CHAPTER 3

(ERP) STUDIES OF COGNITION DURING SLEEP:

IS IT MORE THAN A DREAM?

MARTA KUTAS

Punctate stimuli presented to an awake human leave an electrical trail as they travel from sensory transducers through the central nervous system (CNS). This electrical signature, typically represented as a waveform of voltage in time, is known as the evoked response or potential (EP). Current-day dogma has it that the EP reflects the potential field created from the summation of the excitatory and inhibitory postsynaptic potentials synchronously activated by an incoming stimulus. With the discovery of new brain wave components, it has become clear that some waves are more intimately related to internal events (such as preparing to move) than to external stimuli. Hence, the term evoked was subsumed by the general term event-related (EP being expanded to ERP). The transient ERP comprises a series of negative and positive peaks identified by their time synchrony to an eliciting stimulus. Each peak (also referred to as a component) is identified by its polarity and latency relative to stimulus onset (although on occasion a psychological label finds itself unabashedly attached to an ERP component). An ERP component is further characterized by the relative distribution of its amplitude across the scalp and by its waveshape (i.e., morphology).

Over the past 20 years, much research has been aimed at discovering the specific functions that relate changes in these EP characteristics to variations in stimulus parameters such as intensity, rate of presentation, frequency, and modality (see Regan, 1972, 1989). More important for our present purpose, much energy has also been spent compiling a vocabulary of components that

describe many of the critical information-processing transactions in the brain that underlie cognition. A fuzzy but sizeable vocabulary exists. Thus, for example, some component changes have been related to selective attention (e.g., N1, Nd) and others have been related to anticipation and preparation (contingent negative variation [CNV] and readiness potential [RP]). Still others have been linked to sensory mismatches (N2-P3a), orienting (N2), preperceptual mismatches (mismatch negativity [MMN]), surprise (P3b), novelty (P3a), and semantic processing (N400) (Gaillard & Ritter, 1983; Hillyard & Picton, 1987; Johnson, 1988; Johnson, Rohrbaugh, & Parasuraman, 1987; Kutas & Van Petten, 1988).

One undeniable benefit of some ERP components as measures of human brain activity is that they can be recorded regardless of a person's level of arousal or mental state. Of course, this is not to say that a person's state may not alter the amplitude or latency of the electrical response recorded but rather that a subject need not be awake or fully conscious of the eliciting stimulus to generate a measurable response. Insofar as we know what process is indexed by a part of an ERP waveform, the differential behavior of the peaks and troughs of the ERP can be used to make inferences about the psychology and physiology of the brain, be it awake or asleep. However, insofar as the processes underlying the ERP are less clear, it may be necessary to vary arousal states to learn more about the mappings between ERP components and cognitive operations.

EVENT-RELATED POTENTIALS IN SLEEP

Given that electroencephalographic (EEG) criteria have proven indispensable for differentiating among the various sleep stages (e.g. Rechtschaffen & Kales, 1968), it is surprising how infrequently ERP measures have been chosen as a means of evaluating the psychological and computational capabilities of the brain during the different sleep stages. In part, this may reflect our intuitions and the prevailing belief that not much processing (especially of external stimuli) goes on during sleep. Then again, the lack of cognitive ERP studies during sleep may be a reaction to the difficulty of pinning a cognitive operation uniquely to a specific positive or negative wave, even under conditions in which subjects are unquestionably awake and behaving. If one is fairly certain that there is little in the way of cognition during sleep and one is equally uncertain about interpreting a measure of cognitive activity (within existing frameworks), then one would be a bit foolhardy to jump right into the research fray. Few have.

Indeed, most research utilizing ERP recordings during sleep has focused on clinical rather than cognitive uses of the technique. By this view, the ERP

I extend sincere thanks to C. Van Petten for her helpful comments on this manuscript. Research efforts were supported by a Research Scientist Development Award from the National Institute of Mental Health (MH00322) and by a grant from the National Institute of Child Health and Human Development (HD22614).

is a clinical tool that provides an index of the integrity of the sensory pathways in the central nervous system. For diagnostic purposes, the most informative EP components are those that are consistently elicited by specific stimulus parameters in the face of fluctuations in arousal or attention. Studies over the past 20 years have indicated that the auditory brain evoked potential (ABEP), also known as the auditory brain stem response (ABR) or the brainstem evoked response (BER), provides just such a response. The ABR consists of a series of small vertex-positive waves occurring within the first 10 ms after an abrupt stimulus (Campbell, Picton, Wolfe, Baribeau-Braun, & Braun, 1981; Chiappa, Gladstone, & Young 1979; Stockard, Stockard, & Sharbrough, 1978). Peak 5 of the response is usually recognizable to within 20 dB of the behavioral auditory threshold (Galambos & Hecox, 1978; Picton, Woods, Baribeau-Braun, & Healy, 1977; Picton, Stapells, & Campbell, 1981). While there is considerable intersubject variability in component amplitudes, the latencies of the BER peaks are remarkably stable across replications, provided that the subject's age, gender, and body temperature as well as the physical properties of the eliciting stimulus and recording parameters are held constant (e.g., Moore, 1983). Such reliability, in fact, has sanctioned the development of a normative data base against which deviant responses can be judged. Abnormalities in the timing of various components relative to norms signal a possible cause for concern in either audiological or neurological domains. With a few exceptions that have been difficult to replicate (Lukas, 1980, 1981), it has been demonstrated that the first five waves of the BER are insensitive to attentional manipulations in an awake adult (Picton & Hillyard, 1974; Woldorff, Hansen, & Hillyard, 1987; Woods & Hillyard, 1978). Thus, ABR measurement has become a routine means of objective audiometry, especially for subjects who are unwilling or unable to undergo traditional methods of examination (e.g., neonates, young children, demented individuals).

The insensitivity of the BER to arousal levels and attentiveness in awake subjects suggests that the BER should be the same regardless of whether the subject is awake or asleep. The literature on this question is, however, contradictory. For instance, Amadeo and Shagass (1973) reported latency shifts as large as 0.25 ms in different stages of sleep. In contrast, Hellekson, Allen, Greeley, Emery, and Reeves (1979) found no differences in the latency of any ERP components between waking and light naps. Osterhammel, Shallop, and Terkildsen (1985) noted only minor changes in the latency of Peak 5 for only some subjects and only for low intensity stimuli. Campbell and Bartoli (1986) suggested that such inconsistencies in the literature may be due to a number of factors including (a) failure to control the stimulus input (that is, using loud-speakers rather than headphones), (b) failure to control for covariation in core temperature during the night (Jones, Stockard, & Weidner, 1980; Marshall & Donchin, 1981; Stockard, Sharbrough, & Tinker, 1978), and (c) small sample size.

Campbell and Bartoli (1986) evaluated the BER at different intensities and rates of stimulus presentation during wakefulness, Stages 2 and 4, and

REM sleep while maintaining precise control over the stimulus input (with an ear-mold hearing aid device) and monitoring temperature in 9 female subjects (18–25 years old). They reported that regardless of (a) the stage of sleep, (b) the time of night, (c) the rate of stimulus presentation, and (d) the intensity of the stimulus, sleep did not appear to have a significant effect on any of the components of the click-evoked ABR. They concluded "that auditory information is transmitted without alteration from the periphery through the brainstem relay centres during sleep" (p. 146). Before we accept this conclusion, we must remember that the BER is a sensitive index of the timing operations of the auditory system but is silent about the processing of the contents of the auditory signal.

Components of the auditory evoked response occurring between 10 and 100 ms have also been called into service for clinical hearing assessments. These components, referred to as middle latency responses (MLR), come in the form of transient or steady-state responses. Transient responses are evoked whenever stimuli are presented at slow rates (10/s or less), whereas steadystate responses arise when stimuli are presented at such a high repetition rate that the responses to successive stimuli overlap (e.g., Stapells, Linden, Suffield, Hamel, & Picton, 1984). The auditory MLR has received considerable attention in audiometry because of the possibility that it might allow determination of frequency-specific auditory thresholds. And indeed, threshold estimates from MLRs correlate well with behavioral thresholds, with the estimates from the steady-state response being less variable than those derived from transient responses (Galambos, Makeig, & Talmachoff, 1981; Goldstein & Rodman, 1967; Klein, 1983; Mendel et al., 1975; Shallop & Osterhammel, 1983; Stapells, 1984). Moreover, the effects of stimulus rate on the amplitude of the steady-state MLR are quite similar during wakefulness and sleep. For example, although the amplitude of the response is smaller during sleep, the maximum amplitude is still recorded at stimulus rates between 30-50 tones/s. The amplitude of the response increases as the intensity of the stimulus increases. A similar stimulus-intensity/response-amplitude function occurs at all stages of sleep, although the slope of the suprathreshold intensity-amplitude relation is lower during sleep than waking. Likewise, a similar intensity phase (i.e., latency) relation occurs in all stages of sleep and wakefulness, with the phase of the response decreasing as the suprathreshold intensity of the stimulus increases. Thus, while the steady-state MLR is sensitive to changes between sleep and wakefulness, it is little affected by transitions across the different sleep stages (e.g., Linden, Campbell, Hamel, & Picton, 1985). As such, it holds promise as a viable technique for generating electrophysiological audiograms at different frequencies during sleep.

COGNITIVE EVENT-RELATED POTENTIALS

There are few reports comparing components of the auditory ERPs later than 100 ms to standardized stimuli in the different states of wakefulness and sleep. Although during sleep there seems to be an overall reduction in the amplitude

of electrical responses to sensory stimulation, this is not true of every component. Thus, it is important to consider different time regions of the ERP separately. For example, the amplitude of the N1 (around 100 ms) component is usually reduced during sleep (Anch, 1977; Bell & Campbell, 1988), whereas the amplitude of the following P2 component has been found to be highly variable (Buchsbaum, Gillin, & Pfefferbaum, 1975). The most profound change in the longer latency ERP components is the addition of a large negative N2 wave with a peak latency of 300–500 ms occasionally followed by a P3 component around 800 ms (e.g., Kevanishvili & von Specht, 1979; Ornitz, Ritvo, Carr, Panman, & Walter, 1967; Picton, Hillyard, Krausz, & Galambos, 1974; Ujszaszi & Halasz, 1986; Weitzman & Kremen, 1965; Williams, Tepas, & Morlock, 1962). Components of the auditory ERP later than 100 ms have been found to be too variable to be useful in the clinic.

These data clearly demonstrate that sensory stimuli are afforded some processing during sleep, often with stimulus–response functions, that resemble those of wakefulness. These observations, together with the data demonstrating that people are capable of making both simple and discriminative manual responses during sleep (Granda & Hammack, 1961; Williams, Morlock, & Morlock, 1966), suggest the possibility of applying cognitive ERP paradigms. Very few investigations of this type have been reported. Those that have have focused on ERPs associated with anticipation or with physical mismatches embedded in a stimulus sequence.

During wakefulness, to be forewarned to is to be forearmed. That is, being warned of an impending stimulus makes it possible to react more quickly and more accurately. In most theories, the facilitatory influence of the preparatory set on motor reactions and perceptual judgment is attributed to different subprocesses of attention. Does this adage hold for sleep as well? Is it possible to talk about different types of attention during sleep? In the waking adult, differential preparation is reflected in specific patterns of CNS activity that develop across preparatory intervals. For example, in both human and nonhuman animals it has been found that, during intervals of preparation and anticipation, there develops an expectancy wave; this potential is referred to as the contingent negative variation (CNV), contingent because it is assumed to index the contingency between the warning and imperative stimulus and variation because that is the way the British say potential.

Walter, Cooper, Aldridge, McCallum, & Winter (1964) first observed the CNV in a reaction time task with a fixed foreperiod between the warning stimulus and the imperative stimulus. Accordingly, they proposed that this cortical electronegativity reflected the priming of the frontal lobes for action. Current-day views of the CNV based on human and animal experiments also have proposed major involvement of the frontal lobes as well as the ascending reticular activating system (ARAS) in CNV generation and modulation (Skin-

¹ This N2 and P3 should not be mistaken for the N2 and P3 components that are referred to in the ERP studies of human information processing in awake adults.

ner & Yingling, 1977). Moreover, Marczynski (1978) argued that it is the cholinergic component of the ARAS that is involved in the genesis of surface negative potentials of this type.

Studies demonstrating that CNVs can be elicited without a motor response requirement have shifted the emphasis from response priming to mobilization of effort for anticipated activity, wherein the amplitude of the CNV indexes the total allowance of effort devoted to preparatory activity (for reviews see Hillyard, 1973; Tecce, 1972). Most investigations thus have emphasized the relation between the CNV and intensive (e.g., alertness, arousal, concentration, effort, expectancy) rather than selective aspects of attention. And to some extent this seems reasonable. Generally, attentiveness to the eliciting stimuli yields larger CNVs than distraction, more difficult tasks yield larger CNVs than easy tasks, and anticipation of noxious or informative stimuli yields larger CNVs than benign or uninformative stimuli (for a review, see Donchin, Ritter, & McCallum, 1978). However, the relation is by no means a simple one: Too difficult a task, too stressful a task, or too many simultaneous task requirements often yield a CNV of a diminished amplitude. Despite this complex function between CNV amplitude, attentional resources, and arousal (e.g., Tecce & Hamilton, 1973), it is possible to determine the relation under specified circumstances during waking and to contrast it with that obtained under the same conditions during sleep.

One such study was conducted by Salamy, Lester, and Jones (1975). In this experiment, 3 subjects were asked to respond (i.e., press a microswitch taped to their right hand) to white noise forewarned by a tone occurring 1 s ahead on 3 consecutive nights. Recordings were obtained prior to sleep, during REM sleep, and immediately after morning awakening. The results suggested that, although the subjects responded correctly 65% of the time in REM sleep, a CNV either did not develop or was dramatically attenuated during REM sleep. However, there are aspects of the data that warrant caution. For instance, it would be informative in interpreting the ERPs to compare the waveforms associated with a response against those elicited on no-response trials. Given the limited number of trials per average (n = 16), however, it is unlikely that the data could have been sorted on the basis of behavior (e.g., by reaction time). The noise level of the data presented, especially in the REM condition, indicates that caution is well-advised. Moreover, as the authors noted but dismissed, the reaction times during REM were significantly slower (five- to sixfold) than when the subjects were awake. While the relation between CNV amplitude and reaction time is typically not linear, very prolonged reaction times tend to be associated with very small CNVs. Finally, the recordings were restricted to two electrode locations, vertex (Cz) and the left occiput, each referred to the left mastoid. Given that the latter part of a CNV elicited by a simple, right-handed response typically has a slight contralateral predominance over the motor strip, recordings from the central scalp locations and over the left hemisphere might provide a more precise test of the hypothesis. In any case, a replication of this type of study as well as recordings of CNV in

a discriminative reaction time task would be worthwhile before we close the book on slow negative potentials and REM sleep.

The relation between CNV amplitude and non-REM (NREM) sleep has been studied by Bruneau, Martineau, Ragazzoni, and Roux (1980). Like the study of Salamy et al. (1975), the recordings were limited to the vertex and occiput (in this case the right occiput) referenced to the left earlobe. Recordings were taken when subjects were awake and while they were asleep. During sleep, three fifths of the recordings were obtained during Stages 1-2, approximately two fifths were obtained during Stages 2-3, and several more were obtained during Stages 3-4. The two conditions in which CNVs were anticipated involved a weak sound (i.e., click) followed 880 ms later by an ankle jerk or Hoffman reflex (a monosynaptic spinal segmental reflex triggered by electrical stimulation of the 1A fibers of the muscle spindles in the calf [soleus muscle]). The ankle jerk was elicited in one case by the fall of a hammer beside the Achilles tendon and in the other by electrical stimulation of the sciatic nerve. Given the lack of an explicit task, this is not the most auspicious paradigm for eliciting CNVs. Moreover, the trials in which the click was paired with an ankle jerk were interspersed with blocks in which the click and the ankle jerk were each presented alone in the series, without pairing. Again, this type of equivocation in the warning stimulus to imperative stimulus contingency has been shown to reduce CNV amplitudes somewhat, albeit most severely when the single and paired trials are interspersed within a single experimental run. Although the data from sleep and waking were collected in counterbalanced order across the subjects, there is no indication that order of presentation made a difference on the size of the effects. On the whole, it seemed as if a CNV developed between the warning stimulus and the imperative stimulus during waking, whereas during NREM sleep the results were more variable with a clear large positivity developing prior to the imperative stimulus in some cases. The authors suggested that all of the ERPs recorded during sleep were probably K complexes (biphasic, negative-positive) and that the slow potential changes obtained by the sound and ankle jerk pairing were abolished. Again, I believe that more testing with a design better suited for CNV-elicitation and a more detailed mapping of the potentials across the scalp are warranted before we generalize this conclusion. Given the controversy surrounding the existence of a CNV as a unitary phenomenon as opposed to a combination of an orienting response to the warning stimulus (O wave) and a preparation for movement in response to the imperative stimulus (E wave), it might also be informative to manipulate the foreperiod interval during sleep studies. This latter approach has been used to separate the two components in waking subjects (e.g., Rohrbaugh & Gaillard, 1983).

Thus, is being forewarned helpful in sleep as well? The answer, based on ERPs, is that we do not know. First, one would want to know whether a warned signal is responded to more quickly than an unwarned signal and whether the effect of warning varies with sleep stage. The results of such a behavioral study would aid in the interpretation of slow potential studies and

would be less costly to pursue. If the CNV requires active engagement of attentional mechanisms and this is not possible during sleep, then no CNVs will be expected. However, we must question whether the concept of attention can be invoked in the same breath as sleep. If the CNV is truly an indicator of CNS excitability, one might expect differences in CNV generation and maintenance between REM and NREM sleep. While it would not be surprising to find that CNVs are smaller during sleep than during waking, it is theoretically crucial to determine whether there are any significant differences in CNV amplitude, morphology, and scalp distribution during the different stages of sleep. The critical role of muscarinic cholinergic mechanisms both in REM sleep (e.g., Hobson, 1988) and in the generation of slow negative potentials such as the CNV (Marczynski, 1978; Pirch, Corbus, Rigdon, & Lyness, 1986) suggest that, if CNVs are to be recorded during sleep, they should vary across the stages, especially between REM and NREM. In addition, if the proposed involvements of REM sleep and CNV amplitude in some aspect of memory have some validity, one might expect differential effects of sleep stages on CNV generation and amplitude. Certainly, any study of these issues should attempt to gather the relevant ERP data from the same subjects during the different sleep stages under the same experimental conditions (difficult as this may be).

To date, intuition and evidence lead me to argue that the so-called cognitive ERP components require at least a modicum of attention for their elicitation. The most likely candidate as an exception to this rule is the MMN.² The MMN was first identified and was subsequently studied by Naatanen and associates (Naatanen, 1986; Naatanen & Gaillard, 1983; Naatanen, Gaillard, & Mantysalo, 1978; Naatanen, Sams, & Alho, 1986). The MMN is a negative component beginning around 100 ms after a stimulus change in a repetitive homogeneous stimulus stream, even when the change is not consciously perceived. That is, if subjects are asked to attend to one ear in a dichotic listening task and to ignore stimuli in the other ear, tones in the unattended ear that deviate from the ongoing sequence of tones elicit an MMN. The larger or more obtrusive the deviation and the less frequently it occurs, the larger the MMN. Naatanen has proposed that the MMN reflects the activity of an automatic, preperceptual neuronal mismatch process; namely, the neurophysiological basis of echoic memory in audition. The MMN has been found to be modality specific and is presumed to be independent of and insensitive to attentional manipulations.

The latter presumption was tested in a situation considered to provide "the most extreme case of attention to environmental stimuli, namely sleep" (Paavilainen et al., 1987, p. 247). Seven subjects who were instructed to wake up early in the morning took part in an experiment that same evening, during which they listened to tones as they read a book; gradually, they fell asleep.

² However, I could easily argue that it does not properly belong in the arsenal of cognitive components.

Tone bursts (50 ms of a sine wave at 75 dB SPL) were presented at a constant interstimulus interval of 510 ms. Ninety percent of the tones were 1000 Hz, whereas 10% were 1050 Hz. While awake, subjects read a book and ignored the tones; afterward they slept while sequences of 800 tones were presented. The ERPs to the frequent (i.e., standard) tones were characterized by a P1 and an N1 component. As had been previously reported, the N1 component was reduced significantly as the subjects grew drowsy, and it disappeared as they entered Stage 1 sleep. In contrast, the P1 component grew larger in amplitude with progressing stages of NREM sleep. The reading condition was as expected: The less frequent (i.e., deviant) stimulus was characterized by a large MMN peaking between 150 and 200 ms, followed by a P3a-like deflection between 250 and 300 ms. As drowsiness set in, 4 of the subjects continued to produce significant MMNs and a nonsignificant hint of the P3a. By Stage 1 sleep and thereafter, none of the ERPs to deviant tones contained an MMN. Three of the subjects continued to show a small, late positivity in response to the deviant stimulus during Stage 1 sleep. By Stage 2 sleep there were no differences in the ERPs elicited by standard and deviant tones. Although some data were recorded during Stages 3 and 4, the ERPs to deviant tones were described as distorted by "the low frequency and high amplitude brain waves characteristic to these sleep stages" (Paavilainen et al., 1987, p. 249). In addition, no ERPs were recorded during REM because the subjects' sleep architecture was abnormal. These peculiarities included longer-than-normal Stage 2, spontaneous awakening after one or two sleep cycles, and insufficient numbers of reliable periods of REM. This last observation aside, the results of this experiment clearly show that the MMN is not elicited by deviant stimuli during sleep.

It is important to comment that Paavilainen et al. (1987) were the first to propose that additional experiments with the mismatch negativity during sleep be carried out and to suggest that such follow-up experiments should use larger stimulus deviations. They based this recommendation on the fact that larger deviations are associated with larger MMNs and on the observation by Csepe, Karmos, and Molnar (1987) of an MMN (30-70 ms) during slow wave sleep in cats. Csepe et al. presented 3- and 4-kHz tones as deviant and standard tones, respectively, at a number of different targetto-standard probability ratios (five levels between 5% and 50%) and found MMNs of an increased latency at some recording sites but only at the lowest deviant probabilities. While it is sometimes difficult to compare ERP components across species, the cat recordings met enough of the necessary criteria to be considered similar to the MMN recorded in man. As such, they also suggested that Paavilainen et al.'s (1987) study with humans be modified and tried again. In addition to using a greater deviation as they proposed, I also recommend using (a) variable intervals between stimuli (e.g., between 300 and 500 ms), (b) recording epochs of at least 800 ms poststimulus, and (c) acclimating the subjects to sleeping in the laboratory so that their sleep patterns are normal.

FUTURE DIRECTIONS AND POSSIBILITIES

As is evident from this review of the scant literature on cognitive electrophysiological investigations of sleep, the door is wide open for studies of this type. What follows are some of my thoughts on possible directions to pursue.

Although the CNV requires that subjects somehow appreciate the contingency between two stimuli, there are other slow, long-lasting negative potentials of the same family that are easier to record. For example, recordings made prior to limb movements indicate the presence of a slow-rising negativity that begins in some cases as early as 1.5 s before movement onset (measured relative to the response in the moving muscles with electromyography [EMG]). This negativity, variously called the readiness potential, bereitschaftspotential, and N1 of the motor potential, is largest over the motor strip and, at least in its latter half, has a strong contralateral predominance (e.g., Kornhuber & Deecke, 1965). In a series of experiments, Libet (1985) made inferences about the timing of conscious awareness based on the onset time of the readiness potential relative to each subject's verbal report of the intention to move. Within an experimental psychology tradition, Coles and colleagues (e.g., Coles & Gratton, 1986; Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988; see also de Jong, Wierda, Mulder, & Mulder, 1988) have used the asymmetric portion of the readiness potential as a measure of differential preparation and have used it to make inferences about information flow through the black box we call a brain (Coles, Gratton, Bashore, Eriksen, & Donchin, 1985). Do we need to be paying attention to make use of partial information? What are the odds of recording RPs to spontaneous movement during sleep-during any stage? If a lucid dreamer can make voluntary movements based on presleep instructions, will these movements be preceded by a readiness potential? And if RPs are recorded during sleep, must we modify our conceptions of preparation and voluntariness?

The past 10 years of ERP research have witnessed a strong push in the areas of language processing and memory, sometimes in combination (Kutas, 1988; Kutas & Van Petten, 1988; Neville, Kutas, Chesney, & Schmidt, 1986). For example, ERPs to words and pictures show clear repetition effects (Nagy & Rugg, 1989; Rugg, 1985, 1987; Rugg, Furda, & Lorist, 1988; Rugg & Nagy, 1987). To what extent is attention necessary to yield this repetition effect? How will the brain respond to an auditory passage that it has been exposed to earlier in the day? How will it respond the next morning to a passage that it has heard during sleep, either slow wave or REM? Will anomalous sentences seem less strange to subjects if they have been experienced previously during sleep? Both behavioral and ERP measures may provide answers to such questions, albeit not necessarily the same ones. The ERP measure has on occasion proven to be more sensitive to implicit memories than to reaction time measures (e.g., Besson, Fischler, Boaz, & Raney, 1989). If forced to bet, I would put my money on the ability of implicit memories or procedural learning (linguistic and nonlinguistic) to penetrate the sleeping brain to a greater degree than those of an explicit or declarative nature. If semantic processing truly

53

reflects lexical processing without awareness, as claimed by some researchers (for a review, see Holender, 1986), then two associatively or semantically related words might prime each other during sleep as well. Examining ERP signs of semantic association during sleep might shed light on these issues (e.g., Kutas & Hillyard, 1989). Certainly, my views on language processing, ERPs, and sleep would be affected by empirical evidence on these matters. In fact, it is precisely because my intuitions run counter to any notion wherein people can simultaneously cogitate about external events and enjoy sleep that I have proposed ways to prove myself wrong.

References

- Amadeo, M., & Shagass, C. (1973). Brief latency click-evoked potentials during waking and sleep in man. *Psychophysiology*, 10, 244–250.
- Anch, M. (1977). The auditory evoked brain response during adult human sleep. Waking and Sleeping, 1, 189–194.
- Bell, I., & Campbell, K. (1988). In T. W. Picton (Ed.), Human event-related potentials. EEG handbook (Vol. 3, p. 364). Amsterdam: Elsevier.
- Besson, M., Fischler, I., Boaz, T. L., & Raney, G. (1989). Effects of automatic semantic priming on explicit and implicit memory tests. Manuscript submitted for publication.
- Bruneau, N., Martineau, J., Ragazzoni, A., & Roux, S. (1980). Event-related slow potentials evoked during ankle jerk conditioning in wakefulness and NREM sleep. *Electroencephalography and Clinical Neurophysiology*, 49, 93–101.
- Buchsbaum, M., Gillin, J. C., & Pfefferbaum, A. (1975). Effect of sleep stage and stimulus intensity on auditory average evoked responses. *Psychophysiology*, 12, 707–712.
- Campbell, K. B., & Bartoli, E. A. (1986). Human auditory evoked potentials during natural sleep: The early components. *Electroencephalography and Clinical Neuro*physiology, 65, 142–149.
- Campbell, K. B., Picton, T. W., Wolfe, R. G., Baribeau-Braun, J., & Braun, C. (1981). Clinical evoked potential studies: II. Auditory brainstem responses. *Sensus*, 1, 9–19.
- Chiappa, K. H., Gladstone, K. J., & Young, R. R. (1979). Brainstem auditory evoked responses: Studies of waveform variation in 50 normal human subjects. Archives of Neurology, 36, 81–87.
- Coles, M., & Gratton, G. (1986). Cognitive psychophysiology and the study of states and processes. In E. R. J. Hockey, A. W. K. Gaillard, & M. G. H. Coles (Eds.), *Energetics and human information processing*. Dordrecht, The Netherlands: Martinus Nijhof.
- Coles, M. G. H., Gratton, G., Bashore, T. R., Eriksen, C. W., & Donchin, E. (1985). A psychophysiological investigation of the continuous flow model of human information processing. *Journal of Experimental Psychology: Human Perception and Performance*, 11, 529–553.

³ In a fantasy world of science, I would also be interested in knowing whether segments of the EEG activity of any given individual recorded during the day would correlate highly with a segment of equal length recorded later that evening during REM sleep. That is, is there any evidence for the notion that certain processing sequences are replayed for the purpose of consolidation during sleep?

Csepe, V., Karmos, G., & Molnar, M. (1987). Evoked potentials correlates of stimulus deviance during wakefulness and sleep in cat-animal model of mismatch negativity. *Electrocheephalography and Clinical Neurophysiology*, 66, 571–578.

- de Jong, R., Wierda, M., Mulder, G., & Mulder, L. J. M. (1988). Use of partial stimulus information in response processing. *Journal of Experimental Psychology: Human Perception and Performance*, 14, 682–692.
- Donchin, E., Ritter, W., & McCallum, W. C. (1978). Cognitive psychophysiology: The endogenous components of the ERP. In E. Callaway, P. Tueting, & S. H. Koslow (Eds.). Event-related brain potentials in man (pp. 349–441). New York: Academic Press.
- Gaillard, A. W. K., & Ritter, W. (Eds.). (1983). Tutorials in event related potential research: Endogenous components. Amsterdam: Elsevier/North-Holland.
- Galambos, R., & Hecox, K. E. (1978). Clinical applications of the auditory brainstem response. *The Otolaryngologic Clinics of North America*, 11, 709–722.
- Galambos, R. S., Makeig, S., & Talmachoff, P. J. (1981). A 40-Hz auditory potential recorded from the human scalp. *Proceedings of the National Academy of Sciences*, 78, 2643–2647.
- Goldstein, R., & Rodman, L. (1967). Early components of averaged evoked responses to rapidly repeated auditory stimuli. *Journal of Speech and Hearing Research*, 10. 607–705.
- Granda, A. M., & Hammack, J. T. (1961). Operant behavior during sleep. *Science*, 133, 1485–1486.
- Gratton, G., Coles, M. G. H., Sirevaag, E., Eriksen, C. W., & Donchin, E. (1988). Preand poststimulus activation of response channels: A psychophysiological analysis. *Journal of Experimental Psychology: Human Perception and Performance*, 14, 331–344.
- Hellekson, C., Allen, A., Greeley, H., Emery, S., & Reeves, A. (1979). Comparison of inter-wave latencies of brain stem auditory evoked responses in narcoleptics, primary insomniacs and normal controls. *Electroencephalography and Clinical Neurophysiology*, 47, 742–744.
- Hillyard, S. A. (1973). The CNV and human behavior. *Electroencephalography and Clinical Neurophysiology*, Suppl. 33. Amsterdam: Elsevier.
- Hillyard, S. A., & Picton, T. W. (1987). Electrophysiology of cognition. In F. Plum (Ed.), Handbook of physiology, Section 1: The nervous system: Vol 5. Higher functions of the nervous system. Part 2 (pp. 519–584). American Physiological Society, pp. 519–584.
- Hobson, J. A. (1988). The dreaming brain. New York: Basic Books.
- Holender, D. (1986). Semantic activation without conscious identification. Behavioral and Brain Sciences, 9, 1–66.
- Johnson, R., Jr. (1988). The amplitude of the P300 component of the event-related potential: Review and synthesis. In P. K. Ackles, J. R. Jennings, & M. G. H. Coles (Eds.), Advances in Psychophysiology (Vol. 3, pp. 69–138). Greenwich, CT: JAI Press
- Johnson, R., Jr., Rohrbaugh, J. W., & Parasuraman, R. (Eds.). (1987). Current trends in event-related potential research. *Electroencephalography and Clinical Neuro-physiology*, Suppl. 40. Amsterdam: Elsevier.
- Jones, T. A., Stockard, J. J., & Weidner, W. J. (1980). The effects of temperature and acute alcohol intoxication on the brain stem auditory evoked potential in the cat. *Electroencephalography and Clinical Neurophysiology*, 49, 23–30.
- Kevanishvili, Z., & von Specht, H. (1979). Human slow auditory evoked potentials during natural and drug-induced sleep. Electroencephalography and Clinical Neurophysiology, 47, 280–288.
- Klein, A. J. (1983). Properties of the brain-stem slow wave component: 1. Latency, amplitude, and threshold sensitivity. *Archives of Otolaryngology*, 109, 6–12.
- Kornhuber, H. H., & Deecke, L. (1965). Hirnpotentialanderungen bei Wilkurbewegungen und passiven Bewegungen des Menschen: Bereitschaftspotential und re-

ies

55

movements in man: The readiness potential and reafferent potential]. *Pfluegers Archiv für Gesammelte Physiologie*, 284, 1–17.

- Kutas, M. (1988). Review of event-related potential studies of memory. In M. S. Gazzaniga (Ed.), Perspectives in memory research (pp. 181–218). Cambridge, MA: MIT Press.
- Kutas, M., & Hillyard, S. A. (1989). An electrophysiological probe of semantic association. *Journal of Cognitive Neuroscience*, 1, 38–49.
- Kutas, M., & Van Petten, C. (1988). Event-related brain potential studies of language. In P. K. Ackles, J. R. Jennings, & M. G. H. Coles (Eds.), *Advances in psychophysiology* (Vol. 3, pp. 139–187). Greenwich, CT: JAI Press.
- Libet, B. (1985). Unconscious cerebral initiative and the role of conscious will in voluntary action. *Behavioral and Brain Sciences*, 8, 529–566.
- Linden, R. D., Campbell, K. B., Hamel, G., & Picton, T. W. (1985). Human auditory steady state evoked potentials during sleep. *Ear and Hearing*, 6, 167–174.
- Lukas, J. H. (1980). Human attention: The olivo-cochlear bundle may function as a peripheral filter. *Psychophysiology*, 17, 444–452.
- Lukas, J. H. (1981). The role of efferent inhibition in human auditory attention: An examination of the auditory brainstem potential. *International Journal of Neuro*science, 12, 137-145.
- Marczynski, T. J. (1978). Neurochemical mechanisms in the genesis of slow potentials: A review and some clinical implications. In D. Otto (Ed.), *Multidisciplinary perspectives in event-related brain potential research* (pp. 25–36). Washington, DC: U.S. Government Printing Office.
- Marshall, N. K., & Donchin, E. (1981). Circadian variation in the latency of brainstem responses: Its relation to body temperature. *Science*, 212, 356–358.
- Mendel, M. I., Hosick, C. E., Windman, T. R., Davis, H., Hirsh, S. K., & Dinges, D. F. (1975). Audiometric comparison of the middle and late components of the adult auditory evoked potentials awake and asleep. *Electroencephalography and Clinical Neurophysiology*, 38, 27–33.
- Moore, E. J. (Ed.). (1983). Bases of auditory brain-stem evoked responses. New York: Grune & Stratton.
- Naatanen, R. (1986). Neurophysiological basis of the echoic memory as suggested by event-related potentials and magnetoencephalogram. In F. Klix & H. Hagendorf (Eds.), Human memory and cognitive capabilities (pp. 615–628). Amsterdam: Elsevier/North-Holland.
- Naatanen, R., & Gaillard, A. W. K. (1983). The orienting reflex and the N2 deflection of the ERP. In A. W. K. Gaillard & W. Ritter (Eds.), *Tutorials in event-related potential research: Endogenous components* (pp. 119–329). Amsterdam: Elsevier/North-Holland.
- Naatanen, R., Gaillard, A. W. K., & Mantysalo, S. (1978). The early selective attention effects on evoked potential reinerpreted. *Acta Psychologica*, 42, 313–329.
- Naatanen, R., Sams, M., & Alho, K. (1986). The mismatch negativity: The ERP sign of a cerebral mismatch process. In W. C. McCallum, R. Zappoli, & F. Denoth (Eds.), Cerebral psychophysiology, electroencephalography and clinical neurophysiology (Suppl. 38, pp. 172–178). Amsterdam: Elsevier.
- Nagy, M. E., & Rugg, M. D. (1989). Modulation of event-related potentials by word repetition: The effects of inter-item lag. *Psychophysiology*, 26, 431–436.
- Neville, H. J., Kutas, M., Chesney, G., & Schmidt, A. (1986). Event-related brain potentials during the initial encoding and subsequent recognition memory of congruous and incongruous words. *Journal of Memory and Language*, 25, 75–92.
- Ornitz, E. M., Ritvo, E. R., Carr, E. M., Panman, L. M., & Walter, R. D. (1967). The variability of the auditory averaged evoked response during sleep and dreaming in children and adults. *Electroencephalography and Clinical Neurophysiology*, 22, 514–524.
- Osterhammel, P. A., Shallop, J. K., & Terkildsen, K. (1985). The effect of sleep on the auditory brainstem response (ABR) and the middle latency response (MLR). *Scandinavian Audiology*, 14, 47–50.

Paavilainen, P., Cammann, R., Alho, K., Reinikainen, K., Sams, M., & Naatanen, R. (1987). Event-related potentials to pitch change in an auditory stimulus sequence during sleep. In R. Johnson, Jr., J. W. Rohrbaugh, & R. Parasuraman (Eds.), Current trends in event-related potential research (EEG Suppl. 40). Amsterdam: Elsevier/North Holland.

Picton, T. W., & Hillyard, S. A. (1974). Human auditory evoked potentials: II. Effects of attention. *Electroencephalography and Clinical Neurophysiology*, 36, 191–200.

- Picton, T. W., Hillyard, S. A., Krausz, H. I., & Galambos, R. (1974). Human auditory evoked potentials: I. Evaluation of components. *Electroencephalography and Clinical Neurophysiology*, 36, 179–190.
- Picton, T. W., Stapells, D. R., & Campbell, K. B. (1981). Auditory evoked potentials from the human cochlea and brainstem. *Journal of Otolaryngology*, 10, 1–41.
- Picton, T. W., Woods, D. L., Baribeau-Braun, J., & Healey, T. M. J. (1977). Evoked potential audiometry. *Journal of Otolaryngology*, 6, 90–119.
- Pirch, J. H., Corbus, M. J., Rigdon, G. C., & Lyness, W. H. (1986). Generation of cortical event-related slow potentials in the rat involves nucleus basalis cholinergic innervation. *Electroencephalography and Clinical Neurophysiology*, 63, 464–475.
- Rechtschaffen, A., & Kales, A. (1968). A manual of standardized terminology, techniques, and scoring system for sleep stages of human subjects. Washington, DC: U.S. Government Printing Office.
- Regan, D. (1972). Evoked potentials in psychology, sensory physiology and clinical medicine. London: Chapman & Hall.
- Regan, D. (1989). Human brain electrophysiology: Evoked potentials and evoked magnetic fields in science and medicine. Amsterdam: Elsevier/North Holland.
- Rohrbaugh, J. W., & Gaillard, A. W. K. (1983). Sensory and motor aspects of the contingent negative variation. In A. W. K. Gaillard & W. Ritter (Eds.), *Tutorials in event related potential research: Endogenous components* (pp. 269–310). Amsterdam: Elsevier/North-Holland.
- Rugg, M. D. (1985). The effects of semantic priming and word repetition on event-related potentials. *Psychophysiology*, 22, 642–647.
- Rugg, M. D. (1987). Dissociation of semantic priming, word and non-word repetition by event-related potentials. *Quarterly Journal of Experimental Psychology*, 39A, 123–148.
- Rugg, M. D., Furda, J., & Lorist, M. (1988). The effects of task on the modulation of event-related potentials by word repetition. *Psychophysiology*, 25, 55–63.
- Rugg, M. D., & Nagy, M. E. (1987). Lexical contribution to non-word repetition effects: Evidence from event-related potentials. *Memory and Cognition*, 15, 473– 481.
- Salamy, J., Lester, J., & Jones, K. (1975). REM sleep and contingent negative variation development. Electroencephalography and Clinical Neurophysiology, 39, 201– 204
- Shallop, J. K., & Osterhammel, P. A. (1983). A comparative study of measurements of SN-10 and the 40/sec middle latency responses in newborns. *Scandinavian Audiology*, 12, 91–95.
- Skinner, J. E., & Yingling, C. D. (1977). Central gating mechanisms that regulate event-related potentials and behavior. In J. Desmedt (Ed.), Progress in clinical neurophysiology: Attention, voluntary contraction, and event-related cerebral potentials (Vol. 1, pp. 30-69). Basel, Switzerland: Karger.
- Stapells, D. R. (1984). Studies in evoked potential audiometry. Unpublished doctoral dissertation, University of Ottawa, Canada.
- Stapells, D. R., Linden, R. D., Suffield, J. B., Hamel, G., & Picton, T. W. (1984). Human auditory steady state potentials. *Ear and Hearing*, 5, 105–113.
- Stockard, J. J., Sharbrough, F. W., & Tinker, J. A. (1978). Effects of hypothermia on the human brainstem auditory response. *Annals of Neurology*, *3*, 368–370.
- Stockard, J. J., Stockard, J. E., & Sharbrough, F. W. (1978). Nonpathologic factors influencing brainstem auditory evoked potentials. *American Journal of EEG Technology*, 18, 177–209.

Tecce, J. J. (1972). Contingent negative variation (CNV) and psychological processes in man. *Psychological Bulletin*, 77, 73–108.

- Tecce, J. J., & Hamilton, B. T. (1973). CNV reduction by sustained cognitive activity (distraction). In W. C. McCallum & J. R. Knott (Eds.), Event-related slow potentials of the brain. *Electroencephalography and Clinical Neurophysiology*, Suppl. 33, 229–237.
- Ujszaszi, J., & Halasz, P. (1986). Late component variants of single auditory evoked responses during NREM sleep Stage 2 in man. *Electroencephalography and Clinical Neurophysiology*, 64, 260–268.
- Walter, W. G., Cooper, R., Aldridge, U. J., McCallum, W. C., & Winter, A. L. (1964). Contingent negative variation: An electric sign of sensorimotor association and expectancy in the human brain. *Nature*, 203, 380–384.
- Weitzman, E. E., & Kremen, H. (1965). Auditory evoked responses during different stages of sleep in man. *Electroencephalography and Clinical Neurophysiology*, 18, 65–70.
- Williams, H. L., Morlock, H. C., & Morlock, J. U. (1966). Instrumental behavior during sleep. *Psychophysiology*, 2, 208.
- Williams, H. L., Tepas, D. I., & Morlock, H. C., Jr. (1962). Evoked responses to clicks and electroencephalographic stages of sleep in man. *Science*, 138, 685–686.
- Woldorff, M., Hansen, J. C., & Hillyard, S. A. (1987). Evidence of selective attention in the midlatency range of the human auditory event-related potential. In R. Johnson, J. W. Rohrbaugh, & R. Parasuraman (Eds.), *Current trends in event-related potential research* (pp. 146–154). Amsterdam: Elsevier.
- Woods, D. L., & Hillyard, S. A. (1978). Attention at the cocktail party: Brainstem evoked responses reveal no peripheral gating. In D. A. Otto (Ed.), Multidisciplinary perspectives in event-related brain potential research (pp. 230–233). Washington, DC: U.S. Government Printing Office.