours) that, by experience, had been found to be most restorative or to produce the least impairment upon arousal. Most yachtsmen were able to wake up spontaneously at scheduled times without the use of alarms. It was also reported that, even when stable meteorological conditions would permit them to sleep longer, most tried to avoid recuperative oversleeping. According to them, oversleeping would produce unpleasant feelings of drowsiness and decreased efficiency upon arousal (i.e., it would increase the "sleep-inertia effect"). In other words, nap duration seems to be a stable individual characteristic, and the entrainment of the sleep system to one's own "ideal" duration appears to play an important role in the restorative value of each sleep episode.

Competitors having a mean SED of 20 min (16.6%) reported that they oscillated between SEDs of 10- to approximately 30-min duration. Interestingly, based on studies of experimentally induced sleep disruption, Bonnet (1986) speculates that 10 min is the minimal duration for sleep to be restorative (see also Chapter 13). The most-adopted SED was around 1 hr (33% of OSTAR and 27.6% of RBR subjects); 66.5% of OSTAR competitors had SEDs between 20 min and 2 hr.

Forty-five percent of yachtsmen felt that regularity in timing sleep was important and that schedules should be followed as closely as possible, but only 25% thought that this was possible, because of the unpredictability of demands during a race. The recuperative value of sleep per unit time was reported by 30% of the yachtsmen to be higher while they were at sea. Interestingly, all individuals within this group showed ultrashort sleep patterns. Twenty-six percent reported that they recuperate better at home.

Although most yachtsmen reported that "training" their sleep patterns some time before the race start is a very useful strategy, only 10% were capable of doing so. This was primarily owing to the fact that the sailors

were very busy for several weeks immediately prior to the race with preparation of their yachts. Entrainment to the sleep rhythm adopted in the OSTAR took an average of 3.5 (SD = 1.5) days from race start, and most sailors reported greater fatigue at the beginning of the race than on the last days before arrival at the finish—after 3000 to 4000 miles of sailing. At the conclusion of the race, sleep patterns took an average of 3.6 (SD = 5.5) days to return to normal monophasic sleep, although some subjects reported taking as long as 3 wks to reset their sleep. Younger subjects and "evening types" reentrained faster (significant correlation). Recovery TST after the race was not significantly increased in comparison with baseline levels.

Analysis of the yachtsmen's sleep patterns revealed that there were four preferred sleep strategies for this type of quasi-continuous work situation; these are illustrated for the OSTAR.

1. *Ultrashort sleep.* This group was very concerned with competitive aspects of the race, and it represented the most adopted strategy (66.5% of OSTAR competitors); mean SEDs ranged between 20 min and 2 hr.
2. *Navy sleep.* The traditional navy shift-work system is 4-hr off/8-hr on. Similar patterns were adopted by 18.5% of sailors, with SEDs from 3.5 to 4.5 hr, but generally without a rigid timing. They tried to reach a compromise between subjective need for longer sleep episodes and the race requirements for frequent watches.
3. *Sleep accumulation.* A few sailors (7.4%) reported they were able to sleep whenever possible, with no regular SED or timing; they could also, if necessary, sleep in excess in order to "store" sleep and could then stay awake uninterruptedly for as long as 3 or 4 days.
4. *Monophasic sleep.* Some sailors (7.6%) simply tried to reproduce their habitual sleep habits of a nocturnal 6–8 hr of monophasic sleep, except when in evident danger. Members of this group were either not interested in race success or were incapable of polyphasic sleep.

Comparison between baseline (home) sleep parameters and race sleep patterns revealed no correlation; that is, it was not possible to predict the type of race sleep schedule based on home sleep. Only sailing experience significantly predicted race sleep patterns: experienced yachtsmen tended to adopt shorter sleep episode durations and TSTs. Seven of the 10 competitors considered as most experienced adopted mean SEDs of 20 min to 1 hr.

A preliminary analysis of possible relations between sleep patterns and race performance (mean yacht speed) was done by dividing the competitors into three groups for each race, according to their race result. A clear difference in SED and TST was observed among the groups. The fastest group of competitors tended to sleep less and to fragment sleep into shorter episodes (OSTAR means: SED = 1.3 hr, TST = 4.8 hr) than did the second-fastest group (SED = 2.1, TST = 6.7). Similar differences were

observed between the middle (second) and the slowest group of competitors (SED = 2.7, TST = 7.5).

If competitors are grouped together according to their mean SED, and then the mean handicap speed obtained by each group is plotted against SED (see Figure 10.7), it can be observed that sailors who attained the highest speeds had SEDs between 20 min (best group) and 1 hr, whereas SEDs of 2 or more hr were associated with an abrupt decay in performance. Linear regression analysis performed between mean SED and mean speed of yacht (race performance) for each subject showed a significant negative correlation both for OSTAR ($p < 0.05$) and for RBR ($p < .001$). That is, the shorter a sailors' mean sleep episode duration, the better his race result. Similar significant results were found by substituting handicap speed for true speed.

This finding is in apparent contrast with what might be expected if the sleep-accumulation strategy, with its apparently high degree of flexibility, were assumed to result in the best race performance. Actually, some subjects in this group did show good results, as Figure 10.7 indicates. Three of the seven sailors sleeping for an average of 4 hr ($n = 4$) or 6 hr ($n = 3$) were classified as sleep accumulators. These performed better than sailors

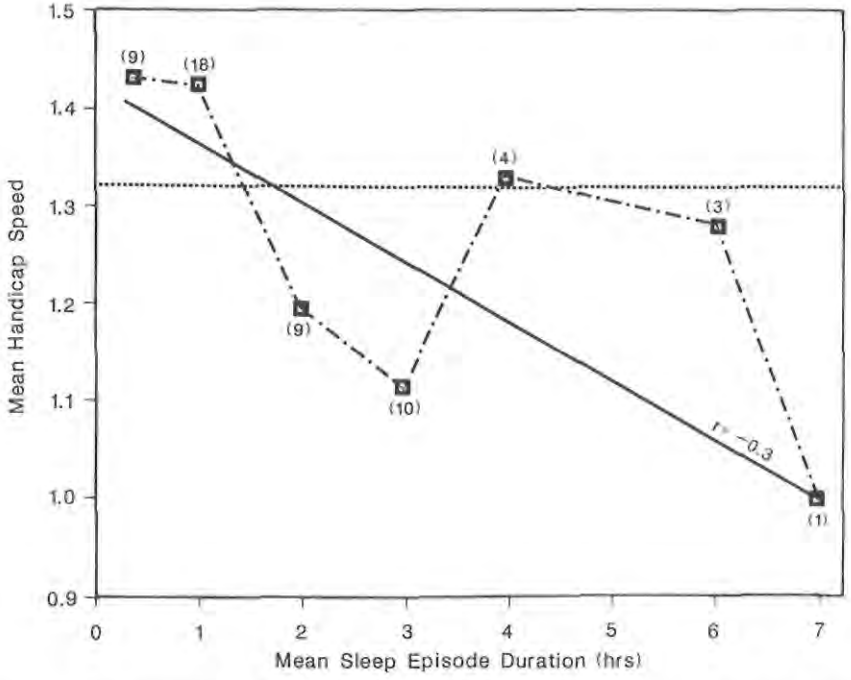

FIGURE 10.7. Performance (mean handicap yacht speed) as a function of sleep episode duration (OSTAR).

sleeping for 2 or 3 hr, but worse than those with SEDs of 1 hr or less. It is possible that if there were "restorative" advantages of having longer sleep episodes, this would compensate in part, but not completely, for the disadvantages in race performance of being on watch less often. However, even if there were hypothetical advantages in the sleep-accumulation strategy (which have never been demonstrated), the capacity to "store" sleep appears to be far more difficult to achieve, or at least to be much more rare (7.4%), compared with the ability to fragment sleep (66.5% had SEDs between 20 min and 2 hr).

The analysis of handicap speed as a function of TST (see Figure 10.8) shows that daily TSTs between 3 and 5.5 hr provided best performance results, but highest speeds were obtained by those sleeping 4.5 hr a day. It must be added that few people slept for 4.5 hr a day or less (11%) and that within the reduced-sleep group (TST of 3–5.5 hr), most (49%) preferred a TST of 5 hr. Subjects sleeping 6 hr or more showed a huge decrease in performance, and the high percentage (28%) of sailors who slept for 8 hr a day did not obtain good results. Linear regression analysis between TST and yacht speed (real time and handicap) showed a highly significant

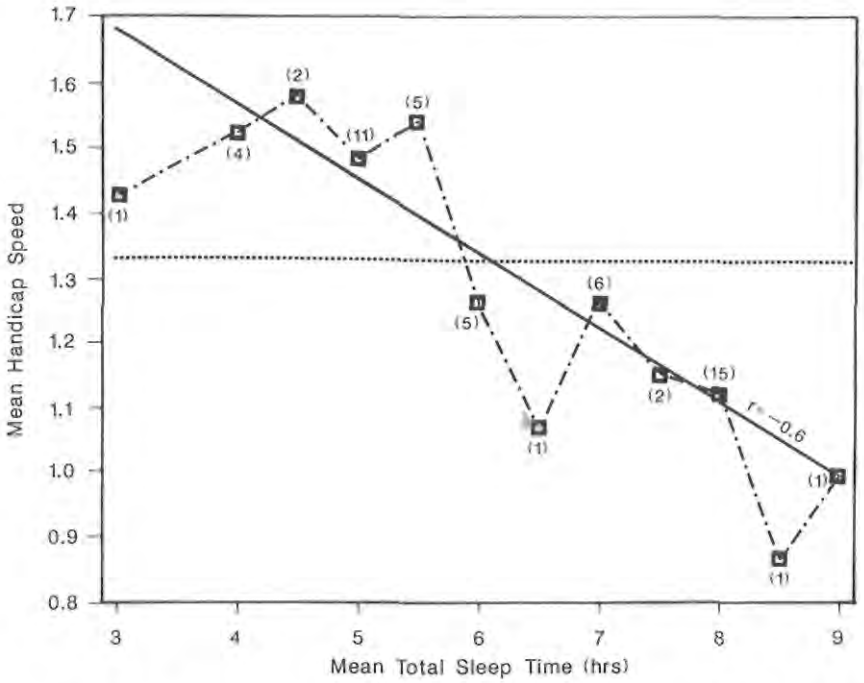

FIGURE 10.8. Performance (mean handicap yacht speed) as a function of total sleep time per 24 hr.

negative correlation ($p < .01$) in all three races: the shortest TSTs predicted the best race performance results.

Provided that TST reductions were not extreme except for a minority of competitors, these findings are in agreement with what is known from studies of partial sleep reduction in normal environments, as discussed previously: namely, reductions by 1 to 2 hr in habitual TST for relatively long periods do not produce impairment in performance, and subjects appear to adapt with relative ease to such reductions. The lab finding of ease of adaptability has, therefore, been confirmed in the field by these studies during conditions of extreme sustained work.

Finally, no correlation was found between the level of sleep fragmentation (i.e., the mean number of daily sleep episodes: TST/SED) and race performance. Race performance, therefore, seems more related to a *combination* of ability to sleep for short periods and capacity to reduce TST to some extent. Competitors tried to overcome the need for staying awake and on watch as much and as frequently as they could, not by greatly reducing TST but mostly by fragmenting sleep. More specifically, under this type of quasi-continuous work demand, people who choose to have (and who are able to adapt to) sleep episodes ranging from 10–20 min to 1 hr will have higher probabilities of better performance, while obtaining a total of approximately 4.5–5.5 hr of sleep per day.

In conclusion, it appears from these studies that most highly motivated individuals (and/or people under particularly extreme task demands) adapt to ultrashort sleep-wake patterns without great difficulty, for relatively long periods, and can maintain (at least for the task at hand) high levels of behavioral efficiency when doing so.

It is unfortunate that obtaining physiological recordings (EEG, EOG, EMG) is virtually impossible under such extreme field studies. Sailors are too busy to self-apply electrodes for ambulatory monitoring. Therefore, no information was obtained concerning sleep infrastructure of sailors during their polyphasic sleep patterns. It could be argued that sleep logs may not provide extremely precise information, which is necessary especially when mean sleep episode durations are so short. To overcome this possible source of noise in the data, a subsequent study was undertaken with the intent of documenting sailors' sleep-wake patterns with objective means (Stampi and Broughton, 1989). A sample of 19 sailors participating to the 1988 OSTAR race were asked to wear continuously wrist or ankle Actigraphs (Ambulatory Monitoring, Inc.). Such miniature data-loggers are able to record wrist or ankle movements, which allow good discrimination between wakefulness and sleep (comparisons between actigraphic and polysomnographic scoring of sleep produce usually 95% epoch-by-epoch agreement, e.g., in Webster et al., 1982). However, even though Actigraphs allow reliable distinction between sleep and wakefulness, they do not allow any distinction between sleep stages.

The actigraphic study (Stampi and Broughton, 1989) basically confirmed the findings of the previous (Stampi, 1985, 1989a) studies, showing that most of the sailors investigated adopted polyphasic ultrashort sleep-wake patterns. The study also indicated that, with a number of improvements that were recommended and subsequently incorporated in the design of the instrument, Actigraphs may be useful rest-activity monitoring devices under such extreme field conditions.

Ultrashort Sleep and Marathon Tennis Play

There are other "natural" models of quasi-continuous operations worth exploring, especially among sporting events, as, for example, during competition for the world record in continuous marathon tennis play. The athletes are compelled to perform under conditions of extended physical exercise and marked sleep deprivation, thereby providing researchers with interesting situations to study the effects of ultrashort and polyphasic sleep behavior. Edinger et al. (1990) studied the combined effects of sleep reduction and exercise on the EEG patterns and subjective measures of sleepiness of two competitors engaged in a 146-hr marathon tennis match. The players earned 5 min of rest for every hour played (total of 2 hr per day). This rest time was pooled so that they could sleep only once per day: they had a 1-hr nap (0300-0400) on the second day of the match, and 2-hr naps (0300-0500) on the third through the seventh (and last) days of the match. Despite such marked sleep restriction, both players continued to obtain their usual (baseline) amounts of SWS throughout the marathon. They also showed a gradually increasing tendency toward daytime dozing across the first few days. This tendency decreased on the fifth day but increased again on the sixth day of the match. However, the players differed markedly in their ratings of sleepiness, mood, recovery sleep patterns, and endurance with respect to the demands of the match. Interestingly, the player who reported less sleepiness during the marathon was also a habitual napper (5 hr of nocturnal sleep plus one 1.5-hr daytime nap), although he refrained from his usual daytime nap practices during the match.

On a subsequent study, Tafti et al. (1990) studied another physically fit athlete attempting to break the world record for marathon tennis play. The marathon lasted 147 hr, 20 min (6.1 days). This time the player adopted a polyphasic sleep strategy (it is not known whether this was decided based on the difficulties experienced by the previous players, who chose a monophasic sleep pattern). During daytime, the available rest time was pooled and the athlete took a 15-min nap every 4 hr, plus 5 min dedicated to health care. During the nighttime, depending on the difficulty of staying awake experienced by the subject, the 4-hr rest cycle was occasionally modified.

Tafti and colleagues recorded two 24-hr EEG periods after respectively 40 and 80 hr of playing, plus the pre- and postmarathon sleep periods. The player succeeded in falling asleep in all 17 naps recorded, with mean sleep

latencies shorter than 1 min. TST per 24 hr were 73 min and 95.5 min after 40 and 80 hr playing, respectively. REM sleep was totally absent from naps, stage 2 occupied 65 and 83% of sleep time, while SWS occupied 23 and 15% of sleep time, after 40 and 80 hr play, respectively. Recovery sleep showed that 37% of the lost SWS amount (based on baseline sleep recordings prior to the event) was recuperated within the second recovery night, while only 7% of REM sleep that was lost had been reclaimed at that time. The number of faults and pauses in the play was markedly increased after 80 hr of play, suggesting a clear performance deterioration. The authors concluded that, as suggested by other sleep deprivation studies, SWS is of greater importance than REM sleep for the function of recovery after sleep deprivation. Unfortunately, because the subjects were not the same and the methods adopted in the two studies were quite different, it is not possible to draw meaningful comparisons between the two different strategies (monophasic and polyphasic) adopted.

The "Leonardo da Vinci" Schedule: Fifteen Minutes Sleep Every 4 Hours

The last set of studies in this review was concerned with understanding the limits of minimal sleep. That is, are there strategies that allow dramatic reduction of sleep amounts *below* what are currently considered the minimum sleep requirements (i.e., 4.5-5.5 hr of sleep per day, as discussed previously)? Findings from some of the most extreme studies described earlier provided some preliminary evidence suggesting that under conditions of marked sleep reduction polyphasic sleep may be more efficient than monophasic sleep. The implications of this hypothesis and speculations on why this may occur are discussed in greater detail elsewhere in this volume (Stampi, Chapter 1). The next two studies described here (Stampi et al., 1990a; Stampi and Davis, 1991; see also Chapter 12 for further details) are the first of a program designed to investigate the hypothesis that *only* polyphasic sleep, but *not* monophasic sleep, allow maintenance of satisfactory levels of functioning under conditions of drastic sleep reduction.

In these studies it was shown that one healthy volunteer was able to adapt well for relatively prolonged periods to polyphasic schedules allowing less than 3 hr sleep per day total. The first study was undertaken for 19 days (Stampi et al., 1990a), and the second lasted 48 days (Stampi and Davis, 1991). The schedules were such that the subject was allowed one nap of 15 min (19-day study) or of 30 min (48-day study) every 4 hr. That is, he slept at regular intervals six times per day. These schedules were based in part on an anecdotal reports attributing such a regimen to Leonardo da Vinci (see Chapters 11 and 12). The Leonardo schedule was apparently adopted successfully for a period of 6 mo during an uncontrolled experiment (Sbragia, Chapter 11).

Confirming Sbragia's personal observations, performance was not significantly reduced below baseline levels during the experiments. The subject adapted remarkably well to the schedules, both subjectively and objectively. Contrary to usual findings from sleep reduction studies, in which only part of REM and stage 2, but not SWS total amounts, are lost, in the Stampi et al. (1990a) and Stampi and Davis (1991) studies *all* sleep stages were proportionately reduced. That is, sleep-stage percentages were remarkably similar to baseline monophasic sleep. This was presumably due to the longer duration of these studies compared to previous studies. This finding suggests that in the medium to long term all sleep components are equally important for sleep function, as was discussed earlier in this chapter. A control study has been recently concluded: the same subject followed a *monophasic* sleep schedule of 3-hr sleep per day (0200-0500) for a period of 3 wks (Stampi et al., 1992b).

Conclusions

The findings from these studies appeared to confirm that adult humans have a natural ability to adapt to polyphasic schedules. In addition, polyphasic sleep is probably the only strategy to allow drastic sleep reduction. One key factor in allowing such adaptation might be centered around the 4-hr sleep-wake cycle that was followed in the "Leonardo da Vinci" experiments: indeed, as mentioned at the beginning of this chapter, there is growing evidence concerning the existence of 4-hr cycles in sleep propensity. However, this may not necessarily be the only reason why a number of studies showed that a given amount of reduced daily sleep provides better performance effectiveness when sleep is split into several episodes, as compared to one monophasic sleep period. At least two additional factors may play important roles in this context: on a reduced sleep regimen, one would expect that individuals can more easily sustain shorter rather than longer periods of wakefulness (e.g., in the Leonardo da Vinci paradigm, 3.5 hr as opposed to 21 continuous hr of wakefulness); it is possible that every time the organism falls asleep some process related to recuperation might be activated, which may be independent from sleep episode duration, and therefore, that overall beneficial effects are related to the number of times the sleep system is activated (within reasonable limits).

Summarizing what was presented in this review, it appears that the organization of sleep within a nap under polyphasic schedules is quite different from that occurring in monophasic nocturnal sleep. Naps are indeed "not miniatures of the normal 8-h sleep pattern" (Weitzman et al., 1974), and only rarely are they replicas of the first part of a normal nightly uninterrupted sleep. For example, REM sleep onset episodes are quite frequent during polyphasic schedules, and it is interesting to note that REM sleep and SWS appear to be mutually exclusive under such conditions: they

rarely occur together during short naps. Despite sleep architecture being remarkably different, long-duration studies indicated that *all* sleep stages (and not just SWS) appear to play an important functional role under these sleep reduction patterns. Indeed, after the initial adaptation period in which daily amounts of all stages but SWS tend to be reduced in amount, sleep percentages become remarkably similar to baseline conditions.

The study of the effects of polyphasic schedules on sleep architecture and performance provides tools that may assist in the development of strategies to enhance and optimize functioning during situations involving quasi-continuous work. From the perspective of sleep management in such situations, the most interesting findings are that the sleep-wake system appears to have a high plasticity in terms of scheduling and sleep episode duration, and that humans appear to adapt surprisingly well to such schedules of multiple naps. However, it also is clear that motivation plays an important role in allowing smooth adaptation to polyphasic sleep schedules, particularly those that involve marked sleep reduction. It is also of noticeable interest that polyphasic sleep appears to be the strategy of choice for individuals engaged in extreme-demand scenarios (solo sailors), allowing for possible speculative analogies to what occurs with mammals living in very dangerous environments. Interestingly, it was found that the greater the experience of the sailors, the shorter the mean sleep episode duration they adopt during their continuous work enterprises. This suggests that it might be possible to "learn" or "train" to adapt to polyphasic sleep schedules, and to benefit from very short episodes of sleep.

The utilization of optimum polyphasic schedules under continuous work requires a framework in which sleep discipline becomes a legitimized and desirable activity. That is, logistics should also consider the following: provision for accessible, quiet, and comfortable facilities that permit frequent and efficient napping without loss of time; education of personnel to take advantage of available slack times to nap; and training of individuals to learn to fall asleep more rapidly by, for example, the use of relaxation techniques.

Indeed, one key factor allowing good adaptation to polyphasic schedules appears to be related to the ability to fall asleep quickly and at almost any time of day. This has been often reported by "semiprofessional" polyphasic sleepers (e.g., solo sailors), and some of the laboratory studies described in this chapter actually revealed that subjects were able to greatly reduce their sleep latencies a few days into the experiments. Increased sleep pressure induced by partial sleep deprivation may be one among the factors —but not the only one — affecting the increased ability to fall asleep quickly. It has been suggested that there are a significant number of individuals who can control sleep onset and that a far greater number than is generally recognized have the potential for learning this skill (Evans and Orne, 1976). Common sense suggests that healthy subjects who are able to fall asleep quickly will also be more likely to adapt to polyphasic schedules.

This brings up the important point of the extent to which individuals vary in their constitutional ability to adapt to polyphasic schedules. It is known that marked interindividual differences exist in a number of sleep-related characteristics. For example, there are natural long and short sleepers, habitual nappers and nonnappers, morning types and evening types, and there are persons who adjust more quickly than others to shift work and jet-lag. Discovering and understanding possible biological or behavioral markers that distinguish people who will more easily adapt to polyphasic schedules would have potential utility in the selection of personnel for special sustained tasks.

There appear to be strong individual differences in terms of "ideal" nap durations. It is worth pointing out that trained subjects (e.g., solo sailors) are particularly concerned about respecting their preferred nap durations: they do not get as much benefit when they sleep for durations shorter or even longer than their "ideal" naps. That is, oversleeping may induce sleep inertia, suggesting that appropriate nap durations depend on the delicate balance between the positive and negative effects of a nap. These effects may have their basis in the duration of sleep that provides maximal recuperation (positive) and minimal sleep inertia (negative). On the other hand, it appears that the main factor affecting sleep inertia is sleep architecture rather than nap duration (the latter may represent just an indirect factor). In particular, subjects waking up from SWS are likely to suffer the greatest amounts of sleep inertia. As Naitoh and Angus (1989) appropriately commented, sleep inertia is often so pronounced after naps during periods of prolonged work that "most investigators either do not test performance for the first 20 to 30 min after a nap or do not include the results of performance tests from this period in their analyses on nap benefits." Yet this topic deserves careful study since sleep inertia represents one of the most important difficulties that might be encountered when designing polyphasic sleep-wake schedules. Is it possible to learn to reduce sleep inertia, analogously to the putative ability to fall asleep quickly described earlier?

It has been hypothesized that one of the key factors allowing the remarkable adaptation found in the "Leonardo da Vinci" schedules (Stampi et al., 1990a; Stampi and Davis, 1991) was the strict regularity of these schedules: subjects had 1 sleep episode every 4 hr. However, in a real-life condition it is not always possible to follow a predetermined schedule. In fact, solo sailors feel that regularity is very important, but that the conditions they are dealing with make it impossible to respect schedules at all times. These field studies revealed that subjects are more concerned with respecting their preferred nap durations rather than the timing of their sleep episodes.

Circadian rhythms do not appear to be affected (with the exception of minor phase-shifts found in some studies) during polyphasic sleep schedules, but more detailed studies are required. Preferred times for sleepiness

bouts and the "forbidden zone" to sleep tend to occur at circadian times similar to normal monophasic conditions. Anchor-sleep periods at constant times definitely appear to improve stability of circadian rhythms. Data presented in this review suggest also that in designing polyphasic schedules the timing of sleep periods should respect the underlying dynamics of biological rhythms. At least three major rhythms should be considered: (1) the *sleep-propensity* cycle (i.e., when transition from wakefulness to sleep is facilitated); (2) the *sleep-efficiency* cycle (i.e., when the same amount of sleep provides higher recuperative value); and (3) the *sleep-inertia* cycle (note that these three cycles are not necessarily synchronous).

One particularly important finding concerns adaptation to these schedules: studies that allowed gradual transition over several days from monophasic to polyphasic patterns usually found an increased adaptive response from their subjects. Indeed, throughout the studies sleep efficiencies gradually increase and subjective and objective ratings of sleepiness decrease. This is the opposite from what one would expect if subjects were not adapting to the schedules. However, the preliminary data presented appears to indicate that, especially in the regimens where dramatic sleep reductions occur, at least 1-2 wk may be necessary before subjects become optimally entrained to the schedules.

What are the most important factors that may affect the benefits of naps taken during extended work, thus determining the criteria to design optimal polyphasic sleep schedules? In other words, which parameters correlate with an individual's ideal sleep episode duration? Combining what Naitoh et al. (1982) and Webb (1985) had already speculated upon, the following six factors were thought by these authors to be important for determining the timing and the minimum amount of sleep necessary to maintain human performance:

1. The length of wakefulness prior to the sleep episode
2. The duration of the nap period
3. The circadian placement of the nap
4. The elapsed time between the end of the nap period and the beginning of postnap performance (this affects the magnitude of sleep inertia)
5. The circadian time of task performance
6. The nature of the performance task

The findings reviewed and discussed in this chapter confirm that such factors play important roles in modulating nap recuperative power. However, a number of additional important modulating factors have emerged from these studies. They are:

7. The sleep architecture (e.g., sleep stages, stage time, delta wave time, sleep latencies)
8. The sleep stage (state) at nap termination
9. The subjective evaluation of the quality of the sleep episode

10. The cumulative sleep debt (i.e., the amount of sleep deprivation relative to the person's normal sleep amount) at the time of the nap
11. The performance and alertness levels immediately prior to the nap
12. The type of activity prior to the nap (e.g., mental/physical work, relaxing)
13. The type of schedule (e.g., polyphasic, semipolyphasic, the degree of sleep fragmentation, its timing)
14. The regularity of the schedule (i.e., whether nap times are kept constant throughout the schedule)

In conclusion, this review showed that sleep under polyphasic schedules "cannot be accounted for exclusively within the framework of what we know about monophasic nocturnal sleep" (Moses et al., 1975). It is also clear that just a few of the questions raised in Chapter 1 of this volume have been answered by the studies reviewed here. Such studies have created many more new questions than those that have been answered. It appears —and it is hoped —that studies adopting the polyphasic and ultrashort sleep paradigms may provide important information on our theoretical knowledge of the sleep system and its functions.

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11

Leonardo da Vinci and Ultrashort Sleep: Personal Experience of an Eclectic Artist[1]

GIANCARLO SBRAGIA[2]

The experience I am going to relate had a particular importance in my life, as I will now explain. At the end of the 1950s, when I was about 30 years old, I became convinced that I was a genius and that demonstrating my genius was only a matter of time. Besides my professional activity in theater, films, and television, my interests were broad; I had also studied music and had a great passion for painting. My activities were very diversified and I felt Leonardesque. I was so intrigued by the phenomenon of sleep, and especially by the waste of time that it represented, that I became obsessed by the possibility of reducing its amount.

Claudio Stampi asked me how I found out about the sleep-reduction method developed by Leonardo da Vinci. This is an obscure point, but perhaps it helps to explain how legends are born. That is, legends obviously settle in one's mind by subliminal stages. I cannot recall exactly where or from whom I gathered information about Leonardo's sleep habits. At that time I had a friend who was a medium and capable of extrasensory perception. She helped me with this experiment. It was probably from her that I learned about the peculiar Leonardo sleep-wake pattern, even though today, 30 years later, I am not completely sure. What *is* certain is that she taught me some yoga techniques in order to be able to reach a state of quasi-perennial insomnia: my goal was to sleep for 15 min every 4 hr, as Leonardo is supposed to have done.

I then started the experiment which, I must admit, turned out to be an extremely difficult task at the beginning. However, by the end of 20-25 days

[1]This lecture was translated from the Italian by Federico Mariscotti and Diana Bombelli.

[2]Giancarlo Sbragia is a playwright and actor who lives and works in Rome.

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I eventually succeeded in adapting to the schedule and achieved my goals. I equipped myself with an alarm clock that I regularly reset every 4 hr. When the alarm sounded, I immediately interrupted any activity I was doing, lay down, and tried to sleep. This task was not as easy as it may appear: it is quite complicated in fact to artificially interrupt normal activities every 4 hr, especially when you do not feel the need for sleep or rest. Very helpful in this regard was a relaxation technique that I decided to adopt, a slow induction of complete muscular relaxation. I used to lay down holding a stone in my hands. When the stone fell on the floor, even though I had not consciously opened my hand, I knew that at that point I had achieved the total relaxation I was aiming for.

Obviously, I had to work out a schedule that included naps during the most appropriate times of day, compatible with my professional needs. If I remember correctly, my napping schedule was set at 0400, 0800, 1200, 1600, 2000, and 2400 in order to allow me enough time to perform on stage. I did not want to be obliged to suddenly interrupt the second act of a play and announce to my audience, "Please excuse me, I'll take a catnap and come back in 15 minutes!"

As I mentioned earlier, this schedule was really difficult to follow at the beginning, and it required a great deal of self-discipline. It took about 3 wk to get used to it. But I soon reached a point at which I felt a natural propensity for sleeping at this rate, and it turned out to be a thrilling and exciting experience. I had stepped into a new world.

Actually, what did 15 min of sleep every 4 hr really mean? It meant a total of 1.5 hr of sleep per day! This was absolutely amazing to me: time increased. This meant that Leonardo did not live for 90 years only, but for a total of 450.[3] I am convinced that this might explain his ingenuity and great achievements.

How beautiful my life became: I discovered dawns, I discovered silence, and concentration. I had more time for studying and reading—far more than I did before. I had more time for myself, for painting, and for developing my career. The first months were really exciting.

However, the world around me was asleep, and was not concerned about what I was feeling or doing. My excitement lasted for about 5–6 months, at which time I started to have a few doubts. It is true that I could read more, that I could paint and play music, but I reached a point where it was just not possible to have enough activities to fill the 22.5 hr, day after day. My house was silent, my children did not pay attention on me, nobody did. I was suffering from loneliness: this was the truth.

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[3]Although there is no agreement among Leonardo experts concerning the date of his death, documents suggest that he died at age 67. Sbragia's calculations are therefore exaggerated: even assuming that Leonardo slept for only 1.5 hr per day for his entire life, allowing him 22.5 hr of wakefulness instead of the conventional 16 hr, this would have given him an equivalent of about 94 "years" of life on a normal sleeping schedule (67 yr at 22.5 hr = 94.2 yr at 16 hr [c.s.]).

It may be interesting to note that from a physiological point of view I was feeling very good, which I think demonstrates that it is perfectly possible to follow such a sleep pattern without any stress whatsoever. My experiment lasted 6 months, an appropriate length of time, I believe, to prove that, indeed, it is possible for a normal person to follow this sleep strategy. Nevertheless, I experienced damages of a different nature which, I would like to stress, are very personal considerations and not scientific observations. After a few months I felt psychologically a wreck, because due to this experience I eventually had to admit to myself that I was not the genius I had thought I was. I was not like Leonardo da Vinci. I started questioning myself and realized that time is something that is not lacking at all in a man's life: on the contrary, there is plenty of time available.

I also experienced a strange sensation that I only understood later: I missed dreams. I am a dreamer; I don't remember a single night in my life without the recollection of dreams; I have always dreamt. Sleeping without dreams was totally unknown to me. Lack of dreams — *not lack of sleep* — restricted me, restricted my imagination. It was evident to me that I needed the subconscious input and production represented by dreams. As this nourishment was missing, my imagination and my artistic activity started to suffer. I felt like I was using the power of a battery without ever recharging it (not a physical battery, but a sort of creative one). I was suffering a kind of imaginative damage, due primarily to lack of dreams. My oneiric self was totally eliminated. I therefore decided to interrupt my experiment.

I then went back to sleeping for 8 full hours. I recovered my dreams and, best of all, I was at peace with myself because I now recognized that my ingenuity was not so great as to require more time than what I had already received from life and from my own physiology.

I do realize how useful this method could be to those people dealing with technical or scientific tasks, for in such cases it may be possible to schedule one's activity quite precisely. I also think the sleep schedule provides an explanation for the technical and manual skills in which Leonardo was a master. As part of the discipline of reducing sleep to a minimum, it would be necessary to cultivate one's own manual skills and engage in manual activities. Artistic invention, on the other hand, has secret times and hidden channels, where sleep certainly has an important role with its oneiric component.

I believe that a strong will could enable someone to overcome normal sleep schedules and to acquire this new habit, without any physiological problem. The problems I had to face were of a personal nature: this experiment obliged me to look at myself in the mirror in a way that made me learn more about me in just 6 months than I might have learned in my whole life. After this experience I learned how to pace myself according to my own rhythms, not only for sleep, but also for working, eating, and other drives.

The experiment was like a long analysis session that forced me to

reconsider myself with humility and in a more objective way. Today, because I am no longer 30 years old but twice that age, I am intrigued by the idea of repeating the experiment. This interest has been stimulated thanks to the interest that this topic has for science. I would now approach it with a totally different attitude, because I no longer have anything to prove. Today, a reason for trying the experiment again is that perhaps this would help me to push away and get through a step that everyone has to deal with: death. It would be like undergoing another long psychoanalytic session to help myself to deal with this unquestionably obligatory passage of one's own existence.

12

What Is the Limit for Prolonged Sleep Reduction? An Objective Evaluation of the Leonardo da Vinci Ultrashort Sleep Strategy

CLAUDIO STAMPI

> O dormiente, che cosa e' sonno? Il sonno ha similitudine colla morte. O perche' non fai adunque tale opera, che dopo la morte tu abbi similitudine di perfetto vivo, che vivendo farsi col sonno simile ai tristi morti?
>
> —Leonardo da Vinci, *Codice Atlantico, 76 v.a.*
>
> Non vale fortuna a chi non s'affatica. Perfetto don non s'ha senza gran pena. Colui si fa felice, [ch]e vertu' investiga.
>
> —Leonardo da Vinci, *Windsor Castle, 12349 v.*

Leonardo da Vinci is a symbol of universal, interdisciplinary wisdom. With his tireless sense of curiosity, Leonardo explored more disciplines than any of his contemporaries. These disciplines included, among others, mathematics, mechanics, ballistics, anatomy, medicine, botany, geology, astronomy, and visual arts. Despite his own prolific writings and the multitude of scholarly works that have since been written about him, however, many aspects of his life remain a mystery. The legends circulated about his inventions and discoveries in all disciplines are numerous, as are postulations concerning his work patterns and life-style (Brion, 1959).

One of his secrets, or so it has been claimed (Sbragia, Chapter 11), was a unique sleep formula: he would sleep 15 min out of every 4 hr, for a daily total of only 1.5 hr of sleep. Therefore, it appears that he was able to gain an extra 6 productive hours per day. By following this unique regimen, he "gained" an additional 20 years of productivity during his 67 years of life. Perhaps this could explain, aside from his genius, the vastness and richness of his work.

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Leonardo da Vinci's Enigma

How much of this theory can be documented? Indeed, can any documentation be found in Leonardo's numerous writings to support this notion, or is it one of the many legends that have been perpetrated about him? Several specialists have been questioned about this aspect of Leonardo's life, all of whom concur that concrete proof does not exist (e.g., C. Pedretti, personal communication; P. Romano, personal communication). But it is also true that the same experts note that what is known for certain about Leonardo is very limited.

What is known today about Leonardo's personality suggests that the hypothesis that he may have "invented" some sort of sleep-reduction strategy may not be unrealistic. A few among these possible "clues" will be briefly introduced:

1. The prolific productivity and capacities of Leonardo appear unlimited, and at times beyond human ability; it is not unlikely that he adopted some strategy to allow for such a high level of productivity.
2. Among the several hypotheses that one can postulate, one could suggest that Leonardo did not adhere to this extreme sleep pattern continuously, but only during certain periods of sustained work. For example, we know that Leonardo's in-depth studies of anatomy and the marvelous drawings he produced were the result of dissection and study of at least 30 cadavers (McMurrich, 1971). We also know that during Leonardo's time the techniques for preservation of biological tissues were very primitive, and that anatomists had to work fast. It is not unreasonable to hypothesize, therefore, that Leonardo adhered to a regimented formula of drastic reduction of sleep in order to maximize his hours of work before the bodies on which he was working decomposed.
3. Leonardo was apparently a practical individual; many of his works appear oriented toward action. He was also a solitary, meticulous, and rigorous personality ("hostinato rigore"). Undoubtedly, such a sleep pattern requires sacrifice, self-discipline, and probably a solitary life-style.
4. Leonardo was an insatiable observer; for example, when he needed to paint a smile he would first try to understand in depth the muscles used to smile (in fact, *La Gioconda* occupied him for so long that it was never delivered to the person by whom it was commissioned). Leonardo also dedicated a great deal of his time to the observation of animals' life. He could well have asked himself why polyphasic sleep is so common in nature, up to the point of subjecting himself to the same type of sleep patterns.

The interest to explore in depth what could appear as one among several bizarre aspects of Leonardo's life is not only a historical one. As many chapters throughout this volume indicate, several chronobiological, evolutionary, ontogenetical, and other issues strongly suggest that adult humans

can adapt to polyphasic sleep patterns. Three specific issues suggest that the putative "Leonardo schedule," although apparently very extreme, makes considerable biological sense, and that it may be worth exploring. These issues will be briefly summarized here (to avoid repetition, the reader is kindly invited to address a more detailed discussion on these and other related issues in Chapter 1, 10, and 13):

1. Chronobiological studies have shown that there are several "gates" throughout the 24-hr span during which transitions from waking to sleep, and vice versa, are facilitated. Among such circadian and ultradian pulses of sleep-wake transition, a 4-hr cycle in sleep propensity has been recently described (Nakagawa, 1980; Zulley and Bailer, 1988).
2. Studies conducted under a variety of quasi-continuous work situations have demonstrated that short naps appear to have robust restorative power on functioning (see also Chapters 1,9, 10, 13, and 14). When *some* sleep is permitted, a significant improvement in performance is normally observed; such performance increments are often disproportionate to the length of the sleep episode. Studies conducted in the lab (e.g., Bonnet, 1986) and in the field (Stampi, 1985, 1989) suggest that approximately 10 min appears to be the minimal duration required for sleep to be restorative.
3. Although no systematic study has yet assessed a direct relationship between the levels of sleep duration and fragmentation, and the habitat demands of a certain species, the general impression is that the greater the struggle for survival in an animal's habitat, the more polyphasic its sleep and the shorter its total duration [e.g., polyphasic and short sleep patterns in the blind Indus dolphin (Pilleri, 1979), and in the giraffe (Kristal and Noonan, 1979); see also Chapters 1 and 2]. That is, under conditions of extreme demand and danger, polyphasic sleep may be more efficient.

Yet, there is one important issue that is in apparent striking contrast with what has emerged in the last 30 years of sleep research. A large body of evidence suggests that if sleep is gradually reduced in amount, most individuals can maintain normal and satisfactory levels of functioning with 60-70% of their usual amounts of sleep (i.e., 4.5-5.5 hr of sleep per day) for relatively prolonged periods (Friedmann et al., 1977; see also Chapters 10 and 13 for a discussion on sleep-reduction strategies). Horne (1988) speculates that "core sleep" typically occupies "the first three cycles —the initial 4-5 hr of sleep" (p. 180), and is mainly identified as all slow wave sleep (SWS) or delta activity and about half REM sleep. It is quite clear that 1.5 hr of sleep per day, which is the only amount allowed by the Leonardo da Vinci schedule, appears too short based on what is currently known about minimal sleep requirements. However, most documented studies of sleep reduction have investigated curtailment of *monophasic*, usually nocturnal, sleep. Is *polyphasic* sleep the "trick"? That is, is polyphasic sleep a strategy that may allow individuals to reduce sleep need to amounts considerably shorter than what is claimed to be "core sleep," analogous to

what appears to occur in certain mammalian species living in dangerous environments?

This challenging question, in itself, would be sufficient to justify the importance of further investigation of Leonardo's strategy. Until recently, Sbragia's (Chapter 11) unique 6-mo experiment constituted the only reported trial of Leonardo's sleep cycle, and his observations suggested that it may be a feasible strategy. The report clearly points out that sleep reduction itself was not a problem. Unfortunately, this experiment was not documented by any of the objective techniques currently available in sleep research. It is not known to what extent performance, the physiological processes, and the biological rhythms of Sbragia where affected by this schedule.

In order to investigate the hypotheses raised above and to further understand the sleep architecture of ultrashort sleep, as well as the fundamental components of "core sleep," a series of studies inspired by the putative Leonardo da Vinci schedule and Sbragia's personal experience were recently conducted (Stampi et al., 1990; Stampi and Davis, 1991) as part of a long-term research program on polyphasic and ultrashort sleep. Three studies have been conducted, and results from the first two will be reported here.

The First Study: A Graphic Artist Intrigued by Leonardo's Schedule

The study was facilitated by the curiosity of another artist, Francesco Jost (FJ), who became intrigued about Leonardo's schedule and volunteered to become a subject for the experiment (Stampi et al., 1990). FJ is a healthy individual with normal sleep architecture and no sleep disturbances. He was age 27 at the time of the first trial (1989). He was a nonnapper and typical monophasic sleeper (0000 to 0800). He was never involved in shift work. He is a graphic artist who lives and works in Ticino, Switzerland. He became intrigued about this schedule because he believes that the sleep-reduction technique may be a means to enhance or modify the quality of his creativity, and because he would like to use such a technique periodically, to be more productive during periods of intense activity.

The first week of the study was dedicated to a gradual reduction and fragmentation of the subject's sleep pattern. FJ's habitual 8-hr sleep was initially divided into 6 episodes of 80 min each, one every 4 hr (the *transition* phase; see Figure 12.1). These 6 daily sleep episodes were subsequently reduced from 80 to 15 min over 6 days, by means of an exponentially decaying function (i.e., nap durations were 80 min on day 1, 61 min on day 2, 46 min on day 3, and so on to 15 min on day 7; the *reduction* phase). During the last 9 days of the study (the *ultrashort* phase), 15 min of sleep were scheduled every 4 hr, providing a total of 1.5 hr of bed time per day.

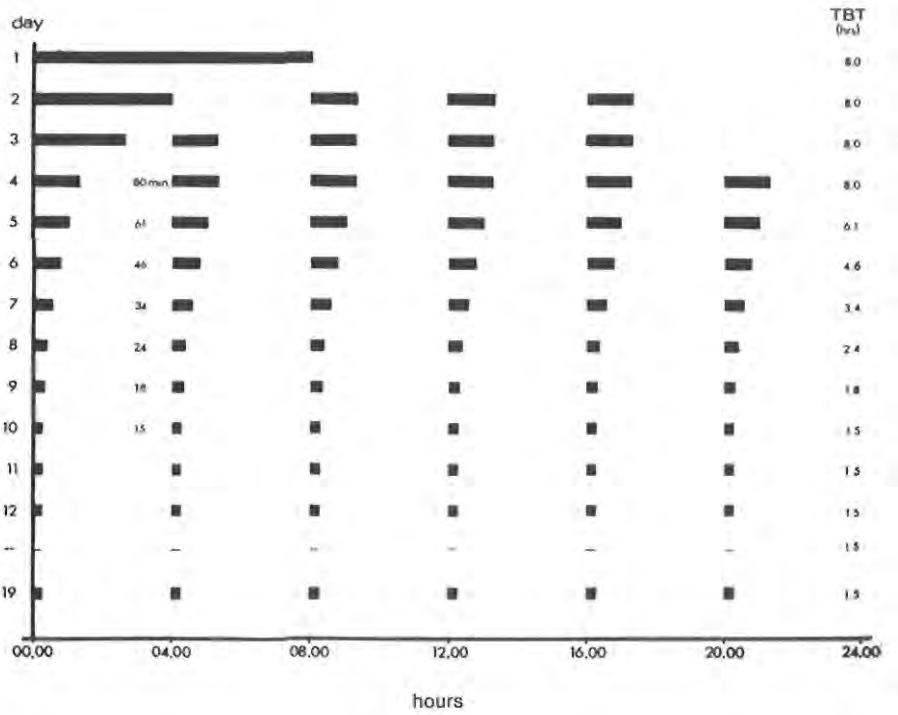

FIGURE 12.1. Sleep-wake schedule followed during the 19-day "Leonardo da Vinci" schedule (black bars: sleep periods). Days 2–4: transition from monophasic to polyphasic sleep; days 5–10: nap reduction from 80- to 15-min; days 11–19: 1.5 hr sleep per day taken as six 15-min naps.

During the whole experiment the schedule was coordinated by a computer program that would wake up the subject and alert him about going to bed at the appropriate times. FJ was awakened by the computer's alarm with intensities progressively increasing from 30 to 90 dB, at 10-dB steps every 6 sec. Performance, alertness, and mood levels were periodically assessed throughout the study (approximately 20 times per day) by means of the same computer program. These 6-min test batteries were done 20 min before the nap, upon arousal, and at 30 and 60 min after arousal. EEG, EOG, EMG, EKG, and tympanic temperature were ambulatory recorded for a total of 17 days with Oxford Medilog (UK) 8-channel units. FJ continued his professional activity during the whole experiment, although it was obviously frequently interrupted by performance testing and napping episodes.

Sleep infrastructure for the 50 naps that were recorded during all but one of the 9 days in the *ultrashort* phase, including naps that occurred at unscheduled times, was analyzed and compared to baseline and day 1 recovery sleep conditions. As could be expected, FJ was not able to follow strictly such an extreme schedule. Nevertheless, he achieved remarkable

levels of sleep reduction: after the first few days on the Leonardo schedule, and following occasional oversleeping for durations longer than 15 min, it was concluded and mutually agreed that his preferred sleep episode durations were slightly longer, namely, of 25 min instead of 15. The schedule was then changed accordingly.

The mean daily total sleep time (TST) during the *ultrashort* sleep period was 2.7 hr (see Figure 12.2), that is, only 38% of TST recorded at baseline (7.2 hr). Sleep episodes showed a median duration of 12.9 min, with a mean of 26.3 min (SE = 6.9). Figure 12.3 displays the sleep episode duration (SED) frequency distribution. Mean nap sleep efficiency [(stages 2 + 3 + 4 + REM)/(total bed time)] of 70.1% was considerably higher compared to previous polyphasic sleep studies (usually at about 50%; see Chapter 10). This occurred probably due to the longer duration of this study compared to previous studies, which may have allowed greater adaptation to the schedule; and the very short allowed TST, which may have considerably increased sleep pressure, and therefore reduced sleep latencies.

Indeed, latencies to sleep from lights-out time were considerably reduced. Baseline means of 16.6 (to stage 1) and 21.5 min (excluding stage 1) decreased to nap means of 5.5 and 9.4 min, respectively. FJ was able to fall asleep during basically all scheduled sleep periods, although he was not able to reach sleep depths beyond stage 1 in 9 naps (18%), with no apparent time-of-day effect. The mean duration of stage 1 in such stage 1–only naps was 3.8 min. The nap mean overall percentage composition of stages 1 (18.9%), 2 (32.8%), SWS (27.4%), and REM (20.9%) was very similar to that of baseline sleep (13.5, 38.6, 26.1, and 21.8%, respectively; see Figure 12.2). The total daily amounts of each stage, however, were considerably and proportionately reduced.

Despite such striking percentage similarity, nap sleep structure was radically different from baseline monophasic sleep. REM sleep and SWS

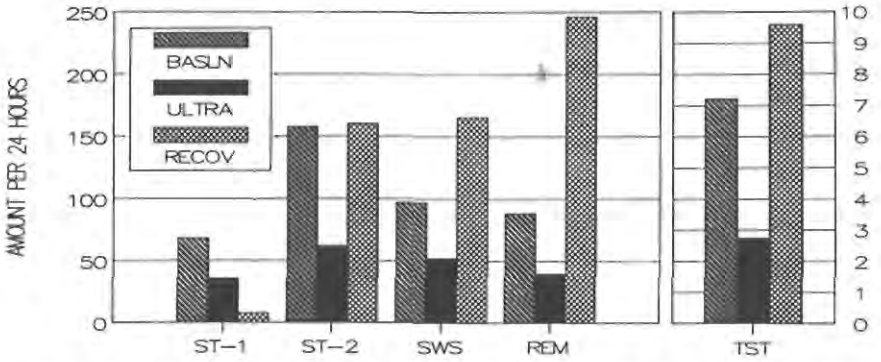

FIGURE 12.2. Sleep architecture during the 19-day "Leonardo da Vinci" schedule (baseline, ultrashort, and recovery sleep periods).

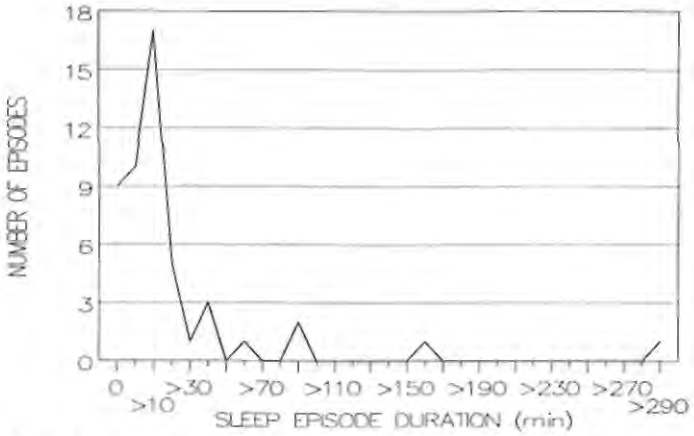

FIGURE 12.3. Number of sleep episodes as a function of sleep duration (19-day schedule).

rarely occurred together. This happened for only 8 naps, during most of which the subject overslept beyond the scheduled 25 min. When REM sleep was present, it showed the usual circadian effects, with peaks between 0000 and 1200. REM sleep frequently occurred in close proximity to nap onset, generating 15 sleep onset REM episodes (SOREMPs) out of 19 naps with REM. Mean SWS latency was 10 min. SWS was present in 50% of all naps.

During the scheduled waking intervals, FJ experienced a total of 6 episodes of drowsiness (stage 1 sleep; mean duration: 3.5 min, SE:0.97) and 5 unscheduled sleep episodes (mean SED: 36.2 min, SE:12.9). There was a circadian effect of sleep pressure: all except one of these 11 episodes were initiated between 0630 and 1530. This occurred especially in the first half of the study; the subject's attempts to fight sleepiness by trying to keep active were generally successful and led to good adaptation during the second half of the study. However, FJ's most difficult task was not to stay awake, but to wake up at the alarm: awakening was delayed by more than 6 min in 11 naps (mean delay: 69.3 min, SE: 23.9, max.: 285 min) with no noticeable time-of-day effect. Day 1 recovery sleep showed a TST of 9.6 hr, with marked increase of SWS (+71%) and particularly of REM sleep amounts (+179%) relative to baseline (see Figure 12.2). Stage 1 sleep was virtually absent.

Previous findings from sleep reduction studies have shown that total amounts of SWS are usually preserved, and that only REM and stage 2 sleep are penalized. Contrary to such findings, in this study *all* sleep stages were proportionally reduced. This may suggest that while over the short term part of REM and perhaps most stage 2 sleep can be reduced to the advantage of the supposed "obligatory" SWS, this may no longer be true in the long term. This study was in fact longer than previous studies (FJ spent

a total of 17 days in the combined transition-reduction-ultrashort sleep schedule). Even by adding the sleep amounts recorded during recovery sleep to what obtained during the 9 days in the ultrashort sleep condition, FJ cumulated only 65.2% of the SWS (and 68.4% of the REM) one would expect him to experience should all SWS (or all REM) be obligatory (based on baseline SWS and REM sleep amounts). Hence, in this study all sleep components appear to be important: not only SWS but also REM (and stage 2 sleep, to a lesser extent) showed similar probabilities of occurrence.

FJ appeared to adapt objectively and subjectively surprisingly well to such an extreme schedule. His performance levels during the polyphasic sleep period were comparable to baseline levels measured prior to the study. Before analyzing performance behavior, it should be noted that one of the most convincing indexes of the subject's high level of adaptation to the schedule was his spontaneous reaction at the end of the study. He immediately volunteered for another study in which the same schedule would be undertaken for a prolonged period.

The Second Study: Forty-Eight Days with 3 Hours Polyphasic Sleep per Day

One year after termination of the first study, FJ returned to the laboratory and, while continuing his professional activity throughout the experiment, followed a polyphasic sleep schedule for 48 days (Stampi and Davis, 1991). This time it was agreed that the target sleep episode duration would be similar to what he had achieved in the first study (mean: 27 min per sleep episode), and therefore a SED of 30 min was selected. This would allow him a total of 3 hr bed time per day. All the other aspects of the original protocol remained identical or very similar. That is, after 2 days of *baseline* sleep recording (baseline, 0000-0800), 6 days were dedicated to the *transition/ reduction* from 8 hr/day monophasic sleep to 3 hr/day polyphasic sleep (see Figure 12.4). In the remaining 39 days of the study, the subject followed a polyphasic schedule consisting of six 30-min naps per day initiated at 0000, 0400, 0800, 1200, 1600, and 2000 *{ultrashort* phase). In order to evaluate the recovery effects of extended sleep on subsequent sleep and performance, on day 34 (two-thirds of the way through the ultrashort phase) and without prior notice, the subject was allowed 1 day (24 hr) of ad lib sleep, after which he resumed the polyphasic schedule. During the ad lib period FJ actually slept for a total of about 10 hr.

Results from the analysis of overall prenap performance (6 measures per day) on the Descending Subtraction Test (DST, Evans and Orne, 1976) and on the Memory and Search Test (MAST, Folkard et al., 1976) (number of correct responses during a fixed time) are compared to mean baseline performance levels. Figure 12.5 shows the mean performance values for

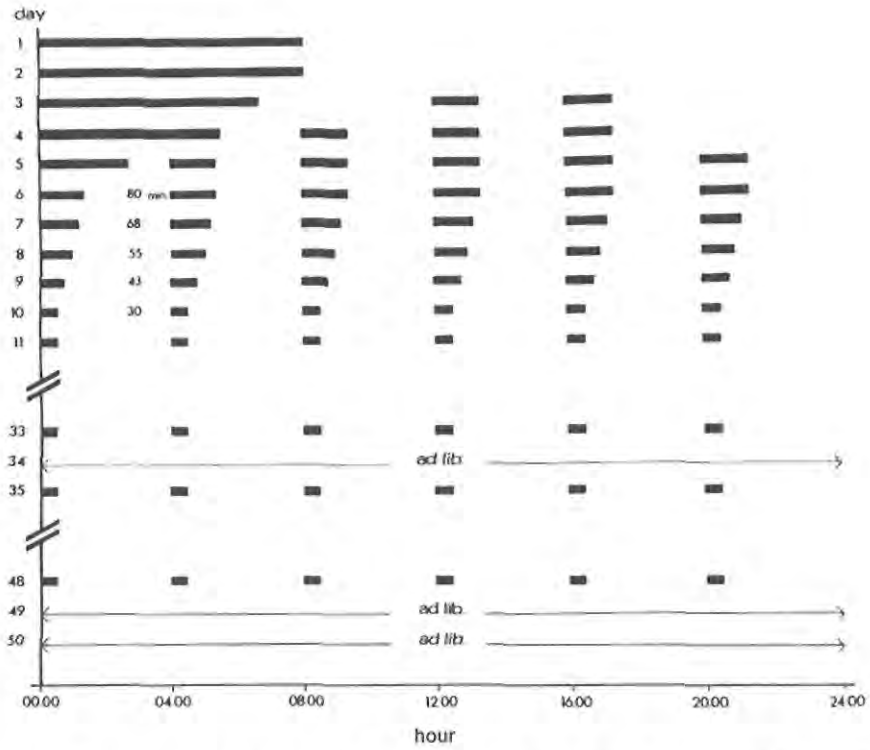

FIGURE 12.4. Sleep-wake schedule followed during the 48-day "Leonardo da Vinci" schedule (black bars: sleep periods). Days 3–6: transition from monophasic to polyphasic sleep; days 6–10: nap reduction from 80 to 30 min; days 10–33 and 35–48: 3 hr sleep per day divided into six 30-min naps; days 34, 49, and 50: ad lib. sleep.

baseline, for the transition period (days 3–9), and for the ultrashort period grouped by *"weeks"* of 6-days each. MAST performance (upper graph) showed a slight decrement relative to baseline throughout days 3–21, followed by an improvement above baseline levels thereafter. DST performance (bottom graph) showed a modest but more prolonged decrement throughout days 3–33, followed by a sharp increase in performance above baseline levels immediately after the ad lib sleep day.

None of these values were significantly different from baseline levels. The initial statistical tests were fixed one-way ANOVAs over 4 conditions (baseline, transition, ultrashort, before and after the ad lib sleep period) and 8 conditions (baseline, transition, and the 6 "weeks" in the ultrashort period). Post hoc comparisons were made using Tuckey's Honest Significant Differences Test for unequal sample sizes. The test showed no significant differences between baseline and any of the "weeks" in the *transition* and *polyphasic* conditions. The only significant difference (at p

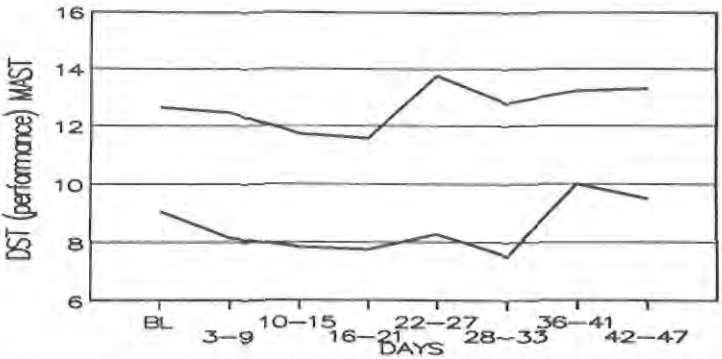

FIGURE 12.5. Performance during the 48-day "Leonardo da Vinci" schedule grouped by "weeks" of 6-days each. *Top:* Memory and Search Test (MAST); *bottom:* Descending Subtraction Test (DST).

$< .05$) was for DST toward the end of the experiment ("week" 7, i.e., days 36–41) being better than "weeks" 3, 4, 5, and 6.

Summarizing the findings from the overall performance analysis, the decrements observed in MAST (2 initial "weeks") and DST (4 initial "weeks") performance relative to baseline were modest and not significant. One could have expected a much poorer performance throughout the study as a result of the extreme level of sleep reduction (3 hr sleep/day) and fragmentation for such a prolonged period. It is difficult to interpret the improvement in MAST performance after day 21 and prior to improvements observed in DST performance; this may be indicative of FJ's adaptation to the ultrashort sleep schedule, and may also further confirm that the DST is a test particularly sensitive to sleep-deprivation and sleep-inertia effects (cf. Dinges et al., 1988). The sharp improvement of DST performance by day 36 appears to indicate that only one day with ad lib sleep was capable of producing remarkable beneficial effects, which were maintained throughout the remaining days of the study. This was also confirmed by examining FJ's subjective assessments of mood and sleepiness.

In conclusion, the subject appeared to adapt surprisingly well to such an extreme polyphasic sleep schedule during both studies, especially considering that he slept for a total of less than 3 hr per day (divided into 6 episodes) for two periods of 19 and 48 days each. It could be argued that chance might have provided a "special" subject, who was either exceptionably adaptable to marked reductions in total sleep time, or to polyphasic sleep. He may be one of the rare natural short sleepers, or perhaps belongs to a new "breed" of natural polyphasic sleepers.

Conclusion

To verify these possibilities more studies are necessary; they are currently being undertaken. At the time this chapter is being written, FJ concluded the third trial of this series (Stampi et al., 1992), which consisted of a control study (monophasic vs. polyphasic) for the first experiment. For a period of 3 wk, he slept for exactly the same total time averaged in the first study, but this time all his sleep was taken in one single monophasic nocturnal episode of less than 3 hr. Thus, each day he was kept awake for slightly over 21 hr uninterruptedly. Other volunteers have been or are currently being submitted to variants of the polyphasic ultrashort sleep schedules described previously.

The implications of what emerged from this series of studies are discussed in greater detail, within the broader context of a review on other polyphasic sleep studies, in Chapters 1 and 10. In brief, findings described in this chapter do not appear to contradict the following hypothesis:

1. Adult humans appear to have a natural ability to adapt to polyphasic sleep schedules.
2. The 4-hr ultradian cycle of sleep-wake pressure previously described may be an important factor in allowing adaptation to polyphasic patterns.
3. The sleep-wake system appears to show a high level of flexibility in terms of sleep timing and duration.
4. Polyphasic sleep may be a feasible, and perhaps the *only*, strategy allowing remarkable levels of sleep reduction during prolonged quasi-continuous work situations, without unduly compromising performance effectiveness.
5. This may be analogous to what is observed in a considerable number of mammalian species, particularly in those living in dangerous environments.
6. Further studies extended to a larger sample of subjects may provide powerful tools for developing sleep-wake schedules for individuals involved in irregular or quasi-continuous work situations.
7. These findings and hypothesis raise challenging questions concerning what is known about the regulatory mechanisms of sleep function.

Leonardo was unquestionably one of the most ingenious and enigmatic personalities that has so far appeared in Western European culture. Yet the aim of the project described in this chapter is not solving one of the many mysteries of Leonardo's life. This interesting dilemma will be left to historians. Most important for sleep researchers, it is expected that these studies may produce valuable insights into the function(s) of sleep. Nevertheless, it would constitute a deserved tribute to Leonardo if one day the knowledge that may emerge from studies partially stimulated by one of his

original ideas could be used by a wider variety of "normal" individuals, who may not need to be endowed with a "Leonardesque" genius to benefit from his secrets.

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IV

Irregular Schedules, Sustained Operations, and Napping

13

Minimal Sleep to Maintain Performance: The Search for Sleep Quantum in Sustained Operations

PAUL NAITOH

Sleep Quantum

The purpose of this chapter is to review some of the current research findings that have contributed to determining the minimal amount of sleep needed to maintain a high level of task performance for days, weeks, and even months.

The least amount of sleep, the "sleep quantum," necessary for efficiently doing a day's work, shows large differences between individuals (Kripke et al., 1979; Webb, 1985a; White, 1975), and also varies considerably within each individual. However, the results of acute partial-sleep-deprivation studies by Wilkinson (1970), Wilkinson et al. (1966), and Haslam (1982) indicate that the amount of sleep necessary to prevent performance impairment on vigilance tasks is 4 or more hours of sleep daily. The results of chronic partial-sleep-deprivation studies have similarly shown that 4.5-5.5 hr of sleep per day are necessary if one wishes to cause no major undesirable behavioral consequences for up to 8 mo (Friedmann et al., 1977; Webb and Agnew, 1974).

It is currently believed that the 4.5-5.5 hr of sleep should be taken continuously (i.e., they should not be fragmented) in order to benefit from maximal recuperation (Bonnet, 1986; Downey and Bonnet, 1987; Levine et al., 1987; Magee et al., 1987). This Continuity Theory of Sleep (see Bonnet, 1985, 1986) implies that a sleep "quantum," the smallest fundamental quantity of sleep, is an uninterrupted 4.5-5.5 hr of sleep.

A search of the literature on sleep quantum, however, reveals field observations that show that short sleep of much less than 4.5-5.5 hr, for example, as short as a few tens of minutes, can help subjects recuperate from fatigue and reduce sleepiness (Stampi, 1985a,b, 1989a,b). Stampi's

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field observations are supported by laboratory studies reporting benefits over task performance with even very short naps (Dinges, 1983, 1989; Dinges et al., 1985, 1986; Haslam, 1982; Naitoh, 1981). In fact, very short naps are often observed in daily routines. Stossel (1970) reported that about one-fourth of the fourth-year medical students he observed napped 3 min or longer during a 1-hr lecture starting at 0830. During a particularly dull lecture, over half of the students napped. Wedderburn (1987) reported on short naps, some lasting only 5 min, taken by shift workers at work sites. He suspected some qualitative difference in these urgent short naps compared with much longer sleep usually taken on a bed. Recent studies, primarily focused on modeling the effects of sleep apnea on performance, mood, and sleep, suggest that a fragmented nocturnal sleep consisting of many short sleep episodes, ranging from 4 min to 2.5 hr, can offer as much recuperation as that expected from much longer, continuous sleep (Bonnet, 1986; Magee et al., 1987). These observations and studies indicate that sleep quantum could be of a magnitude of tens of minutes, not 4.5-5.5 hr in duration.

Core versus Optional Sleep

In determining the relationship between a minimal sleep and an acceptable level of job performance, sleep stages (Rechtschaffen and Kales, 1968) have often been discussed. Each of the sleep stages, such as stage 2, rapid eye movements (REM) sleep, or the combined sleep stages of slow wave sleep (SWS), could play significantly different roles in determining the amount of performance recuperation. Thus, an appraisal of sleep quantum could become very complex because the degree of recuperation after sleep is no longer simply a matter of sleep duration: the stages of sleep must also be considered.

Fortunately, the duration of each sleep stage, especially of SWS or REM sleep, appears to be unrelated to recuperation and performance maintenance (Lubin et al., 1974; Johnson et al., 1974). Lumley et al. (1986) studied the effectiveness of a morning nap (starting at 0900) of 0, 15, 30, 60, or 120 min in neutralizing increased sleepiness due to 1 night of total sleep deprivation prior to the nap. Changes in alertness were measured by the Multiple Sleep Latency Test (MSLT). They found that napping had alerting effects that were strongly related to the duration of the nap (reaching its highest level with a 60-min nap), and only weakly related to the sleep-stage composition. The sleep quantum is measured simply by time, and not by sleep stages or other transient events observed during sleep. In discussion of a minimal sleep, little reason exists for examining sleep stages.

In discussing sleep duration, Horne (1988, pp. 180-217) has offered a cogent argument for partitioning sleep into two kinds: core versus optional

sleep. *Core sleep* is defined as "the first three sleep cycles —the initial 4-5 hours of sleep" (p. 180) necessary for humans to function properly. *Optional sleep* is the remaining sleep that can be eliminated without dire consequences. Home's estimate of core sleep matches well with an estimate of the sleep quantum (4.5-5.5 hr) previously mentioned in this chapter.

Capturing Core Sleep

There are five ways to capture core sleep, that is, to satisfy a minimal sleep demand centering around 5 hr per day. They are:

1. To sleep for 5 unbroken hours
2. To adopt "anchor" sleep
3. To take "prophylactic" sleep
4. To have longish (1-4 hr) naps
5. To take "ultrashort" sleep

Continuous 5-Hour Sleep

The best way to capture the sleep quantum is to sleep continuously for 5 hr. Ordinarily, this is the sleep pattern practiced by almost everyone. However, in certain job environments that demand sustained work, a period of 5 uninterrupted luxurious sleep hours cannot be set aside because it conflicts with work schedules (Angus and Heslegrave, 1985; Dinges et al., 1986; Englund et al., 1985; Mullaney et al., 1983; Naitoh and Angus, 1989).

"Anchor" Sleep

The "anchor" sleep was introduced by Minors and Waterhouse (1981) (see also Chapter 7, this volume). They have divided an 8-hr sleep into two 4-hr sleep periods. One of the 4-hr sleep periods was taken at the same time each day (i.e., it was "anchored" to a local time). The fixed time should be selected to correspond to a time period that is both suitable for sleep after working days and socially acceptable during days off. For example, Minors and Waterhouse set anchor sleep at 0800-1200 for night-shift workers to sleep after work. During days off, they would sleep during the same time period of 0800-1200, leaving the entire afternoon available for social events —a socially acceptable schedule. The second 4-hr sleep period could be taken at irregular times. Minors and Waterhouse have observed that, as long as one 4-hr sleep period was anchored to a fixed time of day, the circadian rhythms became stabilized within a few days with periods indistinguishable from 24 hr. In terms of capturing core sleep, anchor sleep offers a degree of scheduling flexibility. Instead of finding a time spot that allows 5 continuous hours for capturing core sleep, only 4 hr of sleep must

be scheduled to be continuous and occur at a fixed time of day. However, the demands of some work schedules may not permit workers to have the luxury of sleeping even 4 unbroken hours during the fixed time period.

Prophylactic Sleep

Dinges and others (Dinges, 1983; Dinges et al., 1986; see Naitoh and Angus, 1989) proposed napping in anticipation of sleep loss (i.e., prophylactic napping), or sleeping longer-than-normal hours so as to "store" sleep. The idea of storing sleep by sleeping longer than usual before the start of a long work period is attractive. However, independent confirmation that a significant amount of sleep can be stored is not yet available.

Longish Napping

A more traditional way of getting some sleep during extended periods of work is to sleep whenever possible. In sustained-operation research, 1-4 hr naps were interjected during lulls in the work or whenever possible to find whether these longish naps were long enough to partially satisfy the need for core sleep (Angus and Heslegrave, 1985; Englund et al., 1985; Haslam, 1982; Mullaney et al., 1983; Webb, 1985b). However, even a 1-hr nap may turn out to be too long to be taken in some work environments with sustained operations.

Ultrashort Sleep

A view that sleep must be continuous to be efficient and recuperative, the Continuity Theory of Sleep, has been popular. Clinical observations of disrupted sleep among sleep-disordered patients often strengthened the validity of this view. It is believed that the more fragmented a sleep period is (i.e., the shorter the duration of each sleep episode), the more diminished its refreshing power.

However, in the remainder of this chapter, evidence will be offered to show a need to revise a concept of "continuity^ downward from 5 hr to a few tens of minutes. Then, the differences between "fragmented sleep" and "continuous sleep" will blur, as the time requirement for a sleep to be continuous becomes less and less.

Appraisal of Recuperative Power of Ultrashort Sleep

The recuperative powers of repeated ultrashort sleeps in maintaining performance efficiency has been brought into sharp focus by Stampi's dramatic field observations of the sleep patterns of yachtsmen during solo,

long-distance yacht races (1985a,b, 1989a,b). Taking as many ultrashort sleeps as necessary to obviate the need to sleep continuously for 5 hr or longer was shown to be possible, given that there was an opportunity to adapt to the life under ultrashort sleep.

Stampi's field research was not the first to reveal that short sleeps, or napping, were able to replace the major daily sleep period of 8 continuous hours. There are three kinds of research lines, each of which was initially designed to answer some specific questions unrelated to the usefulness of short sleep in capturing core sleep, but that also demonstrated that many periods of short sleep can capture core sleep. These researches are on fragmented sleep, sleep apnea, and the short day (e.g., a 90-min "day").

Fragmented Sleep

Husband (1935) tested 1 subject who slept 8 hr per night for 1 mo, then 6 hr per night in 2 sleep episodes, with one sleep period at 2300-0200, and the other at 0500-0800 during the second month. The time period between two sleep episodes was spent in various activities. Husband used tests of scholastic aptitude, intelligence, and psychomotor skills to determine if interrupted sleep caused mental deterioration as compared with continuous sleep. No consistent degradation of performance was observed due to interrupted sleep during or after experimentation of 1 mo. Hartley (1974) showed that a group of subjects who had three 80-min naps (at 2310, 0530, and 1225 each day) over 4 consecutive days was poorer in task performance than the control subjects who slept 8 continuous hours, but a higher level of performance was produced than by another group of subjects who slept 4 continuous hours between 0100 and 0500.

Sleep Apnea Model

Patients with sleep apnea experience extremely disrupted sleep marked with frequent arousals. In order to model behavioral consequences of severe sleep apneics, Bonnet (1985, 1986) conducted experiments with young, healthy, normal sleepers. In his 1986 paper, subjects experienced four kinds of sleep disruptions over 2 consecutive nights. The four disruption conditions were: (1) brief awakening (as defined by ability to make a verbal report of sleep-wake status or to solve an addition problem) after 1 min of accumulated sleep, (2) brief awakening after each 10 min of accumulated sleep, (3) the same after 2.5 hr of accumulated sleep, and (4) no sleep. In the morning, the subjects were given addition, vigilance, sleep latency, and other tests immediately after their awakening. The results showed that, after 2 nights of disrupted sleep, "periods of uninterrupted sleep in excess of 10 min are required for sleep to be restorative." That is, the sleep quantum is neither 1 min nor 4.5-5.5 hr, but perhaps about 10-plus minutes. However, there are significant differences in sleep stages between the 1-min

sleep and the 10-min sleep: the 1-min sleep had virtually no SWS and REM sleep, whereas the 10-min sleep contained more than one-half of SWS and REM sleep time in comparison with the baseline sleep. Thus, the restorative power of sleep of a 10-min nap may have to be attributed to the presence of SWS and REM sleep, in addition to the duration of sleep.

Downey and Bonnet (1987) included analyses of performance for 5 subjects who did a random two-digit/two-number addition problem given immediately upon awakening from 2 consecutive nights of disrupted sleep. They found that as early as night 1, awakening subjects every 1 or 10 min caused verbal response to slow down. During night 2, the 1-min disruption continued to cause additional marked slowing in verbal response to the addition task: "By night 2, response latencies on the average were 7 times control values in the 1-min condition, remained at 4 times control in the 10-min condition, and nearly 2.5 times control in the 2.5 hours condition" (p. 361). Since the duration of sleep stages were poor predictors of performance, Downey and Bonnet felt that "the data were best explained by sleep continuity theory, which posits that a period of at least 10 min of uninterrupted sleep is required for restoration to take place."

Magee et al. (1987) conducted a study on the extent of voluntary control of respiration during sleep. During a sleep-disrupted night, young, healthy, university students would take a deep breath to a tone presented every 1 or 4 min. Magee et al. measured effects of 1 disrupted night on sleepiness. They found that the subjects whose sleep was disrupted every minute lost almost all SWS and one-half of REM sleep, and slept only about 6 out of 8 hr of bed time. The 4-min disruption group did not differ from the control noninterrupted sleep group in terms of sleepiness. The results of this study by Magee et al. could be interpreted to show that the sleep quantum could be as short as 4 min, instead of 10-plus min as implied by Bonnet (1986).

Short Day

Most sleep studies have been conducted under the constraint of a day having 24 hr, with each day consisting of one sleep-wake cycle of 8 hr/16 hr. However, a few studies examine much shorter,' artificial days (e.g., 90-min "day") with results that appear to contribute to our understanding of sleep quantum.

Weitzman et al. (1974) studied the effects of a 3-hr "day" with a sleep-wake schedule of 60 min/120 min over 10 days. Total bed time under this sleep-wake schedule was 8 hr/24 hr. The polygraphic sleep records showed that the subjects slept an average of 4 hr/24 hr during the 10-day study. Sleep efficiency, as calculated by dividing the sum of sleep stages 2, 3, 4, and REM (i.e., total sleep time) by total bed time was 50.9%. No task performance data were collected in this study.

Carskadon and Dement (1975, 1977) studied a "90-min day." A sleep-wake schedule was 30 min/60 min for up to 6 (24-hr) days. The subjects

could sleep up to 8 hr in 16 sleep episodes, each 30-min long. On average, the subjects were able to sleep less than 4 hr/24 hr across 6 days. Sleep efficiency was 47.6%. No task performance data were collected. Carskadon and Dement noticed that sleepiness increased significantly on the first nap day but decreased to the baseline over the next 4 days, showing a sign of adaptation to the altered sleep-wake schedule.

Moses et al. (1975) and Lubin et al. (1976) examined the sleep-wake schedule of 60 min/160 min across 40 hr. This represented a total bed time of 6.5 hr/24 hr, but the subjects were able to achieve sleep efficiency of only 47.5%. Unlike other "short-day" studies, Lubin et al. (1976) used auditory vigilance, addition, and word memory tests and the Stanford Sleepiness Scale to measure the effects of the sleep-wake schedule that would certainly fragment sleep. The 60-min nap sessions were beneficial and neutralized performance degradation expected from 40-hr of total sleep deprivation.

Mullaney et al. (1983) reported comparisons of performance for three groups under varying sleep-wake schedules. One group was required to work continuously for 42 hr. The second group was required to work continuously, but they were given a 1-hr sleep/rest period every 7 hr (6:1). The subjects in this group repeated this sleep-wake cycle 6 times during the 42-hr continuous work period. The third group was required to work for 18 hr, given a chance to sleep for the next 6 hr (18:6), and then repeated this sequence twice. The authors found that the 6:1 group showed superior performance over the other two groups up to the first half of the 42-hr continuous work period. This was attributed to the benefits of the 1-hr naps. The 1-hr naps were not sufficient to maintain performance at the high level as observed among the subjects in the 18:6 group after their 6-hr sleep. However, the subjects in the 6:1 group performed much better than those subjects who did not sleep at all.

Ultrashort Sleep: Benefits

The converging lines of evidence, as presented in the previous sections of this chapter, seemed to suggest that the duration of each sleep episode must be longer than 4-10 min to be recuperative.

In work environments that demand around-the-clock operations with a minimal number of personnel to share job responsibilities, taking a short nap at, or near, the work site is a pragmatic solution to reduce the fatigue and sleepiness of long work hours, and is highly recommended. Ultrashort sleep of 5, 10, or 20 min taken by personnel right at or near the work site may provide a welcome relief for the workers and enhance productivity and safety. Naps on a chair (Nicholson and Stone, 1987) or on a cot placed at a work site refresh workers and are much easier for management to approve than a 1-hr or longer sleep in a room somewhere far away from the work site during the middle of the "work" period.

Ultrashort naps may also prevent occurrence of a rare, but serious, problem, "behavioral freezing" (Folkard and Condon, 1987), where instead of responding quickly to an emergency, the individual lapses into temporary immobility.

Ultrashort Sleep: Problems

The benefit of using ultrashort sleep to capture sleep quantum in a prolonged work period needs to be balanced against its two major shortcomings, sleep inertia and reduced sleep efficiency.

Sleep Inertia

Immediately after getting up from sleep, irrespective of the hour, one is not at one's best, that is, one experiences sleep inertia (Lubin et al., 1976). Sleep inertia represents a reduced performance capability during a period after being suddenly awakened from sleep. Pigeau et al. (1987) used electroencephalographic (EEG) indices to find that sleep inertia is characterized by EEGs resembling an early phase of sleep. Balkin and Badia (1988) found that a period of sleep inertia is not a novel state, but merely persistence of "typical" sleepiness. These aftereffects of sleep occur from 1-5 min (Dinges et al., 1985; Webb and Agnew, 1974) to 15 min following awakening (Wilkinson and Stretton, 1971). Owing to sleep inertia U.S. Air Force crews have been prohibited from napping while on immediate alert or standby (Hartman and Langdon, 1965; Hartman et al., 1965; Langdon and Hartman, 1961).

Seminara and Shavelson (1969) showed that 4 subjects in a simulation study for a NASA 5-day lunar mission experienced sleep aftereffects in some tasks persisting for up to 12 min, although the largest performance decrements were observed during the first 3 min. Naitoh (1981) reported that inadequate short recovery sleep after a prolonged sleep deprivation resulted in more serious and prolonged sleep inertia.

Because of this sleep inertia, workers who are involved in jobs requiring a fully functional, alert mind instantly upon awakening [such as aviators (Angiboust, 1970) and truck drivers] are not recommended to take any naps during work periods. The cases of infantry soldiers suffering from sleep inertia, as mentioned by Haslam (1982), should be handled differently from the aviators and truck drivers. Infantry soldiers, as well as night nurses and nighttime operators of power generation plants, will have more time for becoming fully awake before appropriate actions are demanded of them. In fact, for some shift-work personnel who have less stringent requirements for speed and accuracy of responses, adopting a simple procedure of washing the face with cold water to dispel sleep inertia quickly (Labuc,

1978, 1979a,b) would be highly recommended rather than fighting off waves of sleepiness.

Loss of Sleep Efficiency

Ultrashort sleep appears to reduce sleep efficiency in comparison with long continuous sleep. Previously, in the discussion of the "short-day" or non-24-hr day, an average sleep efficiency was estimated to be about 50%. This means that under ultrashort or non-24-hr-day schedules, we might be given an opportunity to sleep, but, on average, we can use one-half of that "bed time" for actually sleeping. However, as pointed out by Stampi (1989b), sleep efficiency is expected to improve once we have adapted to a new sleep-wake schedule. Hence, the loss of sleep efficiency in ultrashort sleep appears to be connected to lack of opportunity and/or motivation to adapt to the work environments that demand ultrashort sleep. Suggestions have been made either to use quick-acting hypnotics to induce sleep, or to train sleepers on techniques of biofeedback and autogenic relaxation for rapid sleep onset and for improving sleep efficiency.

Sleep Management: New Mandate

Despite the two drawbacks in practicing ultrashort sleep at work sites, ultrashort sleep offers the management of sleep a flexible tool in capturing sleep quantum without interfering with military or industry operations. Unfortunately, much of the necessary data on ultrashort sleep are not available.

When should workers be advised to take ultrashort sleep on their work sites? Should they sleep on a chair or a cot placed at or near the work site, or should they sleep in a secluded area for maximal sleep efficiency? How many minutes of ultrashort sleep should be allowed? How long will the recuperative effects of an ultrashort sleep last?

The Job of Sleep Manager

While the science of sleep management works toward gaining more information about the recuperative power of ultrashort sleep, sleep managers must concentrate on accomplishing the following two objectives using currently available technical base: to develop a technical database describing the roles of ultrashort sleep in sustained/continuous operations, and to develop a performance model for ultrashort sleep.

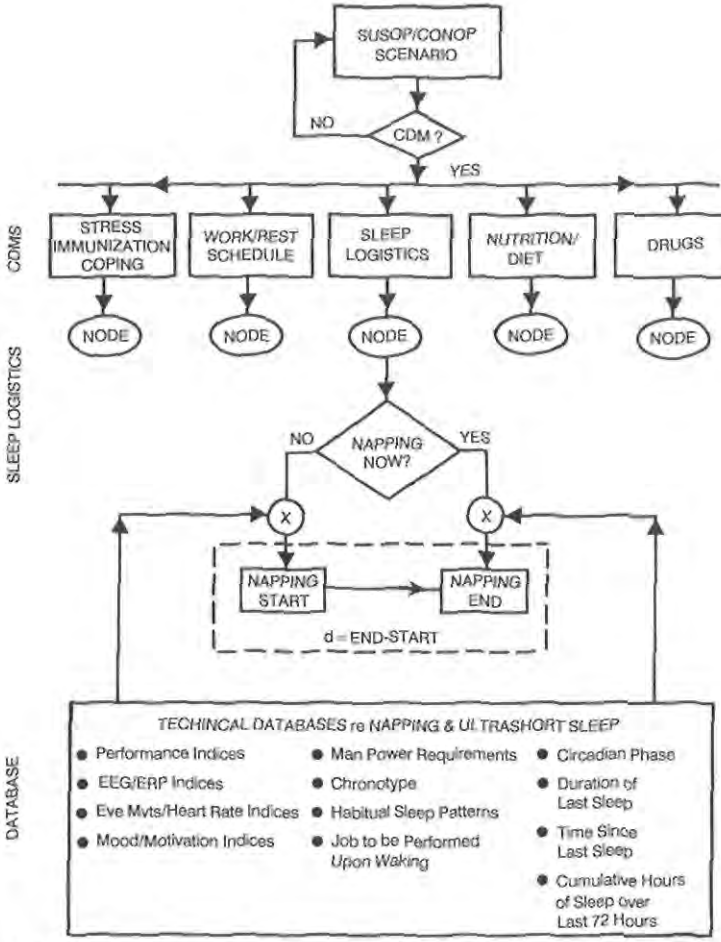

FIGURE 13.1. A flow diagram showing sleep logistics as one of the five measures *to counteract performance degradation during sustained/continuous operations* (SUSOP/CONOP). When a work schedule (scenario) interferes with regular sleep routine, sleep managers need to initiate one or more of counterdegradation measures (CDM). Five CDMs are listed in this figure (Stress Immunization, Work/Rest Schedule, and others). The applications of sleep logistics consists of making an observation as to whether individuals are asleep or awake. If they are awake, a decision must be made as to when a nap is recommended; if asleep, the decision concerns when to wake them. The decision must be based on a technical database (boxed-in by a solid line) on napping and ultrashort sleep. X = Decision point for either napping to begin or to end. d = Napping duration.

Database for Ultrashort Sleep

Figure 13.1 shows a flow diagram describing the role of sleep management, particularly of ultrashort sleep. The work environments presented in Figure 13.1 are quite common among military personnel who are required to work continuously with little or no sleep for a prolonged period of time until the task is completed (Sustained or Continuous Operation, SUSOP/CONOP).

If work is completed in less than 24 hr, there would be no need to invoke counterdegradation measures (CDMs) to support individual and group performance. However, if the task requires personnel to work longer than 24 hr continuously and the work begins to interfere with sleep, the sleep manager needs to apply as many CDMs as available to support the work forces. Figure 13.1 lists five CDMs.

In applying sleep logistics, the first action is to observe whether workers are presently napping. If they are not napping, then the next vital concern is to determine when they ought to start napping. If the workers are found asleep, the vital decision is to determine when they should be awakened.

The decision processes (marked with the large X in Figure 13.1) on when to begin or end a nap will be based on the technical database which is boxed in, for the sake of emphasis, at the bottom of Figure 13.1. The technical database for napping and ultrashort sleep still remains incomplete, awaiting more data from future experimentations and field observations.

Figure 13.1 lists some of the known technical databases with which sleep managers must be familiar. For example, if a group of workers is not napping, its group performance indices are not up to the preset standard, and they show signs of persistent excessive sleepiness, then sleep managers should advise them to start napping. The sleep managers also should know that there are enough workers at the work site to afford letting this group off for a nap. Further development of technical database is necessary just to make the simple, but critical, decision of "to nap or not to nap."

Performance Model for Ultrashort Sleep

Another responsibility of the sleep manager is to develop a performance model similar to a conceptual one given in Figure 13.2.

The *-axis shows a 4-day period of sustained operation. The .y-axis is an arbitrary unit with an arbitrary threshold index value. Performance above the threshold value is regarded as being at an acceptable level of competence. Performance index below the threshold is of unacceptable quality. The *-axis represents a 4-day-long period of continuous work. The solid line represents a hypothetical performance index during a sustained operation when sleep is not allowed across 4 days. The hypothetical performance index shows a prominent circadian rhythm superimposed on a linear decrease in performance index across 4 days. Although the solid line in

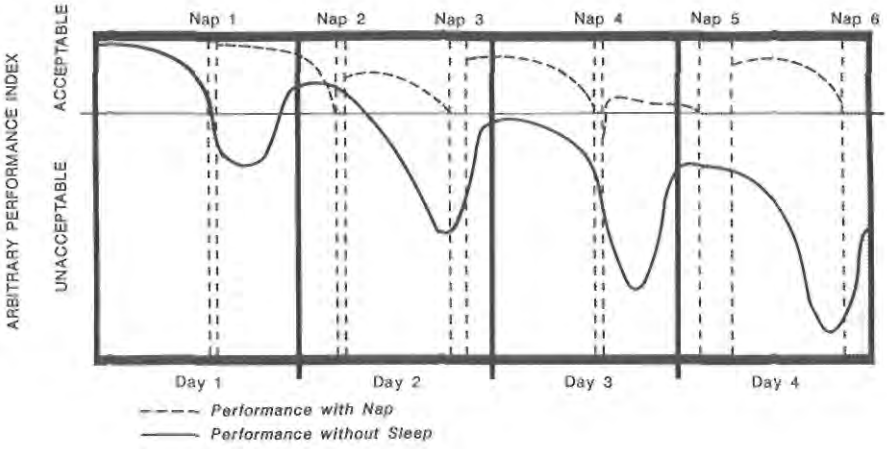

FIGURE 13.2. Hypothetical performance improvement owing to adoption of 6 short nap periods during a 4-day-long sustained/continuous operation. Without sleep, task performance declines in a predictable fashion down to the unacceptable level within 2 days (the solid line). With timely interjection of napping, the overall performance index remains at the acceptable level over 4 days of a continuous work period, overcoming the effects of sleep loss and circadian nadir on task performance (the broken line). Napping is imposed when the performance index fell below the arbitrarily set threshold value for the acceptable level. Napping ends when a sleep manager has judged that a nap was sufficiently long. See text for details.

Figure 13.2 is arbitrarily drawn, it closely follows a generalized performance curve based on data from studies of the effects of 72-hr total sleep-deprivation performance (Thorne et al., 1983).

In Figure 13.2, six naps were allowed by a sleep manager to counter performance impairment due to sleep loss. During day 1, the performance index fell below the threshold due to circadian dip, and the sleep manager decided to permit a short nap, Nap 1. Expected recovery in performance index is shown by a broken line. The duration of the nap is shaded. Early in day 2, the performance index (the broken line) again hit the threshold, so the sleep manager decided to interject another nap, Nap 2. During the same day 2, the sleep manager observed another dip in performance to trigger Nap 3 which is much longer than Nap 1 or Nap 2. The nap duration depends on the sleep manager's experience with Technical Database (cf. Figure 13.1). Currently, very few facts are available to determine how long a nap period should be in order to achieve sufficient, enduring performance recovery. It is relatively easy to determine, using measures taken from brain waves (Pigeau et al., 1987) or performance indices, to conclude that sleep is needed immediately to maintain the level of performance. However, we have as yet to develop some on-line measures to indicate that a nap of sufficient duration was taken to warrant awakening.

In creating Figure 13.2, it was assumed that the science of sleep management has developed a performance model for ultrashort sleep. Figure 13.2 shows that the performance index of the sleep-deprived becomes unacceptable after 1.5 days of continuous work. This means, without the ultrashort nap, the performance over the remaining 2.5 days (60 hr) is of unacceptable quality without any CDMS. Performance was maintained throughout the experiment taking naps 2 through 4. This means that naps totaling less than 10 hr, which are judiciously distributed during a sustained operation, regained about 50 odd hours of "useful time" toward a completion of the mission. The key utilities in developing this computerized behavioral model are how one decides when napping should start and when it should end.

The nap stop time is greatly influenced by nonpsychophysiological factors such as work demands and manpower requirements. Napping will be stopped when work demands require a larger number of workers on the job than are currently awake. The nap stop time can be estimated by knowing each individual's habitual sleep patterns, the duration of last sleep, the time since last sleep, the cumulative hours of sleep during the past 72 hr, the kind of job to be performed, and each individual's "chronotype" (morningness and eveningness). For sleep managers, a question of when to wake the sleeping workers and soldiers following a prolonged period of continuous work remains difficult. This is a vital area of research for the future.

Discussion and Conclusions

The sleep managers' task is to create a humane work schedule for irregular or prolonged work. Sleep managers should remember that most shift workers keep their work hours, not because it is good for them but because it is good for society. The least the science of sleep management can do for workers is to make their work more bearable, safe, and productive by proper sleep management as a part of the overall shift-work planning and manpower allocation. Sleep managers should create new work ethics according to which signs of fatigue such as yawning and sleeping at work sites are looked upon favorably.

In the search for sleep quantum, there appears to be a consensus that a period of 4.5-5.5 (average of 5) hr of continuous sleep per 24 hr would satisfy the daily requirement for core sleep, thereby enabling a high level of job performance for an indefinite period of time. However, there seems to be no agreement among sleep researchers about whether the 4.5-5.5 hr of sleep can be taken in small increments, for example, of 10, 20, and 30 min of sleep. It is known that having many episodes of an extremely short 1-min sleep all night long has no power of recuperation. What is not resolved is the

question of how long each sleep episode should be before it becomes, at the least, behaviorally recuperative.

Another unresolved question is what are the mechanisms through which ultrashort sleep loses its recuperative power? As discussed previously, the sleep inefficiency of ultrashort sleep is due to the fact that one cannot fall asleep quickly during the time periods that are allocated for sleeping. It is not known, however, whether the shortness of sleep, in itself, has reduced recuperative power. For example, if we let sleepers accumulate a total of 5 hr of sleep in 5 separate episodes of sleep by letting them continue sleeping until they have 1 hr of sleep, is this "fragmented sleep" less recuperative than sleep of 5 continuous hours? No definitive data are available. If a fragmented 5-hr sleep has far less recuperative power than a 5-hr continuous sleep, we could certainly conclude that the continuity of sleep itself plays a vital role in determining recuperative power.

There is another unresolved question about ultrashort sleep. As Bonnet (1986) and Magee et al. (1987) noted in their studies, 1-min sleep does not include SWS and REM sleep even after accumulation of many 1-min-long sleep episodes across the entire night. It seems that such sleep is too short to include SWS and REM sleep, and it is also too short to be recuperative. In contrast, when each sleep episode is 10-min long, all-night sleep includes almost one-half of normal SWS and REM sleep, and sleep appears to be recuperative. Comparisons of the differences in sleep stages between 1-min versus 10-min sleep episodes in terms of power of recuperation might revive a familiar argument that the inclusion of SWS and REM sleep are responsible for recuperation. However, Bonnet (1986) argues competently against invoking sleep stages to explain recuperation, because there were no significant correlations between amount of SWS or REM sleep and performance recovery. Bonnet feels that the fact that one sleep episode was 10 min, another was 1 min, is the determining factor in recuperative power of sleep. Sleep stages have nothing to do with recuperation. More experimental evidence is needed to resolve this issue of sleep stages versus duration of sleep episode in terms of the recuperative power.

Finally, the role of adaptation needs to be emphasized regarding ultrashort sleep schedules. Stampi's observations showed a relative ease of adaptation to ultrashort schedules, despite commonly held opinions to the contrary. The recuperative power of ultrashort sleep may depend partly on a degree of adaptation to the life-style involving needs for ultrashort sleep. A majority of research summarized in this chapter does not provide the experimental subjects long enough adaptation time to the ultrashort sleep life-style. Hence, a decline of sleep efficiency under the "short-day" paradigm could have been reversed if the observation periods were much longer.

We used to feel that daytime naps, especially among the elderly, were undesirable events because they tended to degrade the quality of nocturnal sleep. Almost all sleep-disorder patients show fragmented nocturnal sleep

with daytime excessive sleepiness and poor performance; hence, fragmented sleep was regarded as poor, nonrefreshing sleep to be avoided. The Continuity Theory of Sleep predicts that only a period of uninterrupted and continuous sleep is recuperative. In this chapter ultrashort sleep has been presented to be recuperative from daily fatigue and sleepiness. More data are needed to establish the conditions in which ultrashort sleep would benefit us in fulfilling daily responsibilities.

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14

Sustained-Operations Studies: From the Field to the Laboratory

R. G. ANGUS, R. A. PIGEAU, AND R. J. HESLEGRAVE

Advances in military technology (including improved night-vision devices, high-performance vehicles, advanced communications systems, and increased firepower) and resultant changes in doctrine (emphasizing around-the-clock capabilities) have resulted in military forces training for sustained operations. Soldiers are expected to work with minimal sleep during missions lasting from days to weeks. Rest is dictated by the nature of the operation and will be fragmentary at best. When allowed to sleep, participants might be required to awaken quickly and resume operational duties immediately. Many experimental studies have shown that during one or more nights without sleep, as well as over longer periods of reduced or fragmented sleep, decrements occur in mood and cognitive performance (for reviews, see Wilkinson, 1965; Naitoh and Townsend, 1970; Naitoh, 1976; Kjellberg, 1977; Johnson, 1982). The operational consequences of sleep deprivation have also been recognized (Marshall, 1962). Naitoh (1983) has argued that adequate planning for sleep is as important as logistics for transportation, equipment, ammunition, and food. Sleep logistics planning is difficult, however. Work-rest schedules based on orderly civilian patterns are not appropriate for sustained operations, especially in the absence of replacement personnel.

The research program to be described here was designed to predict and modify the effects of operational stressors on mental performance. Our approach has been twofold. First, we attempted to understand cognitive capabilities on the basis of behavioral and physiological data collected during field studies. Second, a laboratory facility to simulate sustained-operations command-post activities was developed. In this facility experimental subjects assume the role of operations officers handling message traffic and related duties during long, intense work periods while undergoing sleep loss.

WHY WE NAP
Evolution, Chronobiology, and Functions of Polyphasic and Ultrashort Sleep

Background

For military operations, knowledge is required concerning how well the results of controlled laboratory experiments generalize to uncontrolled field conditions. For research to be operationally relevant, more complete understanding is required of operational stressors and their interactions with the types of tasks to be accomplished and with individual performance capabilities. Very little research, however, bears directly on the interaction of environmental-situational and individual stressors (cf. Johnson, 1982). Studies attempting to estimate performance degradations for sustained operations have, for the most part, followed the traditional approach to the study of sleep loss. Subjects' capabilities are measured on selected tasks at various times during the periods of wakefulness (cf. Drucker et al., 1969; Banks et al., 1970; Haggard, 1970; Ainsworth and Bishop, 1971; Morgan et al., 1974; Opstad et al., 1978; Banderet et al., 1981; Haslam, 1981, 1982; Naitoh, 1981; Naitoh et al., 1982). This approach engenders several major methodological difficulties.

First, performance tasks are often not representative of operational tasks. Because performance degradation is highly dependent upon the types of tasks to be performed (Wilkinson and Stretton, 1971), the selection of appropriate tasks with a high degree of generality to military operations is required for accurate assessments of performance degradations. For example, while performance on simple vigilance tasks requiring sustained attention may be impaired following partial sleep deprivation (Wilkinson et al., 1966), performance on interesting tasks often shows no visible impairment for up to 42 hr without sleep (Wilkinson, 1964). Similarly, the use of nonrepresentative tasks may lead to false estimates of the effects of sustained operations.

Second, laboratory tests may not be administered with sufficient frequency to afford reliable information regarding performance efficiency. Testing that occurs every hour or two (e.g., Alluisi, 1969; Morgan et al., 1974) to only once per day (e.g., Opstad et al., 1978; Haslam, 1981) may not produce sufficient data to obtain reliable estimates of performance. Such data may also be confounded by circadian variations, with performance being best during the day and worst at night, particularly between 0200 and 0600 (Colquhoun et al., 1968; Hockey and Colquhoun, 1972). Additionally, if subjects are tested infrequently, they may be able to draw on unused reserves or capacity not required during interim periods to enhance performance during test periods. Thus, performance estimates based on intermittent testing may be overestimates based on short-term high-energy expenditure because nondemanding intertest intervals may attenuate the general drain on energy reserves (Harris and O'Hanlon, 1972; Morgan et al., 1974; Naitoh, 1976; Johnson, 1979).

A third problem is that different tests are differentially sensitive to sleep loss. To illustrate this, some studies of partial sleep deprivation (e.g., Webb and Agnew, 1965, 1974; Rutenfranz et al., 1972; Friedmann et al., 1977)

have shown no performance decrements as a result of reduced sleep. As pointed out by Tilley and Wilkinson (1984), this may have been due to the use of insensitive performance tests.

A fourth complicating factor is that the duration of a test will affect its sensitivity. For instance, short tests are often less sensitive to performance changes than longer versions of the same tests. While the loss of one complete night of sleep typically has no effect on the first 5 min of work for most kinds of tests, there is clear impairment when tests are prolonged from 15 to 45 min (Wilkinson, 1964, 1965).

Additionally, many studies may not provide adequate estimates of performance degradation for sustained operations because of relatively low performance demands. Most studies include a mixture of more-or-less sensitive cognitive tasks, intermittent testing, and nondemanding intertest intervals. Thus, their estimates of expected performance degradation may be conservative. Studies providing the best estimates are likely to be those where the environmental demands were more continuous (e.g., Morgan et al., 1974; Naitoh, 1981; Naitoh et al., 1982). Even those studies, however, contain lengthy periods of inactivity, or time devoted to tasks of low cognitive demand.

Closest to the ideal of continuous performance demands is the study by Mullaney et al. (1983) in which subjects were required to work at four tasks that were repeated every 10 min for 42 hr. Serious decrements in performance and disturbing psychological events (e.g., visual illusions, hallucinations, derealizations, and disorientations) began after about 18 hr of testing (about 2300). This is earlier than has generally been reported in sleep-loss studies (cf. Johnson and Naitoh, 1974). However, these results may be limited in how well they generalize to high-intensity sustained-operations conditions because the tasks were repeated every 10 min. This repetition likely potentiated the monotony of the situation resulting in enhanced sleep loss effects (Wilkinson, 1965).

The Defense and Civil Institute of Environmental Medicine's (DCIEM) sustained-operations research program has been designed to address some of the above limitations. It is primarily concerned with estimating the effects of sustained mental work and sleep loss on a range of cognitive abilities related to command-and-control performance. Our approach has been "iterative," involving sequences of operational/field trials and laboratory experiments. The following sections briefly describe our research experiences in both environments.

Field Studies

Simply stated, the long-term aim of the DCIEM sustained-operations research program is to provide guidelines for performance limits, and rest requirements, for personnel engaged in "around-the-clock" operations. As noted above, our decision to include operational and field trials in our

approach to this problem was predicated on the fact that many of the findings of the effects of "sleep loss" on human performance were not directly applicable to "sustained-operations" conditions. The major reason was that laboratory stressors are usually studied one at a time for relatively short periods. To properly estimate the effects of stressors on performance, research for sustained operations must be done under conditions that approximate the anticipated stressors. This implies that at some point sustained-operations research must be done in conjunction with "realistic" field exercises, using measures that will not intrude upon the soldiers' assigned duties. Thus, an important goal of our research is to provide measures that can be used in field conditions. Some of the field situations in which we have tested our methods have included land forces in conditions ranging from arctic to desert operations and both air and ship operations (cf. Angus et al., 1979, 1981, 1985b; Buguet et al., 1980, 1981; Forshaw et al., 1981; Bittel et al., 1981; Kuehn et al., 1981; Allen et al., 1982; Rivolier et al., 1982; Brooks et al., 1988). In many cases, however, the field exercises were not adequate for the study of sustained operations. For example, during a 4-day "sustained-operations" phase of a Canadian Army divisional exercise the cognitive performance and sleep of a small battery of gunners were monitored. Using ambulatory EEG recorders and sleep logs we attempted to determine the amount of sleep that was obtained during the exercise. Table 14.1 shows the duration of each sleep period for each member of the battery during the 4-day exercise. The last column shows the

TABLE 14.1. Amount of sleep (in hours) obtained during a continuous-operations exercise

Subjects	Day 1	Day 2	Day 3	Day 4	TST[a]
S6 LT	4.5	1.5	2.0	2.0	14.0
			3.0	1.0	
S5 WO	6.0	4.5	2.0	1.0	14.75
		0.25		1.0	
S4 GNR	4.5	0.5	2.0	0.75	18.75
		2.0	2.0	2.5	
		3.0		1.5	
S3 GNR	4.5	1.5	1.0	4.25	22.25
		4.5	4.0		
		2.5			
S2 GNR	4.0	5.0	2.0	1.0	26.0
		4.0	5.0	3.0	
				2.0	
S1 GNR	4.0	0.5	1.0	4.0	17.5
		2.25	2.0		
		1.0	2.75		

[a]TST = total sleep time.

total amount of sleep each subject obtained. Although their sleep was fragmented into several short "naps," the subjects did manage to get more than 4 hr per day. As well, the performance data indicate that this amount of obtained sleep was adequate to maintain the subjects' performance (at least on the measures we used). Figure 14.1, showing fatigue rating score results, is typical of a number of objective and subjective measures collected. The first data point occurs about 24 hr after the start of the exercise. There is little change over the course of the study.

In summary, the results showed that the subjects averaged about 4 hr sleep per day and that their performance remained stable. The results of other field studies were similar. In general, personnel were not subjected to work-rest regimens usually expected of "around-the-clock" operations. Although the amount of sleep obtained in many exercises was usually reduced, it was not sufficiently reduced to impact on performance. The question to be asked of these studies, then, is whether they adequately reflect work-sleep regimens that might be expected during sustained operations.

Laboratory Studies

For our laboratory studies we assume a much worse case than those observed in field conditions. Subjects are kept awake, and working (at cognitive tasks), during a period of total sleep loss that sometimes lasts more than 2 days. The experimental methods and design of the studies will be presented first, followed by results of the effects of sustained mental work and sleep loss on cognitive performance. Then, the results from some counterdegradation studies, including the effects of physical conditioning, physical exercise, low mental workload, and 2-hr naps are presented.

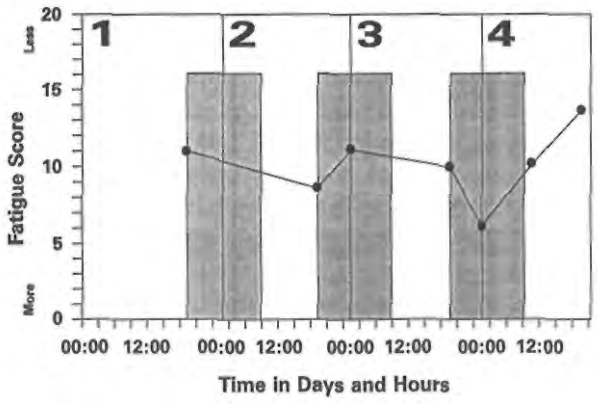

FIGURE 14.1. Fatigue ratings during a continuous-operations exercise.

The laboratory environment was designed to provide a continuous high-demand battery of cognitive measures sensitive to meaningful performance, especially with regard to command-and-control functions. It was developed as a brigade-level command-post in which the subjects assume the role of operations duty-officers whose function is to monitor a communications network. This involves accessing, reading, understanding, interpreting, and filing message information as well as updating tactical maps. This information provides the basis for decisions the subjects are required to make as the scenario progresses. Embedded in and distributed around this "message-processing" task are a variety of cognitive tests.

Sustained-operations experiments are usually run with 4-subject groups resident in the laboratory for 5 or more days. The laboratory is self-contained and the subjects work individually in separate test rooms that are isolated from one another and from the experimenters' control area. Closed-circuit television is used to visually monitor the subjects, and slave monitors display the information on each subject's terminal screen. Continuous EEG, ECG, and various other physiological responses are recorded on ambulatory cassette recorders. The testing system is fully automated for stimulus presentation response collection and data storage and analyses. Subjects have included university students, both young and older military officers, and lower ranks personnel. Both male and female subjects have been tested. On first arriving at the laboratory subjects are briefed on the scenario and given explanations of the military concepts and terminology. They are given extensive training and practice on all tasks. In the evening they relax, are prepared for physiological recordings, and are allowed to sleep for an 8-hr period. Subjects are awakened between 0600 and 0800 and the scenario begins approximately 1 hr later. After 64 hr, the subjects sleep for an 8-hr recovery period (during the same hours as the baseline sleep), and the next morning are tested for recovery effects (usually over a 6-hr period). Depending on the particular experiment, subjects undergo total sleep deprivation, perform physical exercise, receive a nap, or experience other experimental manipulations during their time in the laboratory.

Experimental Design

The experiments follow a general design in which several 6-hr blocks of identical cognitive tasks are presented. Only the content of the military messages changes over the experiment. As an example, Figure 14.2 illustrates the design of a study investigating the effects of a nap between 2200 and 2400, after 40 hr of sleep loss.

The range of activities in each of the 11 task blocks is shown in Table 14.2. Each subject performs the same sequence of blocks and tasks within blocks. In this experiment there were three 2-hr work sessions per block, each separated by short rest breaks. An exception is block 7 where a nap was substituted for the second work session.

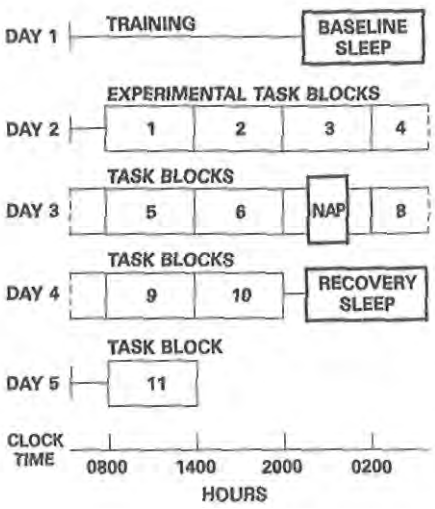

FIGURE 14.2. Design of an experiment investigating the effects of a 2-hr nap between 2200 and 2400.

The experimental tasks range in duration from 5 to 15 min. A 15-min "Scales & Battery" package occurs approximately once per hour at the beginning, and again halfway through, each work session. This package comprises a variety of shorter tasks that have been found to be as sensitive to sleep-loss effects as longer duration tasks (Heslegrave and Angus, 1985). These short tasks are used to monitor performance hourly.

Experimental Tasks

Of the many performance tasks incorporated in these experiments, a few are typically used in many sleep-loss studies. These include a variant of the four-choice Serial Reaction Time task described by Wilkinson and Houghton (1975); an Encode/Decode task similar to that reported by Haslam (1985); a continuous Subtraction task adapted from Cook et al. (1972); and a Logical Reasoning task devised by Baddeley (1968). Some of the tasks were slightly amended to adapt them to the military scenario. In addition, the following self-report measures were collected: the Stanford Sleepiness Scale (Hoddes et al., 1973); the School of Aerospace Medicine (SAM) Subjective Fatigue Checklist (Harris et al., 1971); and the NHRC Mood Scale (Johnson and Naitoh, 1974). It was intended that the results of these "standard" measures be compared with the results of previous sustained-operations experiments in order to determine the effectiveness of this continuous-testing environment. For more detailed information about the tasks and comparisons with results from other studies, see Angus and Heslegrave (1983, 1985) and Heslegrave and Angus (1983, 1985).

TABLE 14.2. Experimental activities and their temporal occurrence during a typical 6-hr task block

Start time (Real)	Start time (Elapsed)	Tasks
	Session 1	
0800	*0000*	*Scales & Battery*
0815	0015	Decode (Normal)
0820	0020	Messages
0830	0030	Decode (Motivated)
0835	0035	Messages
0845	*0045*	*Scales & Battery*
0900	0100	Decode (Group)
0905	0105	Syllogisms
0920	0120	Messages
0930	0130	Decode (Normal)
0935	0135	Missile Defense
0940	0140	•••• Break ••••
	Session 2	
1000	*0200*	*Scales & Battery*
1015	0215	Serial
1025	0225	Messages
1035	0235	Subtraction
1040	0240	Messages
1055	*0255*	*Scales & Battery*
1110	0310	Messages
1120	0320	Logical Reasoning
1130	0330	Missile Defense
1135	0335	•••• Break ••••
	Session 3	
1200	*0400*	*Scales & Battery*
1225	0415	Memory (Training)
1235	0425	Messages
1235	0435	Digit Span
1245	0445	Messages
1255	*0455*	*Scales & Battery*
1310	0510	Messages
1320	0520	Plotting & Memory (Recall)
1330	0530	Missile Defense
1335	0535	•••• Break ••••

The Message Processing task provides the continuous-operations context for the experiments. Subjects begin this task by monitoring two message queues of differing priority displayed on their terminal screens. They are instructed to always access messages from the priority 1 queue (if available) regardless of the number of priority 2 messages. A new message arrives about every 90 sec, with priority 2 messages outnumbering priority 1 messages by a ratio of 8:1. The subjects' task is to read and understand each message well enough to answer a set of questions that follows. The priority 1 questions require subjects to decode the resource state of various brigade

units. Priority 2 questions require subjects to perform such duties as identifying the locations of various units (using the map grid references), describing units' activities (current or intended), selecting the most appropriate unit for a specific task, calculating equipment resources, and estimating travel distances and times of arrival. Most of the questions require short phrases to be typed on the keyboard, while some require the scenario map to be updated. Other questions request that summaries be handwritten and manually filed. Accurate completion of the written summaries is important because previously processed messages cannot be retrieved from the computer; the manually filed information is thus necessary for answering questions asked in later messages.

Experimental Results

The studies reported in this section established the pattern and degree of performance degradation observed during sleep loss in the continuous work paradigm. The results of investigations into several countermeasures will also be presented and discussed.

BASELINE RESULTS

The baseline experiments investigated the effects of 54 hr of continuous mental work and sleep loss on cognitive performance. Figure 14.3 illustrates data from the Fatigue scale that were typical of results from the self-report measures. They are presented in terms of block (6-hr) means. As the figure indicates, subjects became progressively more fatigued (i.e., they had lower

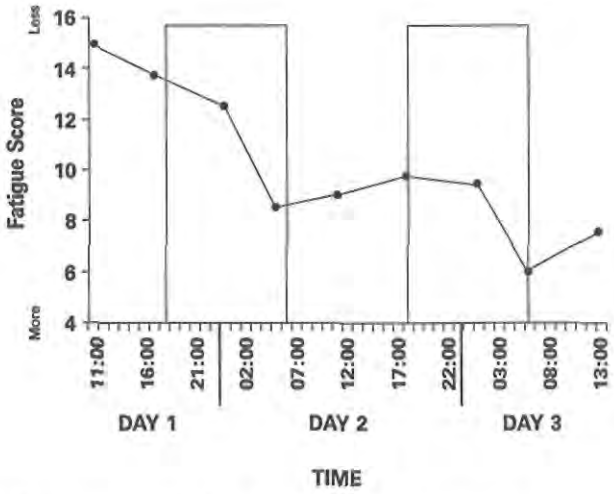

FIGURE 14.3. Fatigue scores during 54-hr of continuous work.

scores) over the course of the experiment. However, the fatigue effect became acute about 18 hr into the experiment (about 0300) and then plateaued for about 24 hr. Following this plateau, another dramatic decline occurred from 0300 to 0900 during the second night. The other subjective scales showed similar declines and plateaus over the 6-hr blocks.

Results from the Encode/Decode task are representative of the objective performance tasks. Data from the 10-min version of this task were collected once in each block of the experiment. Figure 14.4 shows the changes in the number of correct responses and number of errors. A significant main effect over time showed that the declining number of correct responses had plateaus similar to the Fatigue scale results. Subjects maintained accuracy at the expense of speed. As the figure also indicates, throughout the experiment subjects made fewer correct responses and more errors on the Decoding task than on the Encoding task.

Data from the Message Processing task showed similar trends. Figure 14.5 illustrates mean processing time every 3 hr across the experiment. Although the messages themselves were controlled neither for length nor for the number of questions asked per message, the work required was relatively homogeneous across sessions. The results were similar to those from the other measures except that the early sessions showed an initial

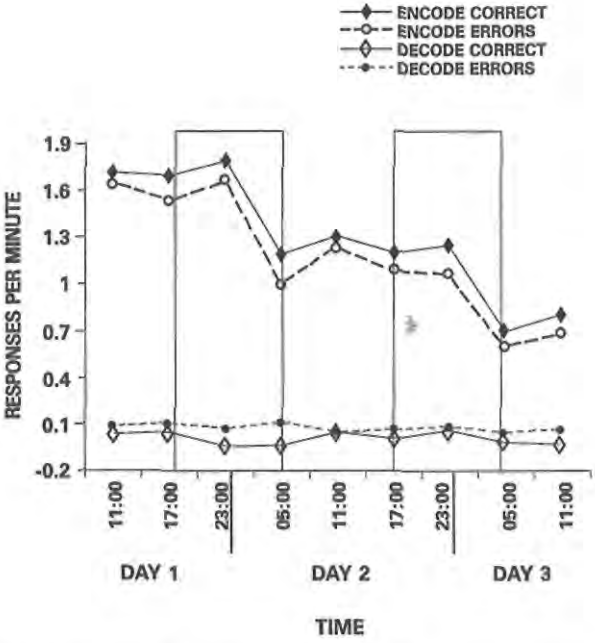

FIGURE 14.4. Encode/decode scores during 54-hr of continuous work.

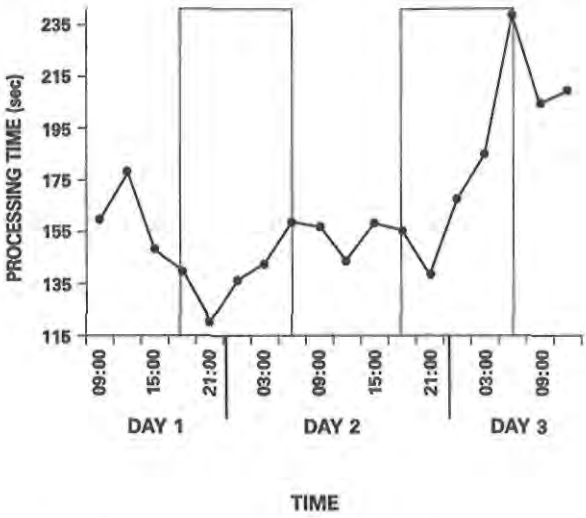

FIGURE 14.5. Message Processing times during 54-hr of continuous work.

improvement in performance (reduced processing time), probably because the subjects were still learning strategies for dealing with the various types of message problems. Once the subjects became familiar with the task (by about 2100 of the first day), a sleep-loss effect was apparent in the increased time required to process the messages. This increase plateaued until 2400 the next night, followed by a dramatic decline in performance (increase in processing time).

Tasks of short duration are usually not sensitive to the effects of sleep loss; subjects are able to maintain their attention for the short test period and override the effects of fatigue (Wilkinson, 1964, 1965). Figure 14.6 contrasts correct response data from short-duration (1-min) and long-duration (10-min) versions of the Reaction Time task. [Results from other short-duration tasks were similar (see Heslegrave and Angus, 1985).] Although the level of responding was higher for the short task, both versions showed the same topography. This demonstrates that short tasks *can* be as sensitive to sleep loss as longer ones, at least in high-demand cognitive work environments. Because these "short tasks" are embedded in long sessions of continuous work, they might be considered as elements of a single, long, heterogeneous task. This interpretation is consistent with Wilkinson's (1969) suggestion that tasks need to be of long duration to be sensitive to the effects of sleep deprivation.

Support for the notion that the long work sessions are functionally similar to long tasks is found in Figure 14.7. The figure presents data collected at the beginning of work sessions ("After Rest") and data collected

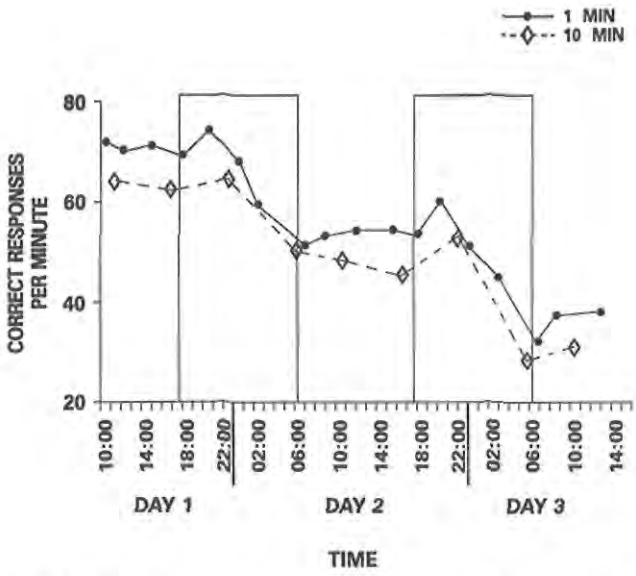

FIGURE 14.6. Serial Reaction scores during 54-hr of continuous work (1-min vs 10-min task sessions).

halfway, through sessions ("During Work"). It shows (after 1 hr) that performance depends upon when data are collected. Once fatigue effects began to emerge (about 2400), the "After Rest" and "During Work" curves began to diverge and did not overlap for the remainder of the experiment.

The other subjective scales and performance tests (e.g., Figure 14.7) showed similar results. In general, once the effects of sleep loss were felt, subjects were less able to override them during the work sessions than they were immediately after resting.

The baseline studies were designed to address limitations in previous sustained operations experiments by providing^subjects with a continuous, high-demand environment of tasks that were both cognitive in nature and bore some similarity to command-post duties. The results demonstrated that 54 hr of sustained mental work produced great declines in performance and mood. Substantial decrements occurred following 18 hr of testing (reductions > 30%), and generally unacceptable performance occurred following 42 hr of sustained wakefulness (reductions > 60%). These levels of degradation are greater than those found in past studies and probably reflect more accurately the decrements to be expected during sustained-intensive operations. Moreover, these data provided baseline information for further experiments in which attempts were made to counter the effects of sleep loss on performance.

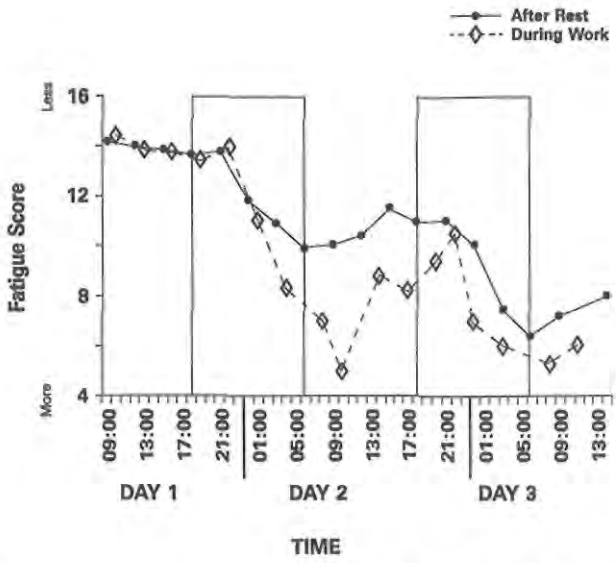

FIGURE 14.7. Fatigue scores during 54-hr of continuous work, showing beneficial effect of short rest breaks during second day of sleep loss.

RESULTS OF COUNTERMEASURE EXPERIMENTS: FAILURES

In order to ameliorate the degradations found in our basic sleep-loss and continuous-work paradigm, a number of countermeasure techniques have been investigated. Our first attempts to improve subjects' performance centered around physical conditioning and physical exercise. The first experiment compared individuals with high fitness levels to those of average fitness. Those findings suggested that when subjects were required to perform intense cognitive work during sleep loss, there was no difference in performance between subjects of varying fitness levels. That is, sleep-loss effects were not dependent on level of physical fitness. This finding pertained to performance on cognitive tasks; performance on more physically demanding tasks may well be different.

A second experiment investigated the effect of "scheduled" physical exercise on performance during sleep loss. The experimental protocol was similar to the continuous mental work experiment except that half the subjects exercised every third hour by walking on a treadmill (at about 30% of their maximal oxygen uptake), while the other half watched television, played cards, read, or studied during the same time period. Both groups spent the other 2 hr performing the continuous cognitive tasks. No differences between the exercise and sedentary groups were observed on either subjective scales or objective measures of cognitive performance (see Figure 14.8). For a more complete account of this work, see Angus, et al. (1985a).

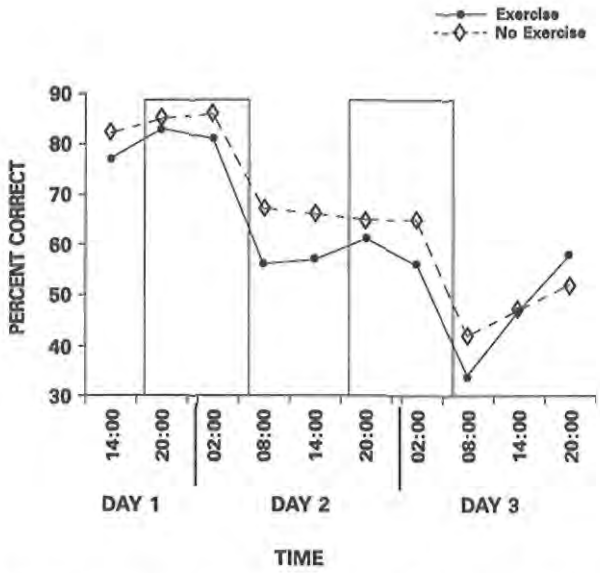

FIGURE 14.8. Vigilance scores during exercise and no-exercise conditions (1-hr Auditory Vigilance task).

A third experiment also involved exercise and continuous mental work over a 54-hr period of wakefulness. Subjects were given 30 min of hard physical exercise (at about 50% of their maximal oxygen uptake) in the middle of the second night of sleep loss (after 44 hr of wakefulness). Results typical of this exercise intervention are presented in Figure 14.9. It can be seen that there were no differences between subjects who exercised in the middle of the night and those who did not. It appears that a brief burst of exercise does not improve cognitive performance in sleep-deprived people who have been expending intense mental effort.

A fourth counterdegradation study was designed to determine if "rest" breaks are beneficial in reviving performance. Would a period of low cognitive workload (rest but not sleep) have a long-term benefit in preventing the large drop in performance observed in the previous continuous-work experiments? For the first 27 hr of the experiment subjects performed the same high-workload duties as in the experiments outlined previously. But, beginning at about 1200 following 1 night without sleep, the subjects' workload was reduced. This was done by requiring them to perform only the Scales & Battery package once every 1.5 hr for 12 hr (until about 2400). In the times between task duties subjects read, watched movies, played games, or rested, but they were not allowed to sleep.

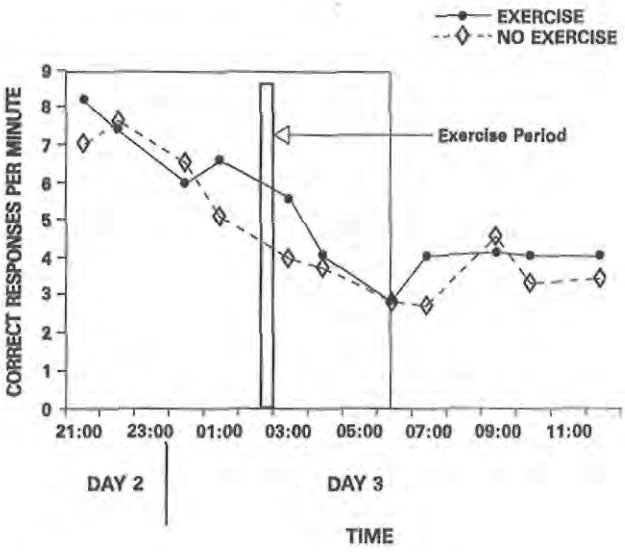

FIGURE 14.9. Effects of 30 min of acute exercise on subtraction scores.

The Fatigue scale data (Figure 14.10) showed no differences between the group that received the period of low workload and a group that performed continuously. Moreover, this intervention did not assist in reducing the large decrements in performance during the second night of sleep loss; performance declined to levels expected of individuals not having the opportunity to "rest" during the previous daytime period. The negligible impact of this low daytime workload is also reflected in the results for the Logical Reasoning task. Thus, it appears that following 1 night of sleep loss and intensive cognitive work, a period of low workload serves neither to maintain nor to recuperate performance in the long term.

In summary, to this point several experiments have been conducted in our continuous-work paradigm. The results have been clear in indicating that sleep loss combined with intensive mental work leads to *greater* decrements in performance than have been reported in previous studies not emphasizing cognitive demands. To ameliorate this performance degradation, studies investigating the effects of physical fitness, physical exercise, and altered workload demands have been completed. All of these interventions have been unsuccessful with respect to altering the effects of sleep loss.

RESULTS OF COUNTERMEASURE EXPERIMENTS: SUCCESSES

The most obvious intervention for counteracting the effects of sleep loss is sleep itself. However, as discussed in Naitoh and Angus (1989), there is

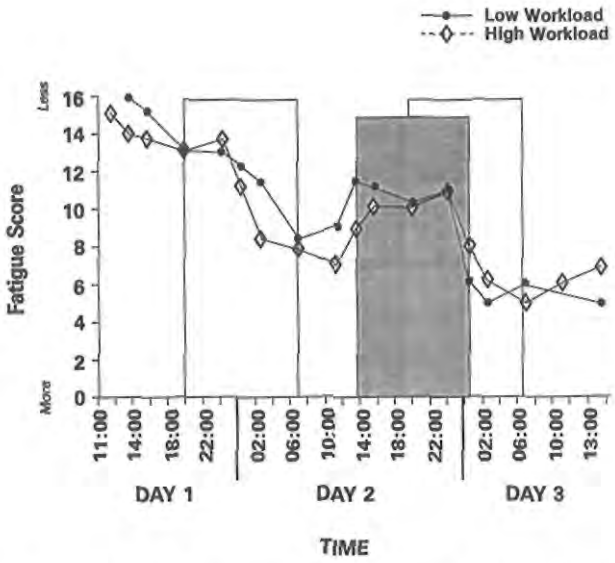

FIGURE 14.10. Low workload effects on fatigue scores (low workload period from 1300, day 2, to 0100, day 3).

controversy regarding how much sleep is necessary to maintain performance and whether it is better to take naps at certain times rather than at others. Using our continuous-work paradigm, experiments are now underway to determine the ameliorative influences of 2-hr naps placed at various times during the circadian cycle.

The studies to be reported investigated the long-term performance "benefits" of naps. Can naps serve to *maintain* performance, that is, to prevent decrements in performance? Can naps be *recuperative* for degraded performance? We were also interested in determining the severity and duration of sleep-inertia effects upon awakening. Two experiments investigating the maintenance and recuperative functions of nap sleep will be described.

In the first nap experiment individuals worked continuously under high workload conditions for 40 hr and then received a 2-hr nap from 2200 to 2400. Subjects did not expect the nap, nor were they informed of its duration. The nap was strategically placed prior to the second night of sleep loss. (The results of the continuous-mental-work experiments described in the previous section showed that performance fell to about 40% of baseline during the second night of sleep loss.) The aim of the present experiment was to determine if a nap taken prior to this expected decline in performance would prevent degradation.

As Figure 14.11 indicates, the 2200-2400 nap had long-term beneficial effects. The figure contrasts data for the Logical Reasoning task from this experiment with data from the continuous-work (no-nap) experiment.

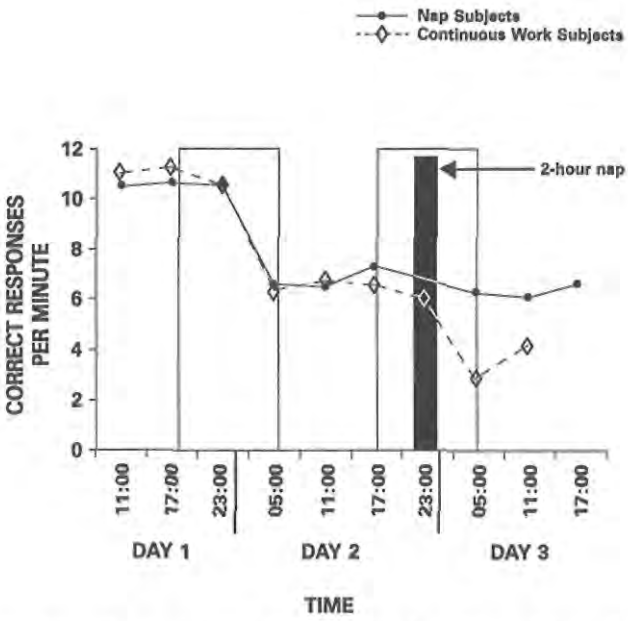

FIGURE 14.11. The effects of a nap (2200–2400) after 40 hr of sleep deprivation on logical reasoning scores (10-min task means).

Performance following the nap remained at about 70% of the levels observed at the beginning of the experiment. Without the nap, performance degraded to about 40% of baseline. Thus, a pre-2400 2-hr nap prevented decrements that usually occur during and after the second night without sleep; performance was *maintained* at the pre-2400 level. Other subjective and objective results were similar.

Figure 14.12 illustrates the effect of sleep inertia using six 2-min Reaction Time data points collected at 2-hr intervals. Three were collected prior to the nap and three afterward. The task was part of the Scales & Battery package which occurred immediately following rest breaks (and immediately after the nap). The results in the figure show that Reaction Time performance (assessed 5-10 min after awakening) was much more impaired after the nap than after a typical rest break. Whether this result is influenced by the amount of prior sleep loss or by circadian-dependent changes in performance cannot be determined from this data. However, 2 hr later (0200) performance returned to the pre-2400 level.

Another study explored the *recuperative* power of a 2-hr nap. This nap (again unexpected) was placed in the "trough" (0400-0600) of the circadian cycle after about 46 hr of wakefulness. This placement was several hours after the beginning of the usual large decline in performance observed on the second night of sleep loss.

Results for the 10-min Logical Reasoning task (Figure 14.13A) illustrate

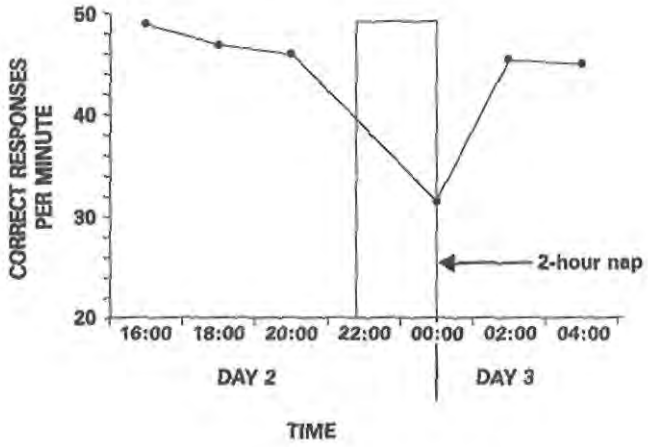

FIGURE 14.12. Sleep inertia effect following a 2200–2400 nap (Serial Reaction Time).

the performance benefits from taking a nap at this time. Performance during the third day of the experiment was the same as that on the second day. Because testing on this task occurred only at 6-hr intervals, and did not occur during the nap period (0400-0600), it appears that the nap served to maintain performance between approximately 2400 and 1200. However, the 2-min Logical Reasoning task embedded within a Scales & Battery package, which was collected every hour, clearly shows that after 2400 the expected decline in performance occurred and the nap served to return performance to pre-2400 levels (Figure 14.13B). Taken together these figures illustrate how infrequent testing may lead to erroneous conclusions. As was the case with the 2200-2400 nap, Figure 14.13B shows that performance was poor immediately following the nap with recovery occurring over the next 2 hr.

In summary, a nap taken at the low point in the circadian cycle, which followed the onset of performance deterioration, was shown to restore performance levels to those of the previous day. Thus, a 2-hr nap taken from 0400 to 0600 does appear to provide some *recuperation* for cognitive performance.

Discussion

Although the operational consequences of sleep deprivation have long been known, sleep logistics have not played a significant role in past or present military planning (Naitoh, 1983). One reason for this is that orderly civilian shift-work patterns are not applicable to the sustained-intensive battlefield. New work-rest guidelines based on the provision for short naps are required. While napping is generally accepted as a useful countermeasure

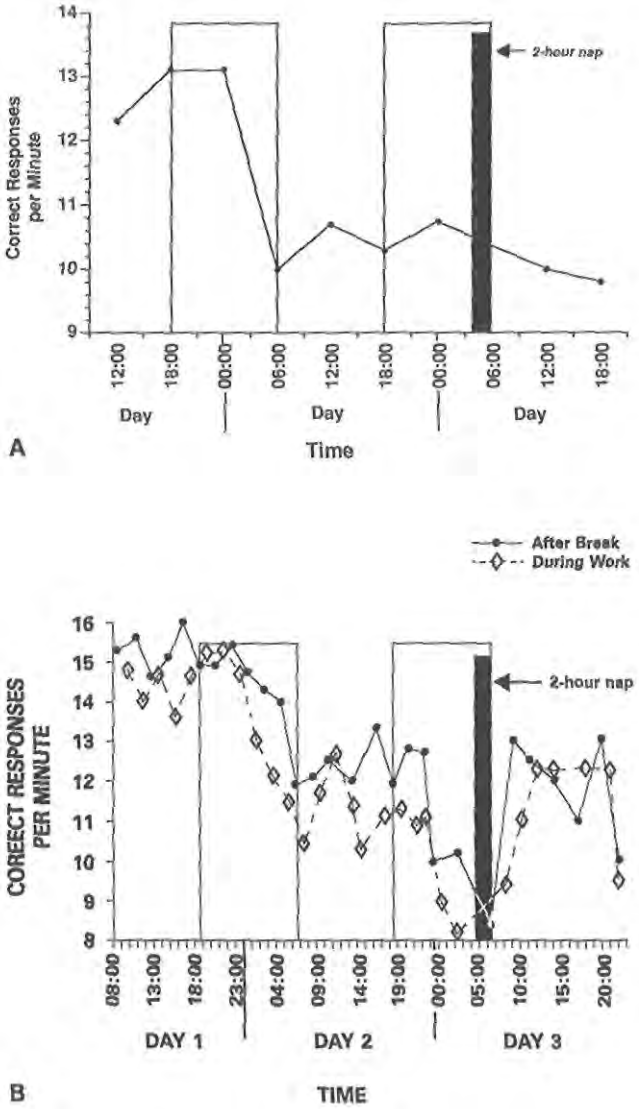

FIGURE 14.13. Influence of 0400–0600 nap after 46 hr of sleep deprivation on logical reasoning performance. A: 10-min tests every 6 hr. B: 2-min tests every 1 hr.

for increasing alertness, there are several factors that must be understood before napping guidelines can be provided. In planning for sustained operations it is important to know the minimal amount of nap sleep required to maintain acceptable performance. The specification of how long one needs to nap, however, may be complicated by the interaction of nap duration with the circadian cycle and the amount of prior wakefulness.

Another important concern is the generality of laboratory findings to battlefield conditions. For the most part, experimental subjects have lead relatively tranquil, low-demand existences. Although performance assessment may have been done at regular intervals, the level of performance required of subjects in past experiments did not demand sustained-intensive attention.

The present research program was designed to address the limitations of previous work. In particular, an environment was developed in which many cognitive tests and measures were embedded in a long and intense duty cycle. Experiments conducted in this environment have indicated that the combination of sleep loss and intense mental work leads to greater decrements in performance than in studies not emphasizing cognitive demands: substantial decrements occur following 18 hr on duty (reductions of about 30%) with generally unacceptable performance occurring 24 hr later (Angus and Heslegrave, 1985; Heslegrave and Angus, 1985). Also, the efficacy of short-duration measurement probes was demonstrated and applied in assessments of various countermeasures for sleep loss.

Some of our results suggest that certain countermeasures against sleep loss are not effective. Subjects' fitness levels, scheduled physical exercise, short bursts of strenuous physical exercise, or periods of low workload do not have long-term beneficial effects. However, short-term ameliorative effects owing to rest breaks are evident in all our studies. Performance and mood scores are consistently better immediately after breaks than an hour into the work sessions. The breaks seem to have a short-lasting positive influence on the subjects, and may provide a means by which temporary increases in performance can be effected during sustained operations. Additionally, both evening (2200-2400) and morning (0400-0600) naps were beneficial following 40 and 46 hr of sleep loss, respectively. This implies, at least for these amounts of sleep loss, that the beneficial effects of naps may override circadian influences. When sleep loss is less severe, naps may provide a maintenance function; when sleep loss is more severe, naps may provide a recuperative function.

Overall, the results demonstrate the efficacy of our experimental paradigm: the continuous work environment amplifies sleep loss effects; the high frequency of testing yields finer and more sensitive results; and the military context in which the experiments are performed increases the validity of the results for operational situations. Efforts are now underway to increase the realism of our laboratory scenario further through the use of war-game techniques.

Research to this point (ours as well as others) has emphasized the effects of sleep loss on *groups* of subjects. A further goal must be to extend these results to *individual* subjects. Our future research will emphasize real-time interventions, first in the laboratory and then in the field. For example, subjects may be given naps at times not dictated by a fixed experimental protocol, but at times dependent upon their individual performance. This

will result in different subjects receiving naps at different times. The goal is to let subject parameters (in real time) control certain intervention aspects of the experiment.

To facilitate this approach, physiological data are also being collected in our experiments (e.g., ECG, EEG, respiration, and actigraphy). Although not mentioned in this chapter, we have found that EEG activity tracks both performance and mood scores throughout sleep loss. In our most recent experiment, we included an "eyes-closed" relaxation period in the Scales & Battery package presented each hour. During this period, levels of drowsiness were measured subjectively by questionnaire and objectively with EEG (e.g., reduced alpha, and increased levels of theta and delta activity). For individual subjects, these EEG-defined drowsiness levels correlate highly with declining performance and mood scores (Pigeau et al., 1987). Also, preliminary results from sleep EEG and ECG, during both the nap and the recovery night sleep, suggest that electrophysiological indices (e.g., EEG delta activity, increased heart rate, and decreased heart rate variability) may exist for determining the amount of sleep necessary to restore performance. Should these measures prove reliable, intervention research will have direct operational consequences. It may be possible, at the level of the individual, not only to specify the appropriate times for restorative naps but also to estimate the optimal durations of these naps.

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V

Polyphasic Behavior, Napping, and Sleep Disorders

15

Napping Behavior in Narcoleptic Patients: A Four-Hour Cycle in Slow Wave Sleep

M. BILLIARD, J. DE KONINCK, D. COULOMBE, AND A. TOUZERY

The issue of whether adult humans may have an endogenous, though masked, polyphasic sleep-wake tendency is of major importance in view of the proposal that people undergoing prolonged periods of work use polyphasic sleep-wake schedules. This question has been approached in several ways: by looking at sleep patterns of animals living in dangerous environments (Mukhametov et al., 1977; Pilleri, 1979); by having adult human subjects maintain different ultradian schedules (Weitzman et al., 1974; Carskadon and Dement, 1975; Moses et al., 1975; Lavie and Scherson, 1981; Lavie and Zomer, 1984); by submitting subjects to conditions of disentrainment (Campbell, 1983); and by observing the effects of self-imposed ultrashort sleep-wake schedules on yachtsmen engaged in prolonged single-handed sailing races (Stampi, 1985).

Another possible approach to this issue is the use of a pathological model of sleep-wake alternation, namely, narcolepsy. Using pathology as a means of understanding physiology is quite common. Knowledge of the behavior of lower and upper respiratory muscles during sleep has been greatly advanced by observation of subjects with the obstructive sleep apnea syndrome, for example, while the physiology of penile tumescence during sleep has benefited from the recording of subjects with impotency of various etiologies. Narcolepsy is characterized by two main symptoms: excessive daytime sleepiness culminating in irresistible sleep episodes and cataplexy —an abrupt and reversible decrease or loss of muscle tone most frequently elicited by emotion. In addition, subjects may experience hypnagogic hallucinations —vivid perceptual experiences occurring at sleep onset—and sleep paralysis —a transient, generalized inability to move or to speak during the transition between wakefulness and sleep. Nocturnal sleep is often disrupted. The symptoms of interest in our context are excessive

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daytime sleepiness and disturbed nocturnal sleep. Excessive daytime sleepiness alternates with normal alertness. It usually recurs several times a day both in the morning and in the afternoon. It may last from a few minutes to several tens of minutes. In some cases the subjects will fall asleep while riding a bike, while teaching a class, or even while performing surgery. The duration of the episode may vary from a few minutes if the subject is in an uncomfortable position to over 1 hr if he is reclining. The narcoleptic subject characteristically wakes up feeling refreshed and alert, and then experiences a refractory period to sleepiness of one to several hours before he is again sleepy. Disturbed nocturnal sleep consists of multiple awakenings throughout the night. Sleep talking is especially frequent in narcoleptic subjects. Associated periodic leg movements are frequent and sleep apneas are not uncommon.

The positive diagnosis of narcolepsy-cataplexy relies on the observed presence of excessive daytime sleepiness and attacks of cataplexy, and in documenting sleep onset REM (SOREM) episodes. A normal subject will always fall asleep in NREM sleep. Originally a single nap with a SOREM episode was considered enough to diagnose narcolepsy. But the finding that a SOREM episode may occasionally be encountered in normal subjects in situations such as shift work or transmeridian flights, or in sleep apneic subjects, led to more stringent criteria. Now subjects must show two or more SOREM episodes on the Multiple Sleep Latency Test (MSLT), a procedure in which the subject is given five opportunities to sleep during the daytime (Richardson et al., 1978). Another procedure is to have subjects continuously recorded for periods up to 36 or 48 hr either in the laboratory, with conventional polysomnography, or in their normal environment by means of an ambulatory recording system. In either case, subjects are instructed not to resist sleep. The second procedure has the advantage of documenting the actual number, duration, and type of sleep onsets of daytime sleep episodes. Furthermore, it provides the opportunity to investigate the presence of rhythms of sleep and wakefulness in narcoleptic subjects.

The first studies of this kind were probably those conducted by Passouant et al. In a first study (Passouant et al., 1968) 5 narcoleptic subjects were polygraphically recorded for 24 hr. The number of daytime sleep episodes varied from 3 to 5. For 1 subject the periodicity of sleep episodes was close to 2 hr (1 hr 49 min-2 hr 16 min). In an attempt to evaluate the influence of rest on sleep episodes 2 subjects were asked to take a reclining position every 2 hr. Both fell asleep in all six opportunities and one of them systematically entered REM sleep. This finding was interpreted in favor of a 2-hr periodicity of REM sleep throughout the 24 hr. In a further study (Passouant et al., 1969), two rhythms were evidenced: an ultradian rhythm of REM sleep with a period of about 1.7 hr and a circadian rhythm of REM sleep, NREM sleep, and wakefulness with an acrophase of REM sleep at -61°, an acrophase of NREM sleep at -32°, and an acrophase of wakefulness at -219°.

Subsequently, Baldy-Moulinier et al. (1976) gathered all the studies that had been performed in the Montpellier sleep laboratory according to different protocols of continuous polysomnography. The circadian influence upon the peak of REM sleep did not differ from that found in normal controls.

Later it was shown by Schulz (1985) that sleep or REM sleep episodes during daytime occurred at intervals that were similar to the REM-NREM intervals of night sleep, a result supporting the Basic Rest-Activity Cycle hypothesis (Kleitman, 1963). However, the degree of regularity of the sleep-wake and REM-NREM cycle during daytime clearly differed between the subjects. In addition, a phase continuity between REM sleep in the night and sleep episodes in the morning was evidenced. Finally, De Koninck et al. (1986), based on 24-hr continuous polysomnographic recordings of 12 narcoleptic subjects, provided statistical confirmation of the synchronization of daytime and nighttime REM episodes with periodicities of 100-115 min.

More recently, a circasemidian rhythm of sleep and especially of NREM sleep stages 3 and 4 [also referred to as slow wave sleep (SWS)] was found in narcoleptic subjects submitted to 24-hr sleep-wake monitoring either of the conventional type (Billiard et al., 1986) or of the ambulatory type (Broughton et al., 1988), thus supporting Broughton's initial hypothesis of a 12-hr bimodal ultradian rhythm governing NREM sleep stages 3 and 4.

The pathophysiology of excessive daytime sleepiness and irresistible episodes of sleep is still a matter of controversy. Following the discovery that REM sleep appears at the initial sleep onset (Rechtchaffen et al., 1963; Takahashi and Jimbo, 1963), the disease was first considered to be one of REM sleep exclusively (Dement et al., 1966). But according to the definition drafted at the first International Symposium on Narcolepsy, held in July 1975 at La Grande Motte, France, the disorder is not considered as a disease of REM sleep but as a disease of both wakefulness and sleep including pathological manifestations of REM sleep. Another pathophysiological orientation has been that narcolepsy-cataplexy could be caused by disorders of rhythmic sleep organization (Kripke, 1976). Narcoleptic subjects display an apparent loss of monophasic sleep pattern with intrusion of sleep during the daytime and of wakefulness at night. Narcolepsy frequently starts after shift work or following an abrupt modification of the sleep-wake schedule. SOREM episodes, although a special feature of narcolepsy, may be encountered in situations such as shift work, transmeridian flight, ultradian regimens, and other wakefulness and sleep patterns that cause a disturbance of the sleep-wake rhythm. However, major circadian rhythms, such as body temperature, REM sleep distribution over the 24 hr, and Cortisol levels, are not modified or only slightly modified in narcolepsy. Furthermore, the overall sleep architecture of narcoleptic subjects is very close to that of normal subjects. Billiard et al. (1986) found that their REM sleep occupied approximatively 22% of total sleep time during night sleep and 24% of sleep time during spontaneous daytime sleep. Similarly, stages 3 and 4, or

SWS, percentages were at 10% both at night and during the day. More recently, taking into account the multiple varieties of dissociated sleep seen in narcolepsy, cataplexy (an intrusion of REM sleep muscle atonia in wakefulness), sleep paralysis (a perception of REM sleep muscle atonia at the beginning of sleep), episodes of EMG suppression recorded in stage 2, episodes of persistence of EMG activity during a typical episode of REM sleep (Montplaisir et al., 1978), and other factors, it has been proposed that narcolepsy could be a disorder of state boundary control (Broughton et al., 1986).

Whatever the pathophysiology of narcolepsy-cataplexy, it is clear that the condition offers a special model of sleep-wake alternation in which subjects experience recurrent daytime episodes of sleep. These episodes are embarrassing for the subjects, yet they are refreshing. This is the common experience of narcoleptic subjects, demonstrated in many different types of studies. In one study of subjects with narcolepsy the restorative quality of sleep was investigated, both subjectively and objectively, after uninterrupted versus interrupted naps (Billiard, 1976). Subjective ratings of the restorative quality of naps were higher ($p < .01$) after uninterrupted naps (mean duration: 67 min 18 sec) than after interrupted naps (mean duration: 10 min 30 sec). In a second study the restorative quality of sleep was also investigated, both subjectively and objectively, in narcoleptic and control subjects, after 5 equally spaced naps taken throughout the day. Subjectively and objectively, control subjects did not benefit by having naps (with or without sleep). But narcoleptic subjects did clearly benefit by taking naps, but only if naps consisted of NREM sleep or of NREM and REM sleep. Similarly, Godbout and Montplaisir (1986) reported improved performances following 20-min MSLT naps in narcoleptics on a psychomotor task (four-choice reaction time test). Furthermore, NREM naps tended to lead to better performances than REM naps.

Altogether these data support the assumption that narcoleptic subjects may benefit from a polyphasic cycle consisting of multiple sleep-wake episodes. Moreover, the positive effect of NREM sleep is stressed.

Encouraged by the fruitful investigations of circadian and ultradian sleep components in the narcoleptic situation, we decided to pursue further our approach and focus on the components of REM sleep and SWS in 34-hr polysomnographic recordings of narcoleptic subjects in an ad lib protocol.

Method

Subjects and Procedure

Eighteen narcoleptic subjects, 15 males and 3 females, aged 16 to 66 yr (median: 47 yr), all showing two or more sleep onset REM episodes on a previously performed MSLT and all HLA DR2-DQ1 positive, were in-

eluded in the study. Subjects remained in the same room for 34 hr. During the daytime the room was lit with natural light. Polysomnography was started at 2200 with the subjects in bed and the lights turned off. In the morning, lights were turned on at the subject's request. From then on subjects were instructed not to resist sleep. A long cable between the subject's head and the head box allowed the subject to move around, sit at a table or in an armchair, or to lie in bed. In order to promote the occurrence of sleep episodes, subjects were allowed to read magazines, but not to watch television, listen to the radio, read books, or engage in personal business. Meals or snacks were brought to the subjects at about 800, 1200, 1600, and 1900, but were delayed if the subjects were sleeping. Otherwise no contact was made with the subjects. At night, lights were turned off at the subject's request or progressively by means of a rheostat, if the subject was already asleep at 2200. Polysomnography was concluded at 0800 on the second day.

Statistics

Durations of NREM sleep stages 1, 2, 3, and 4, of REM sleep, and of total sleep time were computed for the first night (2200-0800) and for the following day (0800-2200). Data from night 2 were not used, but polysomnography was continued. Thus there was no contamination of evening states of alertness because of an expected departure. A correlation analysis (Pearson) and a Mest (Student) were performed on the night and day sleep parameters. In addition, the amounts of NREM sleep stages 1, 2, 3, and 4, REM sleep, and total sleep were plotted hourly for the 24-hr period. Time series and spectral analysis were performed to look for potential rhythms of NREM sleep stages 3 and 4 and of REM sleep.

Results

Comparative Patterns of Nocturnal and Daytime Sleep

Table 15.1 shows the unusually large amount of sleep during the first 24 hr (495 ± 6.05 min at night and 268 ± 10.48 min at day). The percentage of stage 1 sleep was high during both night and day, reflecting the poor quality of sleep; the percentage of stage 2 sleep was somewhat higher during the night than during the day, while the reverse was true for stages 3 and 4. The percentage of REM sleep was remarkably constant during night and day, but with a high variance during the day due to the fact that REM sleep did not occur in all naps. The differences between sleep percentages were not significant. Nocturnal and diurnal total sleep times were positively correlated.

TABLE 15.1. Nocturnal and diurnal distribution of sleep stages[a]

Stages	Night (220–800)	Day (0800–2200)	t	p	r	p
TST (min)	495 ± 6.05	268 ± 10.48	4.06	.001	.48	.05
Stage 1 (%)	21.04 ± 5.85	22.01 ± 8.05	0.50	NS	.42	.06
Stage 2 (%)	52.11 ± 7.85	46.09 ± 14.26	1.87	NS	.19	NS
Stages 3–4 (%)	5.64 ± 4.15	8.99 ± 6.22	2.00	NS	.35	NS
REM (%)	21.21 ± 5.62	22.91 ± 15.87	0.48	NS	.34	NS

[a]Key to column headings: t = Student's t-test; r = Pearson correlation.

Visual Analysis

Inspection of the amounts of NREM sleep stages 1, 2, 3, and 4, of REM sleep, and of total sleep time plotted every hour around the clock revealed interesting findings. Total sleep time per hour was at its peak between 0300 and 0500 (see Figure 15.1), stage 1 between 0300 and 0400 and later between 0700 and 0800 (see Figure 15.2), stage 2 from 2300 to 0100 (see Figure 15.3), stages 3 and 4 at the onset of night sleep between 2200 and 2300 (see Figure 15.4), and REM sleep between 0400 and 0500 (see Figure 15.5). All sleep stages exhibited a secondary peak in the afternoon: stages 3 and 4 between 1300 and 1400 (see Figure 15.4), total sleep time, stages 1, 2, and REM sleep

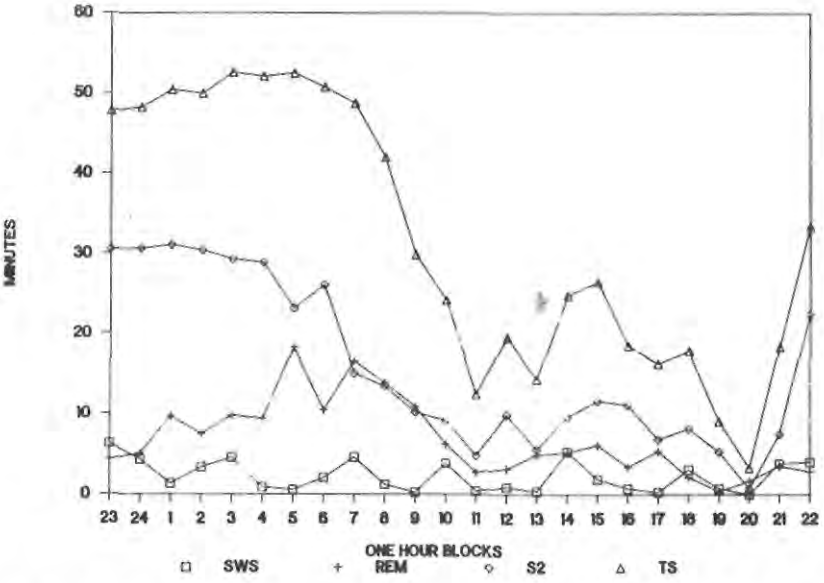

FIGURE 15.1. Twenty-four-hour distribution of total sleep (TS), stage 2 (S2), stages 3–4 (SWS), and REM sleep (REM) within 1-hr blocks.

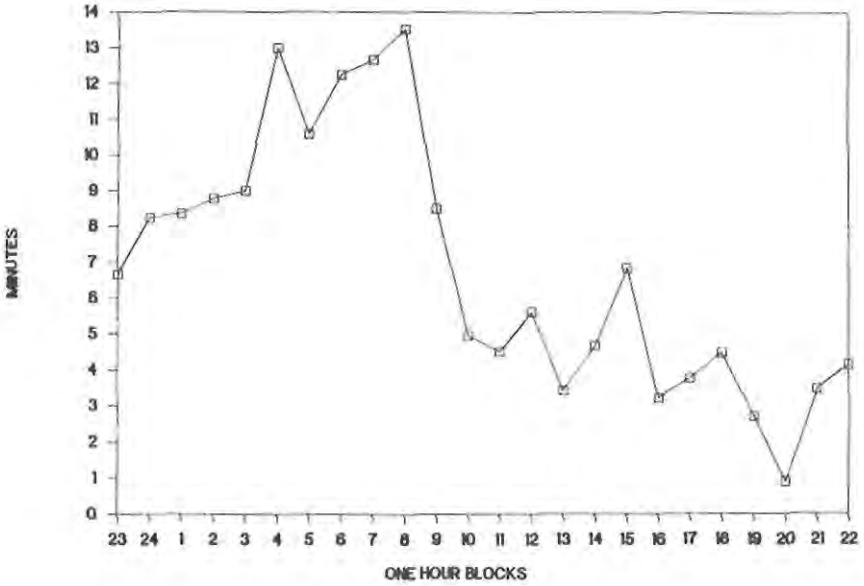

FIGURE 15.2. Twenty-four-hour distribution of stage 1 (close-up) within 1-hr blocks.

between 1400 and 1500 (see Figure 15.1). Thus, there was an interval of 15 hr between the two peaks of stages 3 and 4 and a shorter interval of 10 hr between the two peaks of REM sleep. Corresponding to these two peaks were two troughs. The main one occurred in the evening between 1900 and 2000 for total sleep time and between 1800 and 1900 for REM sleep. The second trough was more complex, including a first component between 1000 and 1100 and a second one after lunch between 1200 and 1300 (see Figure 15.1). In addition to this circasemidian rhythm apparent for all sleep stages, stages 3 and 4 showed additional fluctuations with peaks occurring about every 4 hr (see Figure 15.4).

Time Series Analyses

The 24-hr recordings were divided into 144 miniepochs of 10 min (lags). Calculation of autocorrelation coefficients revealed a clear pattern of coefficients indicating a periodicity of approximatively 4 hr. More specifically, three peaks of significant coefficients were present with highest values at lag 24 or at 4 hr ($r = .281$, $p < .001$), lag 42 or 7 hr ($r = .284$, $p < .001$), and lag 66 or 11 hr ($r = .215$, $p < .01$). A significant autocorrelation was also observed at lag 48 or 8 hr ($r = .182$, $p < .05$). A spectral analysis confirmed the 4-hr (240-min) and 8-hr (480-min) components.

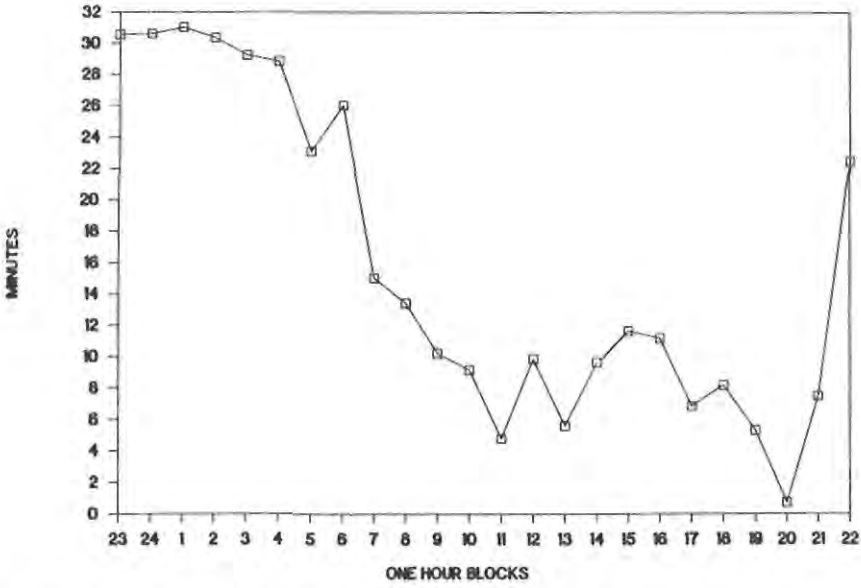

FIGURE 15.3. Twenty-four-hour distribution of stage 2 (close-up) within 1-hr blocks.

Similar analyses on the distribution of REM sleep revealed a pattern of autocorrelation coefficients with significant peaks at lags 11 ($r = .481, p < .001$) and 23 ($r = .204, p < .01$), corresponding to periodicities of a little less than 2 and 4 hr, respectively. Spectral analysis confirmed a 100-min component.

Discussion

Nocturnal and Diurnal Sleep Patterns

In contrast to previous reports (Berti-Ceroni et al., 1968; Montplaisir et al., 1978), our data show that narcoleptic subjects restricted to a sleep laboratory environment exhibit an increased amount of total sleep over the course of 24 hr (495 min nighttime + 268 min daytime). In a previous study including 36 narcoleptic subjects continuously recorded for 34 hr, we found a mean total sleep time of 722 min over the course of the first 24 hr. However, the subjects were systematically awakened at 0730 the first morning and their activity was not restricted to reading magazines. This finding underscores the previously reported influence of recording conditions on total sleep duration (Schulz, 1985). In addition, our results show a

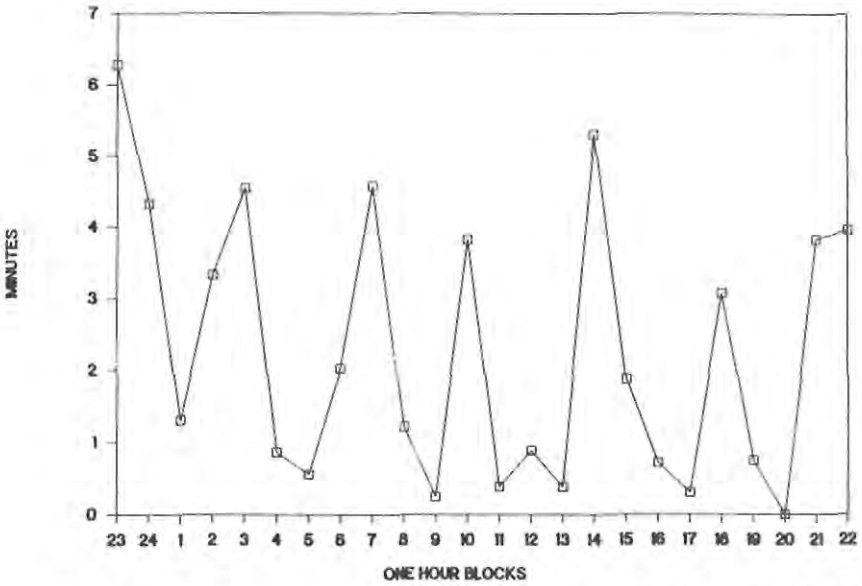

FIGURE 15.4. Twenty-four-hour distribution of stages 3–4 (SWS) (close-up) with 1-hr blocks.

positive correlation between nocturnal and diurnal sleep. In other words, subjects with the longest total sleep time at night are also those with the longest total sleep time during the daytime. This is likely to prove a good index of the severity of the illness. Finally, the high proportion of stage 1 sleep during both night and day points to the equal disruption of sleep during both periods.

Visual Analysis

Our data add further support to Broughton's hypothesis of a circasemidian rhythm governing stages 3 and 4 (Broughton, 1975) and the various sleep stages (Broughton et al., 1988). They are also in agreement with the proposals of a "forbidden zone" for sleep between 2000 and 2200 (Lavie, 1986) and of two wake-maintenance zones between 1000 and 1100 and between 2100 and 2200 (after approximate conversion from circadian phases to clock hours) (Strogatz et al., 1987). It is also worth mentioning that the first sleep stages to peak in the afternoon are NREM sleep stages 3 and 4, suggesting that these stages of sleep are needed the most after a period with relative little sleep. It would be interesting to test the recuperative value of naps at this time of the day compared to other periods.

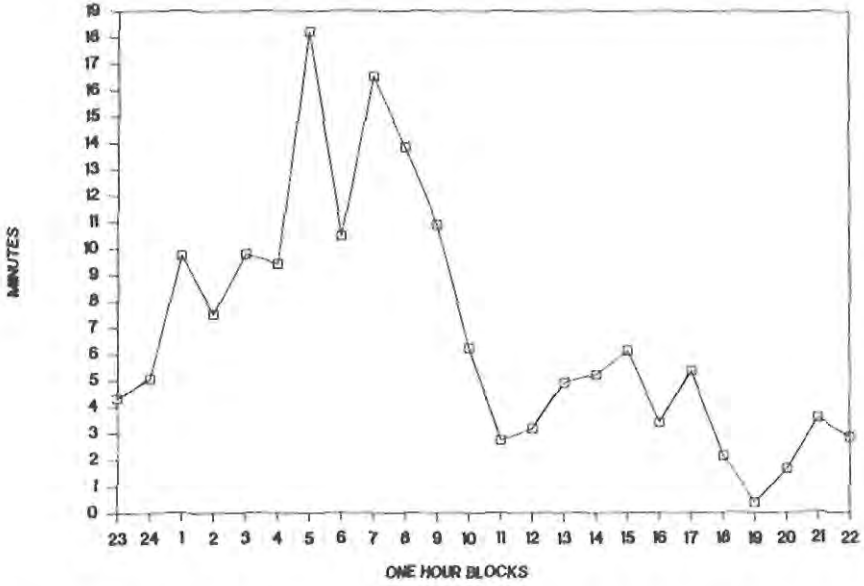

FIGURE 15.5. Twenty-four-hour distribution of REM sleep (close-up) within 1-hr blocks.

Time Series Analyses

Our data demonstrate the presence of a 4-hr rhythm of NREM sleep stages 3 and 4 in narcoleptic subjects. To the best of our knowledge, this rhythm has not been described previously. Yet other evidence of about 4-hr rhythms of sleep have already been reported. Infants in their first month of life display a 3- to 4-hr rhythm of sleep-wake behavior (Meier-Koll et al., 1978). A 4-hr sleep-wake cycle has been recognized in subjects asked to remain reclining on a bed in a supine position for 24 hr (Nakagawa, 1980). Fluctuations in "sleepability" of about 3.5 hr have been noted in subjects undergoing an ultrashort sleep schedule of 15 min waking and 5 min sleeping (Lavie and Scherson, 1981; Lavie and Zomer, 1984). A 4-hr sleep-wake rhythm was described in subjects on "constant bedrest" (Zulley, 1988). Finally, Stampi et al. (1990) found in a pilot study that their subject was able to adapt well to an extremely sleep-reduced schedule (3 hr per day) for 3 wks and later for almost 2 months, by dividing sleep into six 30-min naps, one every 4 hr. The fact that narcoleptic subjects exhibit a more even distribution of SWS across the night (Broughton and Mamelak, 1980) may explain why a 4-hr rhythm of SWS became more evident in our subjects. Thus it is very likely that a 3- to 4-hr sleep-wake rhythm is present in humans and is made apparent in different experimental and pathological conditions.

Given the importance attributed to SWS by some theorists (for example, Horne, 1988), an interesting question arises as to whether the SWS component of naps in narcoleptic subjects is significant for their recuperative value. We mentioned earlier that naps containing NREM sleep have proven to be more valuable than those without NREM (Billiard, 1976; Godbout and Montplaisir, 1986). But what about naps with SWS as a component of NREM sleep? Testing performance on psychomotor or attention tasks such as those used in the above studies but following naps placed at peaks of SWS in comparison to those taken at troughs of SWS would provide useful information. Specifically, comparing the recuperative effects of naps of equal lengths but containing varying amounts of SWS would clarify the issue.

The second finding of this study, the presence of a 100- to 110-min rhythm of REM sleep in narcoleptic subjects, confirms the results of previous studies (Passouant et al., 1969; Baldy-Moulinier et al., 1976; De Koninck et al., 1986) and supports Kleitman's (1963) notion of a Basic Rest-Activity Cycle (BRAC).

In summary, these data support the notion that the human subject has the capacity to adapt to ultrashort schedules of sleep and wakefulness. Our findings disclose a tendency for two types of sleep, NREM sleep stages 3 and 4 (SWS) and REM sleep, to occur cyclically with different periods. It is possible that the restorative effect of sleep is dependent upon the 4-hr rhythm of NREM sleep stages 3 and 4.

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16

Narcolepsy and the Pathological Aspects of Multiple Napping

HARTMUT SCHULZ, JOHANNA WILDE-FRENZ, STEPHAN VOLK, AND PETER GEISLER

The Polyphasic-Monophasic Sleep-Wake Continuum

The placement and duration of sleep within the nychthemeron is under the control of (1) homeostasis, (2) circadian rhythms, (3) individual demands and habits, and (4) environmental conditions. The first two factors are the basis for the two-process model of sleep (BorbSly, 1982). Factor 3 was added by Webb (1988) as an essential behavioral component. Both this third factor and the fourth one are optional factors for sleep-wake regulation. According to the relative strength of each single factor and to their specific combination, very different temporal distributions of sleeping and waking can result.

Under a *phylogenetic* perspective, two strikingly different sleep-wake patterns can be discriminated, namely, a polyphasic one with many short sleep bouts in 24 hr, and a monophasic one with one long sleep episode. Biphasic (dawn/dusk) sleep-wake patterns are usually interpreted as a variant of the monophasic type. Humans and other primates such as apes and monkeys, display monophasic sleep-wake patterns with occasional naps (Tobler, 1989).

During *ontogeny* the sleep-wake pattern of humans develops from a polyphasic one in infancy, through a biphasic one with a regular nap in childhood, into the predominantly monophasic sleep-wake distribution of adulthood. It is mainly via the optional factors mentioned above that even in human adults the monophasic pattern can be transformed into a polyphasic one. Thus, for example, continuous bed rest is a condition that effectively dismantles the monophasic sleep-wake cycle (Campbell, 1984). But even if a destructuring of the monophasic sleep-wake cycle is allowed or explicitly demanded by the experimenter, subjects exhibit a bimodal

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distribution of sleep durations, which are either longer than 5 hr (night sleep) or shorter than 4 hr (nap sleep) with very little or no overlap between the two parts of the overall distribution (Zulley, 1988).

Although there are many occurrences of nap sleep in addition to night sleep in human adults (see Dinges and Broughton, 1989), we would like to stress the concept that the human adult basically exhibits a well-consolidated monophasic sleep-wake cycle.

The Sleep-Wake Distribution in Narcoleptic Patients

In contrast to normal human subjects, narcoleptic patients suffer from a pronounced inability to maintain a monophasic sleep-wake cycle. These patients complain of having lost the ability to stay awake whenever they want to. The destruction of the monophasic sleep-wake cycle is due to three factors: i

1. Excessive daytime sleepiness (EDS), which causes many long periods of reduced vigilance. At these times the patient has difficulties in concentrating, even if the situation demands a high degree of attention.
2. Overt episodes of daytime sleep, whereby the patient has no effective means to stay awake, even in situations when sleep is socially unacceptable or dangerous. In addition, situational factors like monotony, which may induce sleepiness even in normals, are especially deleterious for narcoleptic patients.
3. Night sleep disturbances with frequent sleep interruptions, some of them rather long. The great majority of patients complain of this symptom.

Table 16.1 shows standard sleep parameters that indicate the reduced sleep efficiency of narcoleptic patients in comparison to age-matched normal controls. The distribution of sleep and wakefulness within 24 hr with sleep bouts during daytime and waking spells during nighttime suggests that the patients are caught in a vicious circle that prevents both effective sleeping and effective waking. To test this hypothesis, a single case experiment with daytime sleep deprivation was conducted.

Daytime Sleep Deprivation in Narcolepsy

A 42-yr-old, unemployed, male patient who suffered from EDS, sleep attacks, cataplexy, and hypnagogic hallucinations was studied for 60 consecutive days in the clinic. The core of the investigation was an extended episode (20 days) of daytime sleep deprivation. This episode was preceded by a 7-day wash-out phase subsequent to drug treatment with clomipramin

TABLE 16.1. Comparison of night sleep data between 10 unmedicated narcoleptic patients and 10 age-matched normal subjects

Sleep parameters	Patients	Controls
TIB (min)	473.7 ± 17.6	481.3 ± 22.4
TST (min)	429.6 ± 37.2	457.3 ± 15.1
SEI (%)	90.6 ± 5.6	95.1 ± 2.3

	Latencies (min)	
	Patients	Controls
S1	4.4 ± 4.4	10.1 ± 5.9
S2	21.6 ± 15.0	13.9 ± 7.0
S3	38.8 ± 14.7	28.7 ± 12.4
S4	26.0 ± 26.0	45.0 ± 74.8
REM	24.8 ± 40.3	78.9 ± 19.2

	Sleep stages (amounts)					
	Minutes		% TIB		% TST	
	Patients	Controls	Patients	Controls	Patients	Controls
W	44.1 ± 25.7	24.0 ± 12.7	9.4 ± 5.6	4.9 ± 2.3	–	–
S1	93.8 ± 32.7	28.0 ± 13.3	19.8 ± 6.8	5.8 ± 2.8	21.8 ± 6.8	6.1 ± 2.9
S2	202.6 ± 42.9	228.6 ± 38.7	42.8 ± 8.9	47.7 ± 8.7	47.3 ± 9.5	50.1 ± 9.2
S3	29.8 ± 20.9	42.1 ± 17.2	6.3 ± 4.4	8.8 ± 3.6	7.2 ± 5.2	9.2 ± 3.7
S4	19.7 ± 30.4	50.0 ± 40.9	4.1 ± 6.2	10.3 ± 8.2	4.3 ± 6.5	10.8 ± 8.6
REM	79.2 ± 17.1	102.4 ± 18.5	16.7 ± 3.4	21.3 ± 3.9	18.4 ± 3.2	22.4 ± 3.9
MT	4.4 ± 3.4	6.1 ± 2.6	0.9 ± 0.7	1.3 ± 0.5	1.0 ± 0.8	1.3 ± 0.5
Pause	4.3 ± 5.7	–		–		–

TIB = time in bed; TST = total sleep time; SEI = sleep efficiency index; W = time awake; MT = movement time.

(Anafranil®, 25 mg/day) and followed by a 15-day postintervention episode. Later the patient was first treated with the anticholinergic drug metixen (Tremarit®, 5–25 mg/day), and thereafter with protryptilin (Maximed®, 10 mg/day). Here, only data from the day sleep-deprivation phase will be presented; the effects of the pharmacological treatments will be discussed elsewhere.

Scheduled bed rest with polygraphic recordings was between 2300 and 0700. During the daytime the patient was continuously observed by four accompanying persons taking turns who tried to prevent him from falling asleep by different activities, such as talking, playing, and walking outside. Sleepiness was rated every 15 min on visual analog scales by the patient as well as by one of the observers. There were many occasions where brief sleep attacks could not be avoided. In addition, the patient frequently appeared to be half-asleep and responded only slowly. As Table 16.2 indicates, self-reported sleep duration was less than 30 min during the whole

TABLE 16.2. *Daytime sleep deprivation in a 46-year-old narcoleptic male patient*

Days	Treatment	Self-reported duration of daytime sleep (min)	Night sleep		
			Sleep efficiency index (%)	S3 + S4 (min) (median)	Subjective sleep quality[a]
1–3	Drug treatment Clomipramin (Anafranil® 25 mg/d)	–	86 ± 6	54.3	6.5 ± 0.1
4–10	Drug withdrawal wash-out	–	79 ± 5	70.0	5.3 ± 1.2
11–17	Daytime sleep deprivation	<30	87 ± 5		3.2 ± 1.5
18–24	Daytime sleep deprivation	<30	89 ± 3	78.5	3.0 ± 0.8
25–30	Daytime sleep deprivation	<30	86 ± 3		2.7 ± 0.9
31–37	Postintervention (1st wk)	115 ± 46	80 ± 4	74.0	4.3 ± 1.8
38–45	Postintervention (2nd wk)	136 ± 55	82 ± 2		3.4 ± 0.8

[a] 0 = very good; 10 = very poor.

daytime sleep-deprivation period. This contrasted with the estimated mean day sleep times exceeding 100 min in all other parts of the study. Sleep efficiency was significantly higher during the daytime sleep-deprivation period than in the prior drug-withdrawal period or the subsequent postintervention period. These data suggest that the amount and continuity of night sleep are responsive to the amount of day sleep in narcoleptic patients.

Figure 16.1 displays the temporal distribution of REM sleep episodes for all 60 nights. Sleep onset REM episodes (SOREMs) were ubiquitous with the exception of the times with tricyclic medication. The anticholinergic drug metixen (nights 46-55) did not prevent the occurrence of SOREMs but obviously reduced the amount of REM sleep in the following 3 hr. This inhibition of REM sleep was followed by relatively high amounts of this sleep stage late in the nights.

From a clinical point of view, the treatment was not very effective, since (1) the patient showed many episodes of severe daytime sleepiness throughout the 3-wk deprivation period, and (2) there was no "transfer" of reduced daytime sleep or increased nighttime sleep efficiency into the postdeprivation period. Interestingly enough, none of the other symptoms of the patient (cataplexy, hypnagogic hallucinations) was either aggravated or improved by the sleep-deprivation procedure.

These results show that even an extended period with a strictly administered sleep-wake schedule was not sufficient to readapt a narcoleptic patient to a monophasic sleep-wake cycle. Nevertheless, the increased amount of

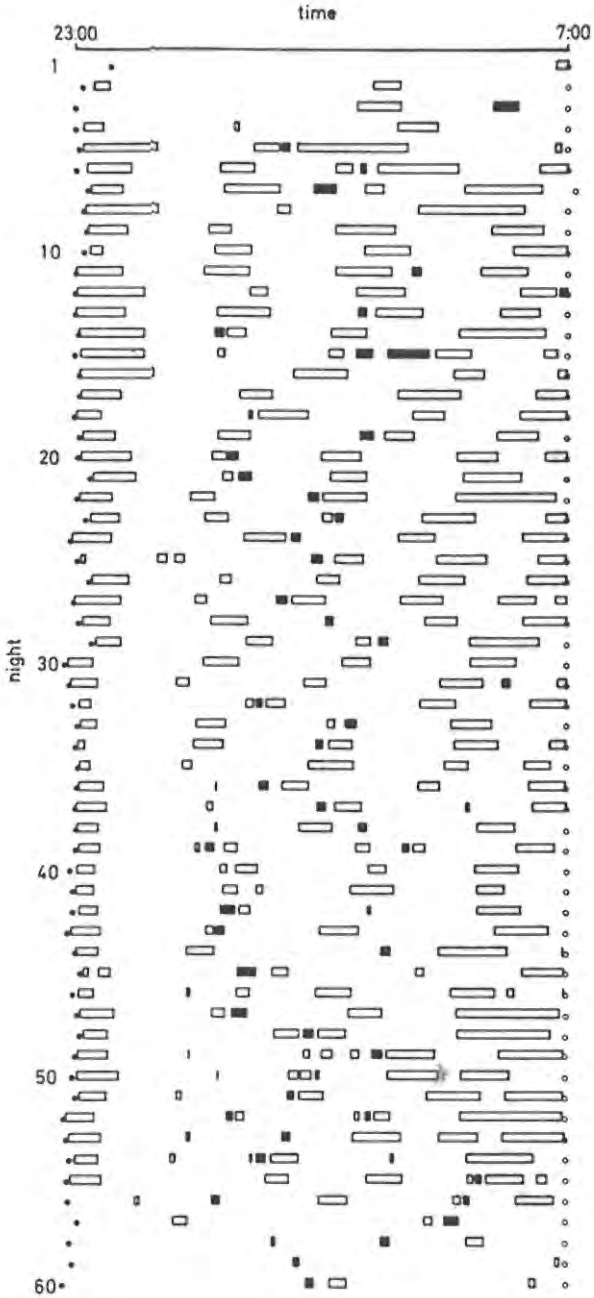


FIGURE 16.1. Distribution of REM sleep episodes during 60 consecutive night sleep recordings in a narcoleptic patient. For treatment conditions, see Table 16.2. Open bars represent REM sleep episodes, closed bars sleep interruptions (restroom visits).

nighttime sleep after daytime sleep reduction indicates that factors that regulate sleep homeostasis are effective in narcolepsy. The same seems to be the case with the homeostatic regulation of slow wave sleep in narcolepsy (Volk et al., 1992).

If this is true, a weakness of other factors involved in sleep regulation, such as circadian processes, could be the basis of the pathophysiology of narcolepsy (cf. Kripke, 1976). To test the hypothesis that the circadian timing system is less effective in narcoleptic patients, we decided to study the sleep-wake behavior and other physiological variables of 2 patients under conditions of temporal isolation. Such experiments permit examination of the internal timing system after exclusion of all external time cues.

The Effect of Temporal Isolation

The first patient studied in a time-free environment (Andechs bunker facilities) was the same one who took part in the daytime sleep-deprivation study (see above). During the 3-wk observational period the patient showed a free-running rhythm of rectal temperature with a mean period of 24.7 hr. The sleep-wake distribution was rather similar to that under entrained conditions, with one major sleep episode within 24 hr and multiple naps during the subjective day. From the beginning of temporal isolation the temperature rhythm desynchronized from the major sleep episodes, since the mean duration of the sleep-wake cycle (32.9 hr) was substantially longer than the temperature rhythm (see Figure 16.2).

The second patient studied in temporal isolation was a 59-yr-old male patient with narcolepsy-cataplexy. Because of the severe form of the disease he was prematurely retired from work. Again, the sleep-wake cycle and other physiological variables were observed for 3 wk. While in the first week the alternation of wakefulness and the major sleep episodes was close to 24 hr, in the last week the sleep-wake cycle was clearly shorter than 24 hr and close to 21.5 hr (see Figure 16.3).

Both cases studied in temporal isolation exhibited free-running sleep-wake cycles with a clear separation of long sleep episodes (subjective night sleep) and shorter daytime sleep attacks. While in the first patient a desynchronization between the sleep-wake rhythm and the rhythm of body temperature could be established, such an analysis could not be performed for the second patient because his temperature curve was highly irregular due to masking effects.

More patients will have to be studied under temporal isolation to explore the coherence of the internal circadian timing system and its role in the disease process. A high frequency of sleep episodes during the subjective days in temporal isolation has also been reported for narcoleptic patients by Pollak et al. (1987).

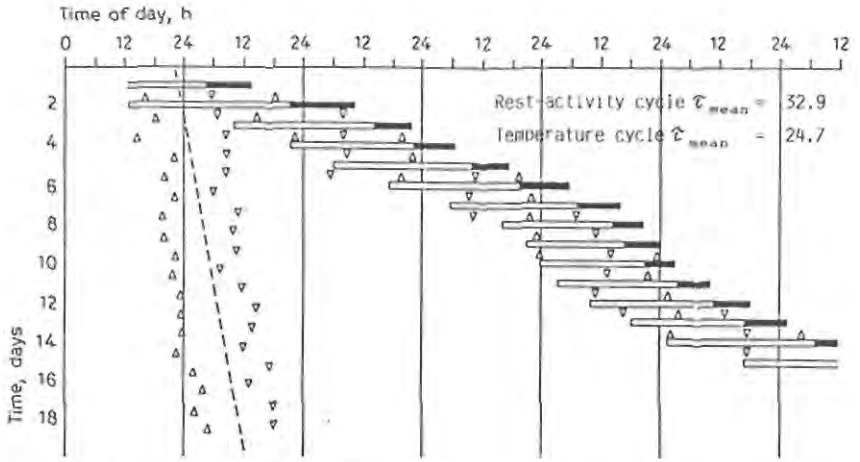


FIGURE 16.2. Free-running rhythms of sleep-wake and deep body temperature in a 42-yr-old male narcoleptic patient who lived in temporal isolation for 3 wk. Sleep is represented by the black part and wakefulness by the white part of the horizontal bars. The triangles pointing upward represent the circadian maxima of rectal body temperature, while those pointing downward represent the temperature minima.

Performance Deficits in Narcoleptic Patients

In this last section the deleterious effects of sleepiness and daytime sleep attacks on performance will be explored.

Although drops in vigilance and performance deficits are disabling consequences of the disease, few systematic studies on this topic have been published (Billiard, 1976; Valley and Broughton, 1983; Meier-Ewert, 1983; Levander and Sachs, 1985; Godbout and Montplaisir, 1986). Of these studies only one (Godbout and Montplaisir, 1986) tested performance, in this case forced-choice reaction time, at different times of the day. We used the critical flicker fusion (CFF) test to determine vigilance, because this test allows repeated measurements at short intervals. In addition, in our experience this test is sensitive enough to detect vigilance deficits in narcoleptic patients. The apparatus used (ZAK Ltd., Simbach/Inn) offers 50 stimuli per session, for 4 sec each, in a randomized order. During each stimulus presentation four light dots are exposed to the subject simultaneously within a dark tube. One of the dots is the critical (flickering) stimulus. The other dots are steady. The task of the subject is to move a pointer next to the critical stimulus by turning a handbar within 4 sec. From the total of 50 runs the detection threshold (in Hz) is computed.

Each patient or control subject was tested at 15-min intervals for 10 hr between 0800 and 1800, equaling 40 test sessions.

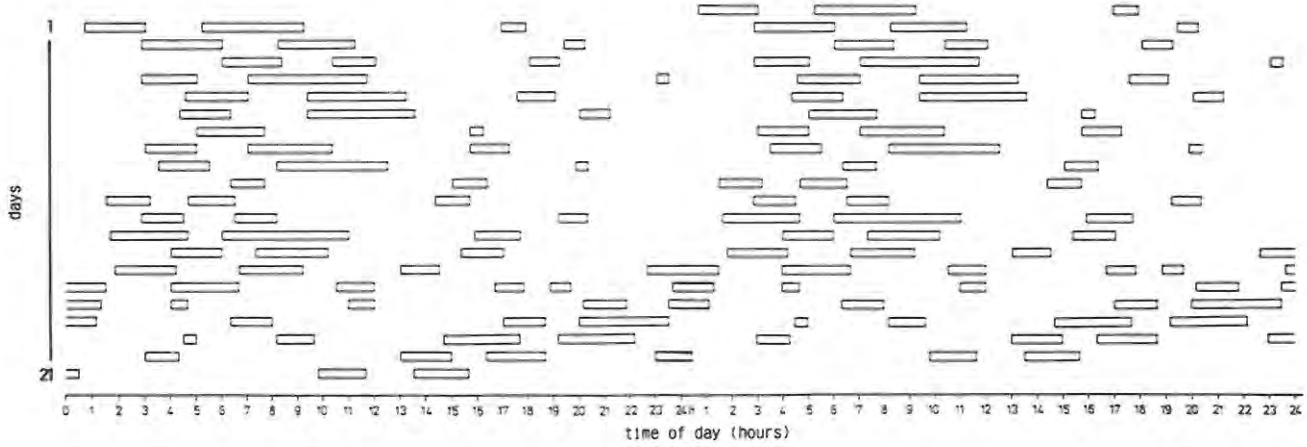


FIGURE 16.3. Double plot of the sleep episodes (subjective nights and naps) of a 59-yr-old male narcoleptic patient living in temporal isolation for 3 wk. Time of day (in hours) is given on the abscissa; days in the experiment are given on the ordinate.

Ten unmedicated narcoleptic patients (4 males, 6 females, mean age: 42 yr, range: 31–58 yr) were compared with 10 age- and sex-matched normal control subjects (4 males, 6 females, mean age: 42 yr, range: 30–60 yrs).

Table 16.3 gives the mean performance data and the standard deviations for each of the 20 subjects. As can be seen, the mean performance rate of the group of narcoleptic patients is slightly lower than that of the control subjects ($\bar{x}_N = 26.68$ vs $\bar{x}_C = 28.61$). This effect is mainly caused by the inability of the patients to perform on a stable level during the 10-hr test session. This is reflected in the significantly greater variances in the narcoleptic patients (see Figure 16.4). While all standard deviations in CFF performance were < 1 in the control subjects, all standard deviations of the patients were > 1, and the group mean was more than double that of the normal subjects.

Figure 16.5 shows three time courses of CFF performance, one from a normal subject and two from narcoleptic patients. While the majority of CFF values of the control subject fall into a small range (27–28 Hz), both patients show wide fluctuations. As indicated for both patients, the troughs in the performance curve occur at fairly regular intervals. The intervals between consecutive low performance values were 90 min (i.e., 6 measurement intervals) and 120 min (i.e., 8 measurement intervals), respectively, in these two cases. A detailed analysis of the data will be given elsewhere. However, it should be mentioned here that more patients displayed irregular than regular intervals between performance deficits.

These data demonstrate that the CFF test is a sensitive measure to detect fluctuations in cerebral vigilance in awake narcoleptic patients. Results from such a test could be validated against sleep propensity as measured by the Multiple Sleep Latency Test (MSLT).

TABLE 16.3. Critical flicker fusion thresholds (mean values and standard deviations) of 10 narcoleptic patients and 10 age-matched normal subjects

Patients		Controls	
Subject	*CFF threshold* [mean ± SD (min)]	Subject	*CFF threshold* [mean ± SD (min)]
DIN	23.6 ± 3.5	MWK	26.1 ± 0.81
PEN	22.7 ± 1.8	MUK	27.2 ± 0.90
DON	29.1 ± 1.2	KUK	27.0 ± 0.58
SIN	25.1 ± 1.8	DOK	29.0 ± 0.66
WON	25.8 ± 2.5	JOK	27.6 ± 0.75
HIN	25.7 ± 2.4	LEK	31.3 ± 0.81
BKN	28.9 ± 1.2	RUK	24.9 ± 0.92
HAN	23.3 ± 2.8	GEK	31.0 ± 0.60
SCN	27.9 ± 1.6	POK	30.4 ± 0.55
HON	31.8 ± 2.8	HLK	31.6 ± 0.55

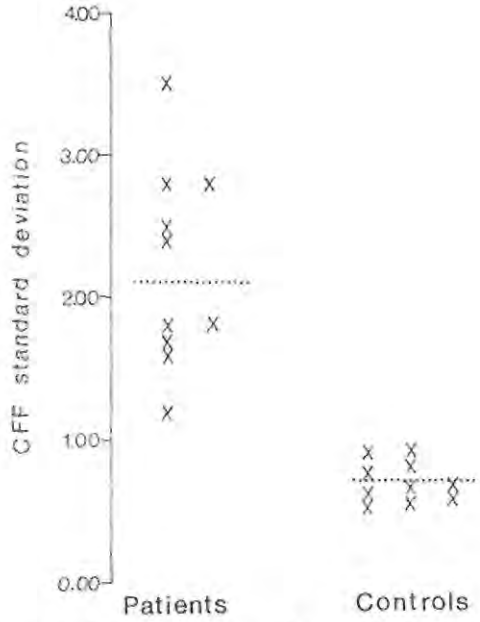


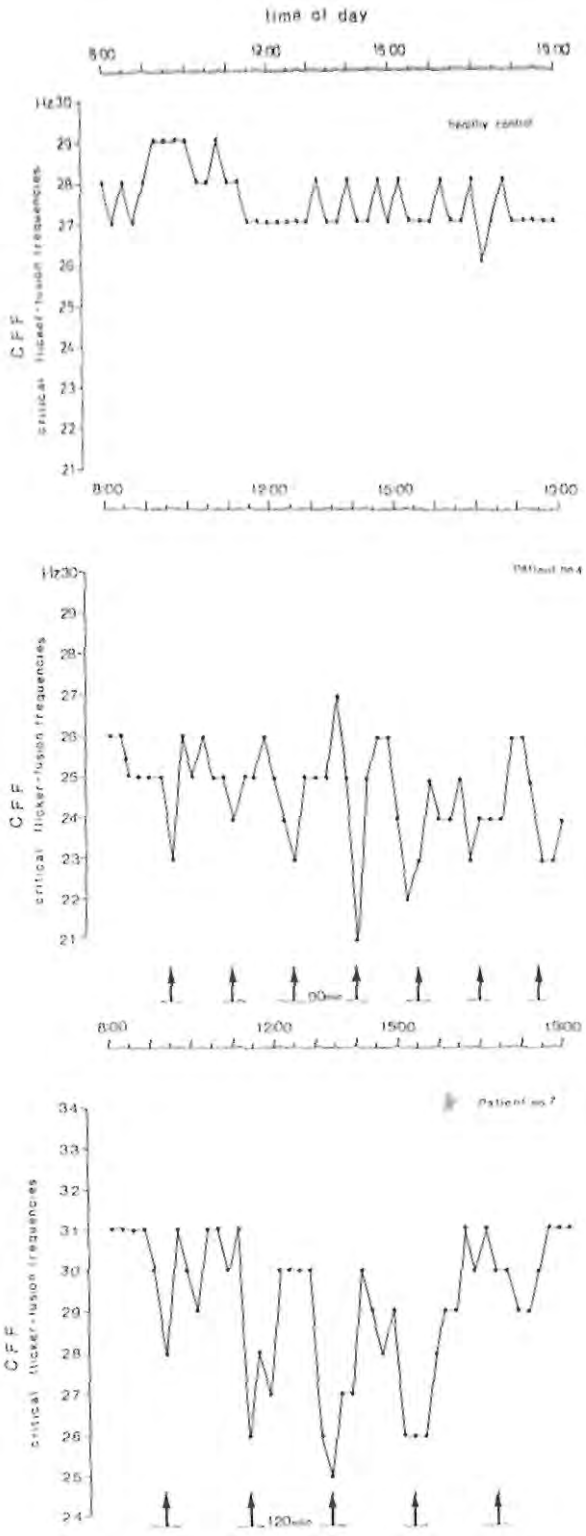
FIGURE 16.4. CFF standard deviations over 40 test sessions each for 10 narcoleptic patients and 10 matched normal control subjects.

Conclusions

Daytime sleep is a Janus-faced phenomenon. In the form of voluntarily placed naps it can be instrumental in functioning on a high level throughout the whole day. But if daytime sleep occurs involuntarily and is overwhelming, it has just the opposite effect. It then indicates, and may even contribute to, a state of insufficient functioning during the waking hours, as in different sleep-wake disturbances, especially narcolepsy.

In a series of diverse experiments some aspects of pathological sleepiness and napping in narcolepsy were studied. The flexibility and adaptability of the disorganized sleep-wake pattern to a strict monophasic rest-activity schedule was tested in a single case study by prolonged daytime sleep deprivation. Although sleep efficiency and the amount of slow wave sleep increased under this regimen, even 3 wks were not enough to transform the polyphasic sleep-wake pattern into a strictly monophasic one. This finding suggests that daytime sleep and nighttime wake episodes in narcolepsy may be the outcome of *a permanent state border control disturbance* (Broughton et al., 1986) rather than a sleep deficit or a sleep displacement.

This interpretation would also be in line with the results of the second set of experiments, where narcoleptic patients were observed under conditions of temporal isolation for 3 wk each. Both patients clearly differentiated


time of day
8:00
12:00
15:00
19:00
healthy control
C F F
critical flicker-fusion frequencies
Hz
30
29
28
27
26
25
24
23
22
21
8:00
12:00
15:00
19:00
Patient no 4
C F F
critical flicker-fusion frequencies
90min
8:00
12:00
15:00
19:00
Patient no 7
C F F
critical flicker-fusion frequencies
34
33
32
31
30
29
28
27
26
25
24
120min

between subjective night sleep and nap sleep. This could be seen from the sleep-wake recordings and the diary entries. One interpretation would be that the strength of narcoleptics' internal circadian pacemakers is not much less than that of normal subjects. Otherwise one would have expected a much greater decay of the circadian component of the sleep-wake distribution in temporal isolation.

To make firm statements about the circadian periodicity of the body temperature and its phase relationship with the sleep-wake cycle, more patients will have to be studied. A detailed analysis of such data in narcoleptic patients is essential, since Zulley and Campbell (1985) have emphasized the role of naps for the concept of internal desynchronization.

Finally, the results of the CFF study show that the discrimination between sleeping and waking by electroencephalographic measures is not sufficient to represent the whole vigilance continuum. The method used here allowed the tracing of fluctuations of cerebral vigilance in narcoleptic patients, who for the most part would have been rated as awake according to polygraphic and behavioral criteria. It is of interest to note that some of the patients showed quite regular fluctuations of vigilance which fall into the range of Kleitman's Basic Rest-Activity Cycle (BRAC) theory (1982).

In the present context of polyphasic and ultrashort sleep-wake cycles this could mean that performance and EEG measures should be applied in combination to cover the whole vigilance continuum.

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FIGURE 16.5. Three time courses of CFF performance over 10 hr—*upper panel:* data of a nomal subject; *middle and lower panels:* data of two nacoleptic patients. Optical fusion threshold (Hz) is given on the ordinate. The arrows indicate equidistant rasters with 90-min intervals (*middle panel*) and 120-min intervals (*lower panel*). The rasters were consructed in such a way to give the best representation of local minima in the performance curves.

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