

PSYCHOPHYSIOLOGICAL ANALYSIS OF  
MEMORY FUNCTION DURING  
THE SLEEP ONSET TRANSITION

by

James Kelley Wyatt

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A Dissertation Submitted to the Faculty of the

DEPARTMENT OF PSYCHOLOGY

In Partial Fulfillment of the Requirements  
For the Degree of

DOCTOR OF PHILOSOPHY

In the Graduate College

THE UNIVERSITY OF ARIZONA

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and recommend that it be accepted as fulfilling the dissertation requirement for the Degree of Doctor of Philosophy

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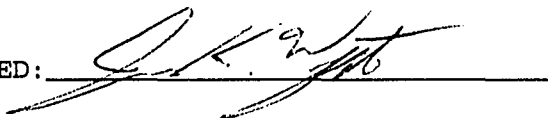
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A handwritten signature in cursive script, appearing to read "J. K. Wright", is written over a horizontal line. The signature is fluid and somewhat stylized, with a prominent initial "J" and a long, sweeping tail.

## ACKNOWLEDGMENTS

The present study could never have been completed without the technical brilliance and dedication of Mark Bakarich and Jonathan Forster. In addition to making an extremely complex protocol possible, they helped me clarify design elements that would not have been addressed otherwise.

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There are not words to express my appreciation of and debt to my long-time research assistant, Jennifer L. Anthony. Jen is among the most promising and dedicated students I have known. Throughout our three years together, I can only hope she learned equally from our triumphs and from my mistakes. She even put up with my bad singing during late nights of data analysis.

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The final section of this dedication goes to my father, David Michael Wyatt. He has provided a role model for me, a constant reminder that hard work and dedication can lead to success, no matter how many and varied may be the obstacles. He has also been behind me in every way, most importantly by unconditionally supporting my actions and decisions. If I work too much and care about my work too much, it is because I am my father's son — may we both learn from this.

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## ABSTRACT

This dissertation presents an examination of explicit and implicit memory for auditory stimuli presented immediately prior to sleep onset. The paper begins with a review and critical analysis of the research findings published in the areas of sleep and memory, event-related potentials and sleep, and event-related potentials and memory. In the present study, thirty undergraduate subjects (17 female and 13 male) were presented with auditory stimuli in an oddball paradigm (single-syllable concrete nouns and 50-msec 1000-Hz beeps in a 1:4 ratio) until sleep onset. They were allowed to accumulate either 30 seconds or 10 minutes of sleep, awakened, and tested for free recall and recognition memory for the meaningful stimuli. Results from event-related potentials recorded during the stimulus presentation phase supported the conclusion that subjects continued to process the meaningful stimuli until sleep onset. After 10 minutes of sleep, but not after 30 seconds of sleep, subjects had profound amnesia on free recall for stimuli presented in the four minute window prior to sleep onset. Increased beta EEG power during the sleep period correlated positively with recall of stimuli in the four minute window. In the 30 second condition (versus the 10 minute condition), subjects responded significantly faster in the recognition task to words correctly recognized. It is concluded that when allowed to sleep for 10 minutes, subjects evidenced a mixed anterograde and retrograde amnesia for auditory stimuli presented in the four minute window prior to sleep onset. The results are discussed in terms of stimulus encoding, memory consolidation, and information retrieval. It is hypothesized that during the sleep onset transition, explicit memory systems switch from processing new information, to becoming a dedicated system for reprocessing information presented during the presleep

period. Suggestions are given for further research, including studies of various sleep-disordered populations and the use of modified protocols.

### 3. INTRODUCTION

This introduction presents a summary and critical evaluation of the past research related to the present study of the psychophysiological correlates of amnesia for material presented immediately prior to sleep onset. The first section ("Sleep and Memory") covers observations of the interrelationship between sleep and memory. The next section ("ERPs and Memory") addresses the recording and interpretation of event-related potentials (ERPs), as well as studies applied to memory function. The next section ("ERPs and Sleep") covers event-related potential studies of sleeping subjects. The introduction concludes ("Conclusions and Suggestions for Further Research") with a synthesis of these areas and a transition to the present study.

From the review of sleep and memory in the first section of this introduction, one can conclude that there is a link between the processing of memory, perhaps especially memory consolidation, and sleep, especially REM sleep. It appears that further processing and consolidation of explicit memory are strongly associated with post-learning REM sleep. There is also evidence for a link in the other direction; chronic or acute sleep deprivation has been found to impair daytime cognitive performance. It is curious that despite this link between sleep and memory, the ability to transfer explicit or implicit memory for stimuli presented during sleep into long-term storage has not been shown to be possible. Perhaps the resolution to this seeming anomaly can be found in the research of memory processing during the transition between wakefulness and sleep. Several older studies and preliminary results from our laboratory have shown that the transfer of information from short-term memory to long-term memory is significantly impaired, if not eliminated, by the sleep onset transition.

It is clear from the literature covered in the second section of this introduction that ERP recordings are a reliable, unique memory assessment procedure. In designs where

target stimuli are task relevant, subjectively improbable, and presented in a way for subjects to perceive a high percentage of available information about them, the P300 component functions as an indicator of on-line cognitive processing, as well as a predictor of subsequent recall and recognition.

From the research reviewed in the third section, one finds that there is considerable evidence that the early components of the ERP waveform are present, unchanged, during at least the early stages of sleep. Studies of the later ERP components report that these components undergo changes in amplitude and latency during sleep, but most are still present.

Given these observations, it appears that event-related potential recordings could be a very sensitive tool for examining memory during the transition to sleep, as well as during sleep itself. The final section of this introduction makes explicit the links between the extant ERP findings and applications to the present study of memory and sleep.

### 3.1 Memory and Sleep

#### 3.1.1 Introduction

In intact humans, memory can fail for a variety of reasons. Complete failure to encode information can occur as a result of deep coma states or during certain surgical anesthetics. Encoding can also be shallow during situations where the subject is either hyper- or hypoaroused. The memory consolidation process can also be blocked, keeping information from being transferred from short-term memory to permanent or long-term memory. Additionally, various factors can interfere with the process by which information is retrieved from long-term memory.

The purpose of this section is to summarize and comment on the extant scientific literature on the interrelationship between sleep and memory. The first subsection details the research on memory for events presented during sleep -- otherwise known as "sleep learning". The second and primary subsection reviews memory functioning at the transition from wake to sleep. The final subsection covers suggestions for further research in the area of memory at the sleep onset transition.

#### 3.1.2 Memory for stimuli presented during sleep: Sleep learning

Sleep learning is the examination of whether or not individuals can process and store new information presented during sleep. However promising it has appeared in theoretical papers and in our imaginations, the ability of subjects to demonstrate, explicitly or implicitly, long-term retention of auditory stimuli presented during sleep has failed to be shown (e.g. Bootzin, Fleming, Perlis, Wyatt, & Schacter, 1991; Eich, 1990; Wood, Bootzin, Kihlstrom, & Schacter, 1992).

Methodological flaws in the studies are commonplace (reviewed in Wyatt & Bootzin, 1994). Much of the work from the former Soviet Union failed to use polysomnography (PSG) to monitor sleep. Thus, although these researchers found evidence of retention of material presented during sleep, it is likely that their subjects were aroused to wakefulness during stimulus presentation. Of the PSG studies to have shown positive findings in sleep learning, only the studies whose subjects showed signs of electroencephalographic (EEG) arousal showed later free recall and recognition of the material presented during sleep. Therefore, there is little evidence that material presented during uninterrupted sleep can subsequently be retrieved, perhaps due to a failure of consolidation processes by which information is stored in long-term memory. One could hypothesize that the consolidation of presleep information that occurs during sleep maximally utilizes certain underlying neuronal memory systems. Therefore, these systems are not available to consolidate new information presented during sleep.

### 3.1.3 Memory at the transition from wakefulness to sleep and from sleep to wakefulness

In light of the failure of demonstrate sleep learning in human subjects, it would be of theoretical and practical interest to locate the time period during which the failure of on-line consolidation occurs. As we know that subjects can easily transfer new information to long-term memory during wakefulness but not during sleep, it is likely that neurophysiologic and neuropharmacologic processes associated with the transition from wakefulness to sleep could account for this failure of consolidation. Similarly, the ability to consolidate new information could return during the transition from sleep to wakefulness.

One of the earliest comments about the interrelation between memory and sleep noted in a scientific publication came from Jenkins & Dallenbach (1924). In a study that eventually concluded that memory decayed less during sleep than in comparable periods of wakefulness, the authors noted a curious observation to the contrary.

After a few experiments *E* found it difficult to arouse the *O*s and difficult to know when they were awake. The *O*s would leave their beds, go into the next room, give their reproductions, and the next morning say that they remembered nothing of it. Unfortunately, it did not occur to *E* to apply special tests; he judged by the behavior of the *O*s, and assumed that they were awake (p. 607).

In the current language of sleep disorders medicine, the subjects described in the quotation were evidencing automatic behavior — seemingly normal behavior accompanied by later amnesia for one's actions. As this type of behavior is seen most often in individuals with disorders of extreme hypersomnolence like severe obstructive sleep apnea and narcolepsy (Diagnostic Classification Steering Committee, Thorpy, 1990), one must suspect a connection between sleep or perhaps sleepiness and memory impairment. One could speculate that subjects awakened during the night for memory testing, as was done in Jenkins & Dallenbach study, might be impaired not only by falling asleep during the testing, but by something called "sleep inertia."

Sleep inertia can be defined as a short-term, reversible impairment of cognitive abilities occurring after awakening from sleep. The duration and severity of sleep inertia have been shown to be related to the stage of sleep from which one is awakened and positively correlated with the amount of NREM Stage 4 sleep accumulation prior to the awakening (Dinges, Orne, & Orne, 1985). The effects of sleep inertia have been shown as far from awakening as 20 minutes or more (Stones, 1977; Tilley & Statham, 1989).

However, the effects of sleep inertia on memory performance immediately upon awakening have not been reported in the literature.

Guilleminault and Dement (1977) designed a pilot study to examine memory at the transition from wake to sleep. They presented two subjects with single words, one word per minute, until sleep onset, in an afternoon nap. They allowed the subjects to accumulate either 30 seconds or ten minutes of sleep, and then awakened them for memory testing. Guilleminault and Dement gave the subjects a test of recognition memory wherein the subjects were told not to guess. The results of the study showed a dramatic amnesia for words presented in the five minute window prior to sleep onset in the trials where the subjects had slept for ten minutes (see Figure 1 below). However, the subjects were sleep deprived to 2 hours of sleep on the night prior to the study, perhaps increasing the degree of memory impairment beyond that seen in normal subjects. Also, only recognition memory was tested and the small sample size prohibited the authors from running statistical analyses on the data. Thus, the generalizability of the results and the conclusions that could be drawn were greatly limited.

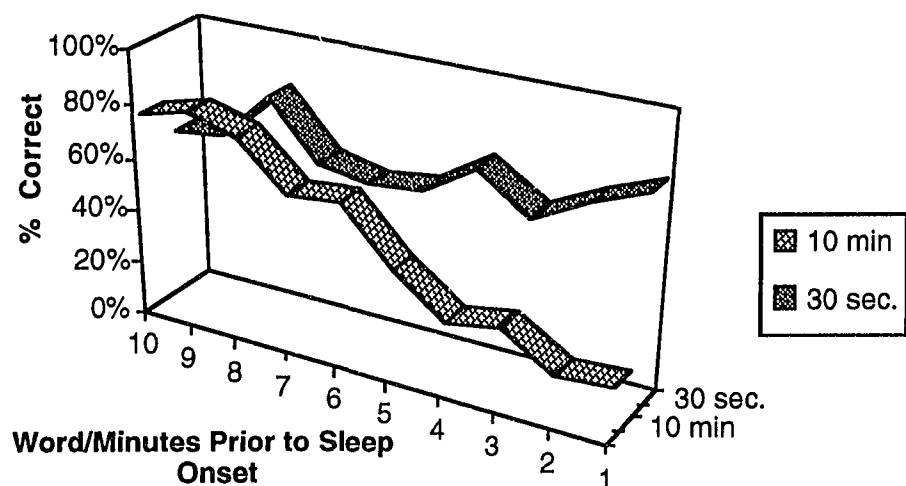


FIGURE 1. Guilleminault & Dement, 1977

Work by Goodenough, Portnoff, & colleagues (Portnoff, Baekeland, Goodenough, Karacan, & Shapiro, 1966; Goodenough, Sapan, Cohen, Portnoff, & Shapiro, 1971) further demonstrates the amnesic effects of sleep when it quickly follows a learning period. Their protocol involved awakening subjects from sleep, having them view stimulus material on a slide-projector screen, allowing them to return to sleep either immediately or after a brief delay, and testing their long-term memory in the morning. They observed in the original experiment and in a replication that, within the delayed-return-to-sleep condition, sleep latency correlated positively with morning memory performance — the faster the return to sleep, the worse the morning memory performance. However, in interpreting their results, the authors could not absolutely distinguish two theories for what may have caused their results. It could have been the case that sleep impaired consolidation of the stimulus material. If sleep came too quickly after the presentation, then the material would not be consolidated into long-term memory. Alternatively, on the trials where the subjects returned to sleep quickly, it is likely that they were much less aroused than in trials where the return to sleep was longer. Thus, the subjects' hypoarousal during presentation may have led to the poorer performance on the morning memory testing.

As a caveat, it should be mentioned that a similar study (Bonnet, 1983) failed to find such a correlation between delayed return to sleep and better memory performance. However, the subjects' sleep latencies post-stimulus presentation were all, on average and across conditions, four minutes or longer. Thus, the proximity between stimulus presentation and sleep onset was likely too great for the effect to be seen.

Our laboratory has conducted a two-phased study specifically examining the issue of memory for auditory stimuli presented immediately prior to sleep onset (Wyatt, Bootzin, Anthony, & Bazant, 1994). This study was an attempt to employ procedures

from the area of cognitive memory research, while maintaining the design put forth by Guilleminault & Dement (1977).

The experimental design involved two phases: sleeping subjects and waking subjects. For the purposes of simplicity, the following description will use the term "sleep" but will apply equally to the subjects who were asked to remain awake (deviations from comparability will be mentioned). During the protocol, subjects were asked to listen to pairs of words and to repeat each word pair aloud, after hearing it. They were instructed to keep repeating the individual word pairs each time they heard them, but also to let themselves fall asleep (awake subjects were told to remain awake). Following any questions, the experimenters turned out the lights (dimmed lights for the awake subjects), started the PSG recording, and started the audio presentation.

The computer program presented each subject with a single word and its most common associate, one pair per minute, until terminated at the onset of sleep (awake subjects were given 12 word pairs, the mean number heard per trial by the sleeping subjects). After sleep onset was determined by the presence of 15+ seconds of sleep EEG on the polygraph (or at the end of the twelfth word pair for the awake subjects), the experimenters allowed each subject to accumulate up to either 30 seconds or ten minutes (selected at random) of sleep (or wake), depending on the trial type. After an abrupt awakening with a 1000 Hz tone, the subject was immediately given a free recall task, matched explicit and implicit paired-associate tasks, and a recognition task. Tasks were derived from a study of explicit and implicit memory for events occurring during surgical anesthesia done by Kihlstrom & colleagues (Kihlstrom, Schacter, Cork, Hurt, & Behr, 1990). After memory testing, the subject was given another trial of the other sleep (or waking delay) condition.

As found by Guilleminault and Dement (1977), our team found a profound deficit of explicit memory for verbal material presented prior to sleep onset, on trials where

subjects accumulated ten minutes of sleep. The free recall data revealed a dramatic amnesia for the word pairs heard in the three minutes prior to sleep onset in the ten minute trials. In contrast, in the 30 second trials, sleeping subjects' recall rates were highest for those same three minutes prior to sleep onset -- perhaps demonstrating an intact recency effect (see Figure 2 below). On recognition testing, there was again a deficit in performance for the ten minute trials relative to the 30 second trials, but for only the single minute prior to sleep onset (see Figure 3 below).

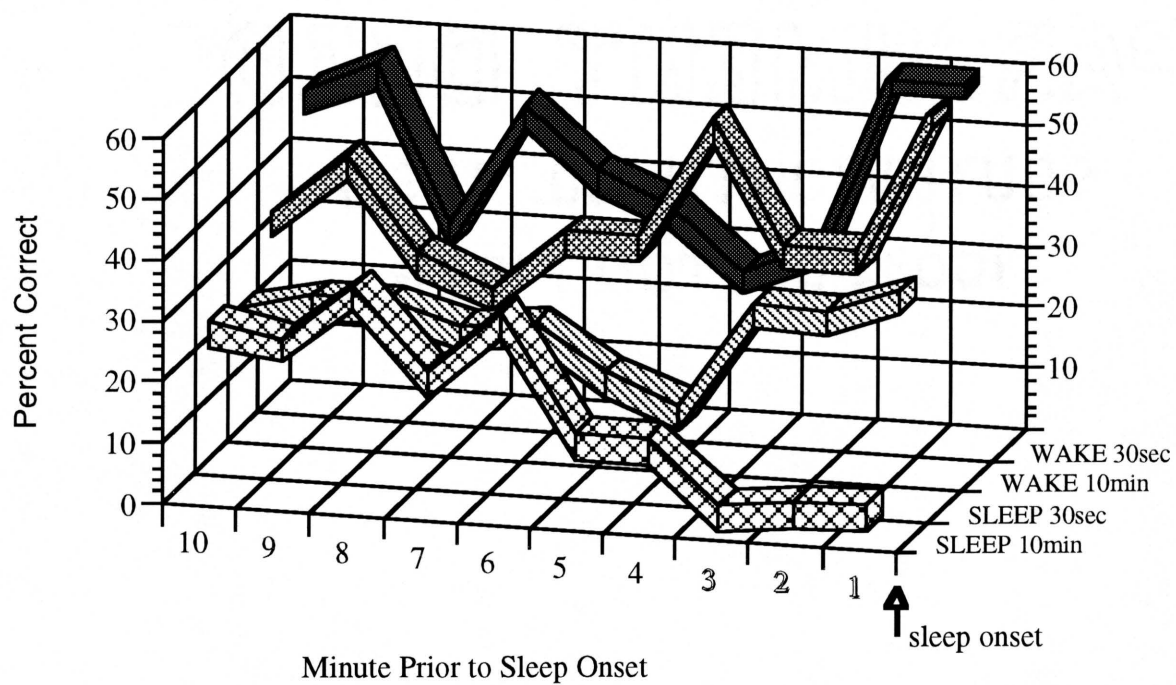


FIGURE 2. Free Recall Performance

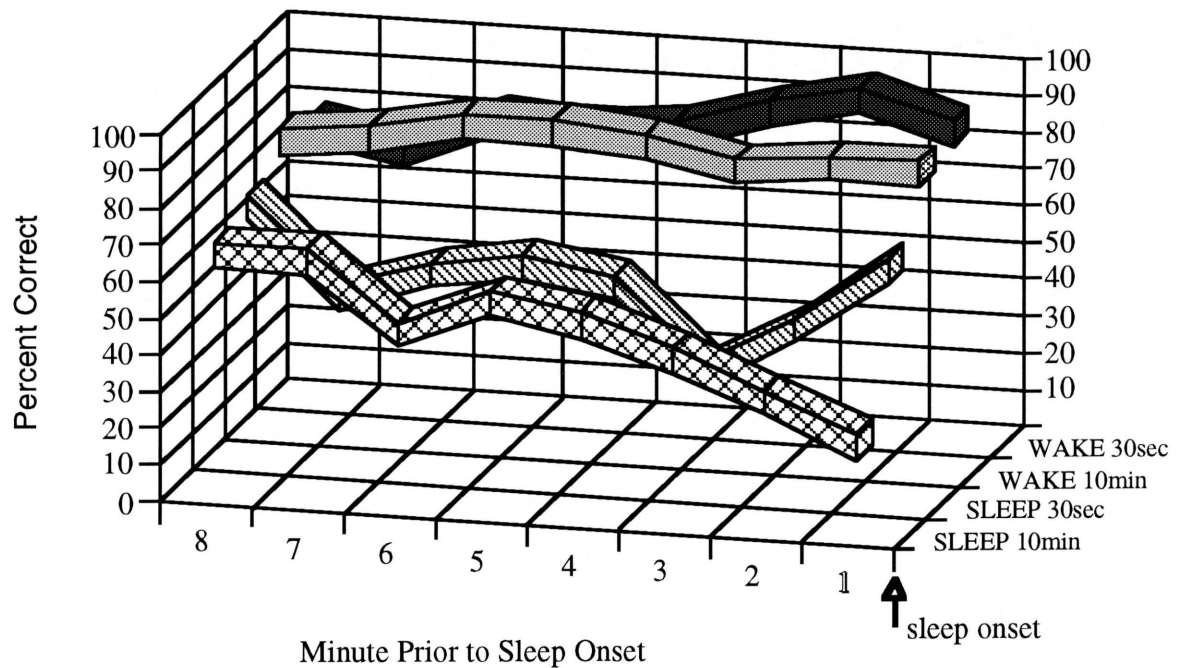


FIGURE 3. Recognition Performance

However, when these data were compared to the results from the awake subjects, an equally compelling effect emerged. Awake subjects performed significantly better overall, across the entire time series, on both the free recall and recognition tasks. Unfortunately, the results from the matched explicit and implicit paired-associate tasks were equivocal; there was clear evidence of priming in the implicit task but we did not observe an explicit/implicit dissociation. However, it was likely the case that explicit and implicit memory were confounded in the task, due to the experimental design and a ceiling effect.

Our interpretation of the results mentioned above raise two likely theoretical explanations. First, it seems quite clear that some depth and/or duration of sleep subsequent to sleep onset is associated with impaired retrieval of auditory information presented in the three minute window prior to sleep onset. As the subjects were able to reduce this window of impairment to one minute when given a recognition task, it seems

likely that the word pairs presented three and two minutes prior to sleep onset were indeed stored and were retrievable, but were apparently encoded poorly and/or processed so shallowly as to make free recall nearly impossible. The word pair presented one minute prior to sleep onset, however, was in more of a gray area -- it was hit or miss whether or not recognition testing would bring sufficient cueing to allow for correct response.

Our second interpretation of the data derives from a comparison of the sleeping and awake subjects. As the first interpretation posits causal action to processes occurring after the onset of sleep, the second interpretation focuses on what is occurring prior to sleep onset. As there were no significant differences between the 30 second and ten minute delay conditions for the waking subjects, it appears that the length of delay may not have played a role. And, as many sleep researchers would not consider 30 seconds of Stage 1 EEG official sleep onset (e.g., Johnson, 1973), it is not unreasonable to state that the primary neurophysiological difference between the 30 second sleep condition and the 30 second awake condition was the decline in arousal level that preceded sleep onset.

Therefore, if one considers the event of sleep onset as an arbitrary Time Zero, then the effects of the decline in arousal level prior to sleep onset support a conclusion that anterograde amnesia was observed. The observation that the depth and/or duration of sleep subsequent to sleep onset led to differences in the data support a conclusion that retrograde amnesia was also observed.

#### 3.1.4 Summary and criticisms

In summarizing the past research on memory during the sleep onset transition, several points need to be addressed. First, it would have been valuable to have had an on-line index of subject arousal level during the presleep period. It may be hypothesized,

though without objective evidence in support, that a decline in arousal level was related to the overall impairment of performance in the sleeping subjects. As will be described later, ERPs can provide just such a measure. Secondly, the test of implicit memory used in our study was most likely quite confounded by contributions from explicit memory. The matched explicit/implicit tasks probably worked well in the Kihlstrom et al. (1990) study because the subjects were unlikely to have had much explicit memory of the stimuli to confound the implicit task. This was obviously not the case with our study -- the subjects were awake and clearly able to register the stimuli, evidenced by the fact that not a single subject failed to repeat a word pair aloud during the presleep presentations. Thus, a different type (or types) of implicit measure would be a worthwhile improvement in further research. Finally, it would be useful to have an on-line measure of the amount of or type of processing each stimulus received. It could be hypothesized that the stimuli in the three word-minutes prior to sleep onset were gated at the level of the periphery. Alternatively, it could be the case that the stimuli made it into the central nervous system (CNS), only to receive little in the way of cognitive processing. Again, ERP technology can address the issue of sensory registration and cognitive processing of auditory stimuli. With these improvements, the present study takes a step toward explaining why memory is impaired for information presented immediately prior to sleep onset.

### 3.2 ERPs and Memory

This section begins with a subsection ("Definitions") on the definition of an event-related potential. The next subsection ("Late Components and Cognitive Processing") gives a brief overview of the most commonly-studied late ERP components and their cognitive correlates. Subsequent subsections ("The von Restorff Effect" and "Repetition Priming") review studies examining ERP correlates of memory processing.

The final subsection ("Summary") draws conclusions from the ERP and cognition literature and discusses applications to studies of memory at sleep onset.

### 3.2.1 Definitions

Perhaps the simplest definition of an event-related potential is that is the brain's response to a discrete event, either external or internal, as reflected in the electroencephalogram. An ERP can be associated with motor, sensory, or cognitive events (Hillyard & Hansen, 1986). A good description of the standard procedure for obtaining ERPs comes from a chapter by Coles, Gratton, and Fabiani (1990).

The most common procedure involves averaging samples of the EEG that are time locked to repeated occurrences of a particular event. The number of samples used in the average will depend on the signal-to-noise ratio. However, in all cases the samples are selected so as to bear a constant temporal relationship to an event. Since all those aspects of the EEG that are not time locked to the event are assumed to vary randomly from sample to sample, the averaging procedure should results in a reduction of these potentials leaving the event-related potentials visible (p. 413)

The EEG waveform remaining after this averaging procedure is a series of negative and positive potentials. Through the manipulation of subject and stimulus properties, one can infer relationships between the eliciting stimulus and a particular negative or positive peak, or component, of the waveform.

When reading the literature on human event-related potentials, one is easily overwhelmed by the number of components for which names have been proposed. Depending on the authors, components at the same latency from the stimulus and with similar properties may have different names. There are two common categorical

divisions into which one may divide the ERP components. The first is simply a latency grouping: early, middle, and late components. The other classification system groups stimuli not based on latency, but on their reactivity to stimulus manipulations.

Exogenous components are those who vary as a function of stimulus characteristics, such as intensity. In contrast, the endogenous components are those who vary more as a function of subject characteristics, such as attention and arousal state. There is obviously great similarity between the two classification systems, as well as much overlap across categories within each system. Attempts will be made to remain true to the original terminology in the review that follows. The components will be discussed in the context of both the stimulus characteristics used in the experiment and, where available, the subject characteristics. As this introduction is primarily concerned with sleep and memory, the following review will focus only on the late, more cognitive components of the ERP waveform.

Below is a table listing the various ERP components, as well as terms used roughly synonymously (adapted from Grass & Stockard, 1978, Hillyard & Picton, 1987).

TABLE 1. ERP Components

Auditory ERP Components			
Latency	Name	Synonyms/Variants	Correspondence*
Early	I	ABR	auditory nerve
	II	ABR	cochlear nuclei (medulla)
	III	ABR	superior olivary complex (pons)
	IV	ABR	lateral lemniscus (pons)
	V	ABR	inferior colliculus (midbrain)
	VI	ABR	medial geniculate (thalamus)
	VII	ABR	auditory radiations (thalamo-cortical)
Middle	No		unable to locate information
	Po		"
	Na		"
	Pb		"
	Nb		"
Late	Nd	negative-difference wave	attending in one channel
	P1	P100	attention
	N1	N100	attention
	P2	P200	
	N2	N2a, N2b, N200	stimulus evaluation; classification
	P3	P3a, P3b, P300, LPC, Dm, P3c	context updating in working memory
	N4	N4a, N4b, N4c, N400	detection of semantic mismatch
	CNV	contingent negative variation	preparation for motor or cognitive activity
	MMN	mismatch negativity	mismatch detection

\* Correspondence refers to the most commonly noted neuronal or cognitive correlate of the component, only for those where consensus exists in the literature

### 3.2.2 Late components and cognitive processing

There is considerable evidence that the N1 component is enhanced in selective attention tasks. In a variant of an auditory dichotic listening task, N1 and not P2 was significantly related to the attended-to ear (Hillyard, Hink, Schwent, & Picton, 1973).

In their review of negative components of the ERP waveform, Ritter and colleagues (Ritter et al., 1984) noted during a consensus conference that N2 is observed in response to infrequent items in a series, regardless of task relevance. They also noted that the amplitude of N2 varied inversely with the probability of the infrequent stimulus: the less frequent the stimulus, the higher the N2 amplitude. One of the members, Näätänen, observed that the N2 appeared to be related to the detection of a mismatch, hence, the term mismatch negativity. The same group stated that the N400 wave could be seen in response to a semantic incongruity during the presentation of stimuli. Members of the group disagreed as to whether or not the N2 and N400 components were different subsections of the same component.

In a more recent review of the late negative components, Pritchard, Shappell, and Brandt (1991) presented a thorough discussion of the past research and theory on the N200 and N400 components. They stated that, as above, N400 has been shown to be elicited in response to semantic incongruities. They raise the important caveat that the N400 does not come from merely a semantic incongruity, but rather when the subjective expectancy of congruity is broken.

With regard to the continued debate as to whether or not N200 and N400 are the same component or different ones, they provided a unique compromise position. They viewed them as separate functional components, but as both belonging to the same "supracomponent." Depending on the level at which one asks questions, and hence how one designs a given experiment, N200 and N400 can be observed to be dissociable or linked.

The authors further clarified the distinction between N4a, N4b, and N4c. Based on the designs and results of past research, they present N4a as an automatic, left, fronto-central component; N4b as an index of attention and violation of abstract priming, with a

more or less symmetrical, centro-parietal distribution; and N4c as an index of abstract classification, with less clear laterality and scalp distribution characteristics.

In a discussion of the P3 set of components, Sutton and Ruchkin (1984) listed seven criteria upon which to describe these waves: latency, amplitude, order of occurrence, distribution across the scalp (topography), relation to stimulus physical properties, relation to behavioral response, and relation to subject state and trait variables. In a panel discussion from the same volume, Donchin and colleagues (Donchin et al., 1984) raised several methodological concerns common to P3 or P300 research. They noted that stimuli are usually task relevant, that the interstimulus interval is usually less than five seconds, and that there are usually between ten to fifteen stimuli averaged to obtain a reliable P3/P300.

In a unique variant of an oddball paradigm, Simson, Vaughan, and Ritter (1976) examined negative and positive potentials in response to missing stimuli within a train of regularly-occurring stimuli. They noted the presence of both negative and positive slow potentials following the missing auditory stimuli, with latencies of 230 msec and 465 msec, respectively. Although not explicitly stated in the report, it could be hypothesized based on the latency and scalp topography that the components might have been N2 and P300. Regardless, the results given in the report were insufficient to permit clear interpretations and comparisons to other data.

The literature is replete with different names for similar late, positive components of the ERP. Donchin and Coles clarified that the classic P300 was also referred to as P3, P3b, and the LPC, or late positive component (Donchin and Coles, 1988). They further noted that the probability of eliciting a P300 and its amplitude increased as the probability of occurrence of a stimulus decreases. Furthermore, they noted that in several studies, the subjective probability of a stimulus occurring influenced the probability of observing a P300. Finally, they noted that the relevance of the stimulus to the task (ie.,

instructing the subjects to attend to or ignore a class of stimuli) correlated positively with the probability of observing a P300 and P300 amplitude.

At the heart of the article, Donchin and Coles outlined their model of P300: that it is a representation or an index of context updating. They differentiated two types of information processing: tactical and strategic. They defined "tactical" processing as that which occurred as a part of a stimulus – cognitive mediation – behavioral response chain. In contrast, they used "strategic" processing as that which operates at a more general level, including attention allocation, longer-term planning and control of behaviors, and evaluating stimulus input – all necessary for updating internal representations the context of one's environment. They cited evidence that P300 can occur after an immediate behavioral response to show that P300 is not a part of the tactical processing system.

In an earlier paper (Donchin, 1981), Donchin summarized past research on the P300. He noted that the latency to P300 appeared to be a good indicator of the amount of time required to categorize stimuli. He also wrote that the amplitude of P300 may, among other things, be useful as an index of perceptual workload. He was careful to distinguish perceptual workload from response demands, from which P300 amplitude seems to be independent.

In a more recent publication, Fabiani, Karis, and Donchin (1990) invoked the term "working memory" as the functional location of the context updating mentioned above. They further stated that this updating of information in working memory could add cues or markers to the information in working memory, thus serving as retrieval aids for subsequent recall and recognition.

Presented in the same journal issue as the above paper, Verleger (1988) presented an alternative perspective on the P300 component. Verleger stated that P300 reflected "context closure", which occurs when subjects perceive an expected event during a monotonous, structured task.

It is problematic that immediately after he presented a list of studies supporting Donchin's updating hypothesis and only a few that directly refute it, Verleger wrote that there was "scant" empirical support of the hypothesis. While he agreed that the P300 was an index of strategic processing, he disagreed that the theory of P300 being related to subjective expectancies could coexist with the updating hypothesis. Again, Verleger's reading of the literature, while being thorough, simply appeared to be a different interpretation of existing data. Finally, in presenting evidence for his context closure hypothesis, Verleger seemed to be finding data to support his theory, rather than viewing the data and abstracting a theory. While it is not the intention to argue against a priori theories, it is possible to entertain the idea that the P300 could simply be an index of numerous processing operations arising from the activity of various, interacting brain regions. Depending on the type of experimental design, different cognitive demands could be made of the subjects that could elicit an apparently similar bioelectrical signal, as measured at the level of the scalp. Thus, simply because the context updating hypothesis cannot explain all published reports does not necessitate its refutation.

By way of metaphor, if one observed a person lying still, supine, with closed eyes, it could be stated that the person was asleep. If the observer learned that the subject was in fact under surgical anesthesia, comatose, or dead, it does not violate the observation that such an appearance could under other conditions be a consequence or correlate of being asleep.

Johnson (1985) presented an alternative hypothesis regarding the functional significance of the P300 component's amplitude. In his triarchic model, his first two elements can be found in the writings of Donchin: subjective probability and stimulus meaning (or task relevance). Johnson's unique addition is the element he called information transmission. He defined information transmission as the percentage of potential information of a given stimulus that a subject actually perceives. In his model,

both attention to the stimulus and degree of equivocation (how certain the subject is that the information perceived matches the original stimulus) independently contribute to information transmission. In Johnson's triarchic model, the amplitude of the P300 is determined by the additive effects of subjective probability and stimulus meaning, both of which are modulated multiplicatively by information transmission.

It appears that nothing in Johnson's model directly refutes Donchin's context updating model. In fact, Johnson's information transmission factor fits in well with Donchin's model, as a necessary prerequisite for efficient context updating. Thus, by viewing the context updating model as a superstructure, the triarchic model of the P300 amplitude can be subsumed without revision.

In conclusion, one can divide the ERP components into early, middle, and late components. The late components have been shown to be robust indicators of cognitive processing, a brain-behavior relationship. However, there is by no means a strict one-to-one mapping of a particular cognitive functions to a single ERP component, rather it appears that there are stimulus- and design- and subject-dependent variables that interact with the physiological systems that underlie the human evoked potential.

### 3.2.3 The von Restorff effect

Studies comparing ERP waveforms generated during learning periods and subsequent memory performance have uncovered interesting connections between ERPs and memory. Donchin & colleagues (Fabiani, Karis, & Donchin, 1986; Karis, Fabiani, & Donchin, 1984) have found that the amplitude of the P300 component recorded during initial stimulus presentation was larger for words later recalled than for words not recallable. However, the predictive value of the amplitude of the P300 component only held for subjects who were engaging in simple, rote memory processes; the effect was

absent in subjects who were employing mnemonic strategies during the initial presentation. In the introduction to the 1984 study, the authors provided a concise overview of Donchin's theory of what the P300 means:

The label von Restorff, or isolation, effect refers to the enhanced learning of an "isolated" item... Since isolated items are both novel and task-relevant there is a striking similarity between their attributes and the attributes of stimuli that elicit the P300 component of the human event-related potential (ERP). In ERP experiments novel, task-relevant, events elicit a positive potential with a latency to the peak of at least 300 msec following the eliciting stimulus. This component of the ERP, commonly called P300, is a manifestation at the scalp of intracranial activity involved in cognitive processing... The data currently available on the conditions under which P300 is elicited suggest that P300 reflects processes invoked when there is a need for "context updating"; that is, when there is a need to revise the current representations in working memory (p. 178)

Karis, Fabiani, and Donchin (1984) employed the von Restorff paradigm in a protocol involving ERP recordings, response times, and free recall and recognition memory tasks. They presented twelve subjects (age range = 18-21; all females) with three different sizes of visual text (small, medium, and large) on a computer screen. The stimuli were presented for 200 msec., with an interstimulus interval of 2 sec. Results of the free recall task showed that subjects who evidenced a higher von Restorff effect (defined as recalling more isolated words) recalled a lower percentage of words overall. For the recognition test, the subjects responded significantly faster to the isolated words than to those not isolated. Subjects' post-test descriptions of their memorization strategies showed that those with the highest von Restorff effect relied more on rote

memorization, while those with the weakest von Restorff effect utilized complex mnemonic strategies.

The ERP results indicated two primary findings. First, for all subjects, only the isolated words elicited large P300 components. Second, for only those subjects who showed a large von Restorff effect, the amplitude of P300 to the isolated words could predict subsequent recall: larger P300s were associated with better recall. This effect was largest in Pz, versus Cz and Fz. In contrast to this P300 effect, ERPs from the subjects who showed the lowest von Restorff effect contained a slow, positive, frontally-maximal component that was of greater amplitude for the isolated words, and for the recalled isolated words.

The ERP data collected during the recognition test were congruent with the results from the free recall data. Regardless of whether the words were isolated or not isolated at presentation, when repeated during recognition testing, they elicited larger P300s than new, unrepeated words. Also, correctly-recognized repeated words were associated with larger amplitude P300s than those not recognized.

The authors concluded that the isolated words, as they had hypothesized, produced larger P300s than the words not isolated because they were novel and task relevant. As noted above, in oddball paradigms, it is exactly the task relevant and novel stimuli that produce P300s of higher amplitude.

In an effort to further clarify the interrelationship between the context updating/P300 hypothesis and the learning or encoding strategy, Fabiani, Karis, and Donchin (1990) conducted a within subjects manipulation of encoding strategy; subjects were run with separate instruction sets on separate trials to use rote memory or elaborative encoding strategies. For the rote instruction set, subjects were asked to repeat the words they viewed to themselves. In contrast, the elaborative encoding instructions required the subjects to form more complex semantic formations of the stimulus word,

and/or to form visual images of the words. Results from the ten subjects (age range = 18–21; all female) indicated that, as in their prior research, subjects recalled a significantly higher percentage of isolated words minus nonisolated words under the rote instructions, but a significantly higher percentage of words overall in the elaborative encoding instructions. In the ERP results, there were significantly higher-amplitude P300s elicited to isolated words, versus nonisolated words, across the two instruction sets. There was also a significant interaction between instruction set and recalled/nonrecalled items, such that there was only a significant difference between P300 amplitude between recalled and nonrecalled words within the rote condition.

As in their earlier work, the authors noted the presence of a slow, positive component (though without a clear peak morphology) within the 800 – 1180 msec window. This positive slow wave was significantly larger within the elaborative encoding condition and in association with words later recalled, across conditions.

In a similar study by another group (Paller, McCarthy, and Wood, 1988) subjects were required to complete semantic decision tasks (edible or inedible, and interesting or uninteresting) on words presented visually. The authors also purposely avoided using the term P300 to describe the interval(s) of the EEG waveform where they observed an amplitude difference for words later recalled or not recalled.

To avoid prejudging the relationship between this ERP difference and ERP components such as P300, Paller et al. (1987) introduced the term *Dm* to refer to ERP Differences based on subsequent memory performance (p. 270).

Thus, Paller et al. (1988) focused on the time spans during which the ERP waveforms differed in accordance with later retrieval performance. They binned the EEG data into 100 msec intervals and found that for predicting free recall performance, *Dm* was apparent for the 400-500 msec, 500-600 msec, and 600-700 msec poststimulus intervals.

They found near-significant results for the 600-800 msec interval prediction of recognition performance, but their design was handicapped by low statistical power, so the failure here may not be a problem.

#### 3.2.4 Repetition priming

In a study of the developmental nature of visual versus verbal processing, Berman, Friedman, and Cramer (1990) used a repetition priming paradigm to test children, adolescents, and adults. The report began by citing evidence that explicit memory is typically found to be better for pictures than for words. Thus, their study proposed to examine the ERP correlates of picture and word processing and recognition, hypothesizing that electrophysiological evidence would be found that would suggest differential processing. In the ERP data, they found an anterior negativity at around 350 msec that was larger for picture tasks than word tasks, but this difference was only significant for the children. Across the age groups, the amplitude of this negativity was greater for unrepeated items, while P3b amplitude was greater for repeated items.

Bentin & Moscovitch (1990) presented a series of studies examining the effects of repetitions of visual stimuli on explicit and implicit memory for those stimuli. As stated in the article, the studies had two purposes:

Our first purpose in the studies we report, then, was to demonstrate that electrophysiological responses to repeated events can also serve as an implicit test of memory. Having shown that, our second purpose was to study the relationship between electrophysiological measures and more traditional behavioral measures of performance on memory tests (p. 346).

The electrophysiological measures consisted of ERP recordings. In particular, the experimenters were interested in analyzing the differences between the P300 latencies

and amplitudes elicited by words that were novel, repeated once, or repeated twice. The behavioral measures were the subjects' response times and the correctness of response to visual stimuli. For all behavioral responses, subjects were asked to respond as quickly and accurately as possible.

In these experiments, the subjects went through three phases: study, explicit testing, and implicit testing. During the first or study phase, the subjects were asked to press either the button in their right hand or their left hand, the hand dependent on certain stimulus properties. One group of subjects performed a semantic decision task (press the button in your right hand if the word you see is an animate object; press the button in your left hand if the word you see is an inanimate object;  $N = 16$ ); the other group performed a lexical decision task (press the right button if what you see on the screen is a word; press the button in your left hand if what you see is not a word;  $N = 16$ ). The subjects viewed 64 words in the study phase of the semantic task. In the lexical decision group, there were 96 stimuli, half of which were words. Each group was allowed to rest for five minutes between this task and their explicit task.

The explicit task required subjects in both groups to decide whether or not they had viewed the stimulus words before (in the study phase). The subjects had to press one button if they recognized a given stimulus as a word from the presentation phase and the other button if the word appeared to be new. As an explicit task, the subjects in the semantic decision group viewed all 64 of the words from the study phase plus another 128 new words (called "lures" in the article). For the lexical decision group's explicit task, the subjects viewed the 48 words from the study phase plus 96 lures. It is not mentioned in the article whether the same 48 word / 48 nonword ratio applied to these 96 lures. Another five minute rest period followed the explicit task.

The implicit task required that the subjects perform a categorical decision task (semantic or lexical) that matched the study phase. For the implicit task, the subjects in

the semantic decision task viewed the 64 words from the study phase, plus half (48) of the lures from the explicit (recognition) test, plus another 48 new lures. Finally, the implicit task for the lexical decision subjects included the 48 words from the study phase, plus 24 words from the explicit (recognition) task, plus 24 new words. The stimuli also included the 48 nonwords from the study phase, plus 48 new nonword lures. (see the Figures 4 and 5 below for a graphic depiction of the information given above).

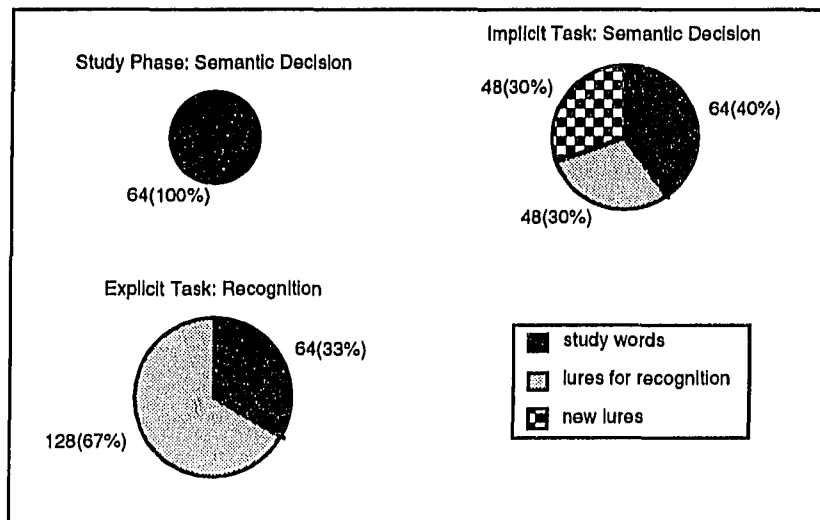


FIGURE 4. Semantic Decision Task

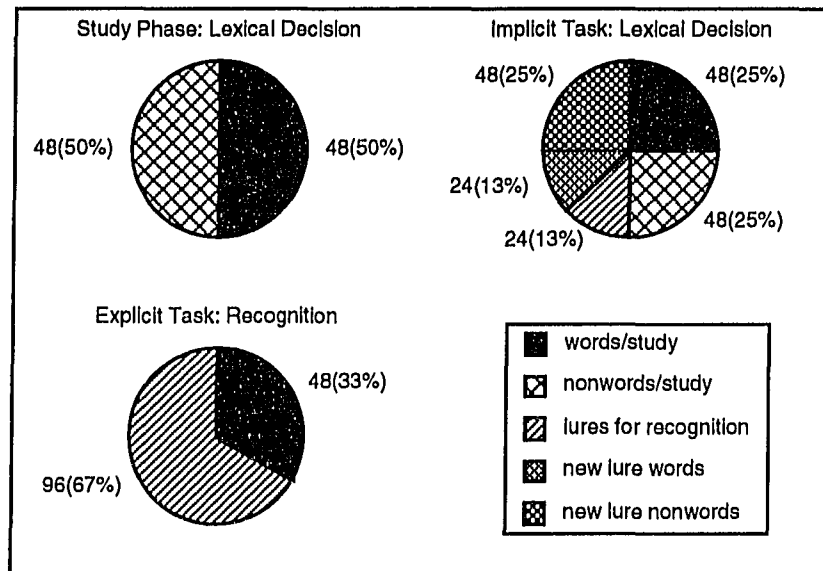


FIGURE 5. Lexical Decision Task

In the results section, Bentin & Moscovitch distinguished among four types or categories of behavioral responses for the explicit tasks: hits, correct rejections, misses, and false alarms. For both groups, responses that were hits and correct rejections were faster than misses and false alarms. However, the authors found no significant differences between responses to repeated stimuli (words that were from the study phase) versus words that were lures. The analysis of the reaction times for both groups' implicit tasks was much more complex. In contrast to what was found in the explicit task, words that were repeated were associated with significantly faster response times in both groups.

For the ERP waveforms generated during the explicit task in both groups, the waveforms for the repeated words (hits and misses) were collapsed, as were those from the new words (correct rejections and false alarms). The authors noted significantly higher mean amplitudes of the P300 components in both subject groups for the repeated words ("mean amplitude" corresponding to the average value of the ERP waveform

during the 64 milliseconds prior to and the 64 milliseconds following the P300 peak). They also noted a significant main effect of electrode site: the mean ERP amplitude was greatest in sites Pz and Cz, as one would expect for P300 components. The authors did not report statistical analyses looking at the ERP waveforms associated with correct and incorrect responses, citing an insufficient number of incorrect responses. However, they noted a trend in the direction of shorter P300 latencies for correct responses.

For the implicit tasks, the mean P300 amplitudes (during testing) for stimuli repeated twice (once during the study phase and again in the explicit task) were higher than those repeated once. Similarly, the mean amplitudes of the P300s elicited by words repeated once were higher than those elicited by novel lures.

In summarizing the results from the two groups, Bentin & Moscovitch stated that the ERP analyses were much more sensitive to the degree of repetition than were the behavioral measures (with both ERP and behavioral measures collected during recognition testing). The authors also noted the need to run an additional study, with the purpose of examining the relationship between the  $d'$  statistic and the amplitude of the P300 component.

In that study's explicit task, the amplitude of P300 was observed to covary with  $d'$  and also varied with repetition. This repetition effect was observed whether or not the subject made the correct behavioral response. In the implicit task, the ERP measures were more sensitive to the repetition effects and to the time-since-presentation of the stimuli than were the behavioral measures. In interpreting these results, the authors put forth that their results would not be inconsistent with the conclusion that P300 amplitude was an index of the "presumed strength of the memory trace" (p. 351).

In two follow-up experiments, Bentin, Moscovitch, and Heth (1992) further examined ERP indicators of explicit and implicit memory. The stated purpose of the study was to examine the interrelationship between ERPs and repetition, in designs where

the effect of conscious recollection could be separated. Subjects were run in protocols involving recognition testing of repeated and nonrepeated (new) words, with separate trials of a lexical decision task or a semantic decision task.

In analyzing the data from the first experiment, they found that the P300 component of the ERP was of significantly larger amplitude for repeated than nonrepeated words within the lexical decision task and nearly significant in the semantic decision task. There was not a significant difference in the amplitudes of the P300s to the correct and incorrect responses. However, behavioral response time was significantly shorter to correct than incorrect responses, but not significantly related to repetition effects. In contrast to P300 amplitude, the latency to P300 was significantly shorter for correct than incorrect responses (respectively, 598 msec [both tasks] versus 657 msec [semantic] and 653 msec [lexical]). Similar amplitude effects were observed for implicit memory category decision tasks.

The purpose of the second experiment was to make the recognition test more difficult, by increasing both the number of stimulus items and the delay between presentation and recognition testing (the interval filled with a distraction task). Also, a slight variation in the protocol allowed them to assess the effect of varying the number of repetitions. The ERP measures, but not the behavioral reaction times, were related to the repetition manipulation and to the delay.

In contrast to the results from the Fabiani et al. and Bentin et al. studies, Johnson, Pfefferbaum, and Kopell (1985) found a similarity between response times and P300 latencies and amplitudes in a repetition priming paradigm. At test, repeated items recognized elicited P300s of significantly greater amplitude and shorter latency, as well as faster reaction times. In contrast, at presentation, latency and not amplitude was a significant predictor of later recognition performance. There was a trend, however, for

the amplitude of P300 to predict recognition performance, and the low statistical power from only twelve subjects may have handicapped this finding.

In a rather difficult to interpret priming experiment, Rugg (1985) found that both reaction time and amplitude of N400 were decreased for both semantic primes and repetition primes. This N400 effect was greater for the semantic primes. He also found a significant increase in P300 amplitude for only the repetition primes. In addition to the difficulty in interpreting the results section of this report, it does not appear that the author attempted latency adjustment procedures, to account for variability in the N400 and P300 latencies.

In an extremely thorough report, Paller and Kutas (1992) were able to dissociate conscious recollection and priming. They had subjects perform both an imagery study task (thought to involve more elaborative processing) and a letter study task (thought to involve much less processing of the meaning of the word stimuli). Recollection was greater on both the free recall and recognition tasks for stimuli in the imagery task versus the letter task, as hypothesized. Evidence for priming, however, did not vary across the tasks. The ERP results showed a dramatic difference in the amplitude of a positive component in the 500–800 msec window (perhaps P300), the amplitude being greater in the imagery task. This was interpreted as an indicator of the differential reliance on recollection between the two tasks.

In a unique application of an event related potentials, Allen, Iacono, and Danielson (1992) examined ERP and implicit behavioral responses in a repetition priming paradigm where subjects were explicitly told to attempt to conceal the fact that they had memory of a previously-learned list of items. The subjects were informed that brainwaves could give away the fact that they had seen these words before, and that they should try to keep this from happening. Although a combination of the two implicit behavioral measures (total incorrect responses and mean response time) slightly outperformed the ERP

measures in detecting the deception, the combination of the ERP measures and the implicit behavioral measure gave 100% classification accuracy.

### 3.2.5 Summary

In conclusion, there is considerable evidence that ERP measures can provide both divergent and convergent information about memory functioning in human subjects. Several studies have shown that with von Restorff paradigms or repetition priming experiments, late ERP components can 1) provide an index of on-line memory processing, and, 2) predict subsequent memory for the stimulus material, both within subjects and within groups.

If Donchin & colleagues are correct about P300 being a reflection or index of information updating in working memory, then P300 could be very helpful in studies of memory functioning during the transition from wakefulness to sleep, verifying that subjects were continuing to process the auditory stimuli into working memory during the presentation phase. Also, if there is a decline in the activity or the effectiveness of this processing as sleep onset nears, then ERP recordings should be able to measure the effect. One could also use an oddball paradigm to see if von Restorff or Dm effects hold for information presented immediately prior to sleep onset. It could be hypothesized that for words presented in the three minute window prior to sleep onset, only stimuli eliciting the largest P300s would be later recalled or recognized. ERPs could also be recorded during the postsleep recognition memory tasks, to look for repetition priming effects. Perhaps repeated words that were not correctly recognized would elicit a larger P300 than nonrepeated words, serving as evidence of implicit memory.

### 3.3 ERPs and Sleep

The following subsections ("Early Components", "Middle Components", and Late Components") will review studies of the early, middle, and late components during sleep. Again, due to the primary aim of this introduction, the following review will focus mainly on the late components during sleep. Following these subsections is a discussion and critique of these studies ("General Summary and Criticisms").

#### 3.3.1 Early components

The literature on the early components presents a mixed picture regarding whether or not these components change as a function of being awake or asleep. Campbell and Bartoli (1986) appropriately cited flawed stimulus delivery procedures, circadian fluctuations, and insufficient sample sizes as potential confounds to these studies.

In their 1986 study of the early ERP components during sleep, Campbell and Bartoli tested nine subjects (age range = 18–25; all female) for two consecutive nights' sleep. The early components were recorded on only night one. Noteworthy was that the auditory stimuli were delivered through a hearing aid-like device, to ensure the subjects were able to perceive the stimuli. Also, the experimenters varied both the stimulus rate (standard condition = 11 clicks/sec., versus other conditions of 41 clicks/sec. and 81 clicks/sec.) and the intensity of the stimuli (standard condition = 70 dB nHL, versus other conditions of 50, 30, and 10 dB nHL) during the presleep baseline and sleep testing.

There were significant main effects of stimulus rate across all early components latencies except peak I. There were mixed results for amplitude of the early components, with significant main effects of stimulus rate, where increased stimulus rates were

typically associated with decreased amplitudes. They did not find significant main effects of sleep stages or significant interactions of sleep stages with presentation rate for any of the early components.

The authors found significant main effects of stimulus intensity on both the latency and amplitude of the early components; the higher the stimulus intensity, the shorter the latency and the larger the amplitude. However, there were no significant main effects of sleep stage or significant interactions of sleep stage with stimulus intensity.

It is interesting to note that the authors did not find significant contributions of either circadian fluctuation, as indexes by tympanic temperature recordings or time-of-night. A potential problem in this study was the use of mastoid references for the EEG measurements from Cz and Oz. This reference placement could allow for the intrusion of muscle and EKG artifact, which could have been minimized using linked earlobe references.

In an improved study design, Deacon-Elliott, Bell, and Campbell (1987) tested the wake and sleep brain auditory evoked potentials in eight subjects (age range = 22–34; two males, six females) during a night's sleep in the laboratory. The major improvement in this design was the direct comparison of awake versus sleep early auditory evoked potentials at the same stimulus intensity. The results indicated that the amplitude and latency of wave V were exogenous (responsive to changes in stimulus properties) both when the subjects were awake and when they were asleep. Furthermore, there were not significant differences between the wave V amplitude or latency between the awake data and any of the sleep data (early Stage 2, late Stage 2, or REM), nor were there any differences in amplitude or latency between the sleep stages. Similarly, there were not significant differences between the intensity thresholds for the evoked responses between the awake and sleep data.

The studies reviewed above give evidence that the early auditory ERP components do not appear to be differentially affected by wakefulness and sleep. These early components maintain their exogenous characteristics during sleep, changing with stimulus intensity and rate.

### 3.3.2 Middle components

In a study of 14 subjects (age range = 22–55; 7 male, 7 female), Erwin and Buchwald (1986) found mixed results. Analyses of amplitude measures revealed no significant changes in ABR V, Pa, or Nb. Only P1 showed a statistically significant change with arousal level, decreasing in amplitude from awake to NREM Stages 2-4 and increasing during REM. Latency analysis of ABR V and Pa found that only the latency to Pa increased significantly from awake to NREM Stages 2-4. This study was particularly valuable, in that EEG recordings were made with appropriate filter settings (10-300 Hz bandpass) to be sensitive to and selective for the middle latency components. A potential problem was that the authors combined awake and NREM Stage 1 in their analyses, states which may not be cognitively or physiologically comparable.

### 3.3.3 Late components

In one of the earliest reports of evoked potential recordings during sleep, Williams, Tepas, and Morlock (1962) studied three subjects (age range unknown; all males) during a night's sleep in the laboratory. The experimenters used a signal generator to deliver 85 dB clicks to the subjects, with an interstimulus interval of two seconds. Computer averages were calculated for every one hundred stimuli presented, with EEG recordings from Cz. Data were reported from wakefulness, Stage 1, Stage 2, Stage 3,

Stage 4, and Stage 1rem (now referred to as REM). There was clear evidence of the presence of P1, N1, P2, and N2 in all NREM stages, though sleep did appear to affect the amplitudes and latencies of the components. The amplitude of P1 increased with descending sleep stages, while the amplitudes of N1 and P2 decreased. Though the reporting of the statistical analyses of these changes was scant, it was clear that all changes were significant. The amplitudes of these components during REM was roughly comparable to Stage 1 NREM, but it is difficult to interpret from the article if statistical comparisons were performed on the REM data.

It is interesting to note that the authors observed a late, positive component during their recordings, becoming more prominent with descending NREM sleep stage. The figure of the component given in the text resembled the P3 component and had a latency of approximately 600-1000 msec.

Ujjaszi and Halasz (1986) reported a study of six subjects (age range = 20–30; 3 males, 3 females). In their study, they presented 1200 Hz tone-pips, of a 70 msec duration, to subjects across two consecutive nights in the sleep laboratory. Data were presented only for recordings made during NREM Stage 2 sleep. In presenting their data, they separated the late components based on microvolt amplitude ( $< 81 \mu\text{V}$ ,  $81\text{-}200 \mu\text{V}$ , and  $> 200 \mu\text{V}$ ) and on morphology (N300-P400, N300-P800, and N600-P800). They considered the N300 component to be the traditional N2, the P800 to be P3, the N600 to be N2b, and the P400 to be P3a. The amplitude/morphology pattern observed most often (21% of observations) was the  $81\text{-}200 \mu\text{V}$  N300-P800 (N2-P3) pattern. The results of this study must be viewed as purely descriptive, as no statistical analyses were reported. Also, as they did not employ an oddball design, the data do not address level of cognitive processing usually thought to be associated with P300. Finally, these authors reported amplitudes that far exceed those reported in the ERP literature, casting doubt on their assertion that they were observing the N2-P3 complex.

In the communication from a 1986 conference, Campbell, McGarry, and Bell (1988) reported data from seven subjects they studied during a single night in the sleep laboratory. In the presleep awake testing, there were significant increases in N1 and P2 amplitude with increases in stimulus intensity. N1 amplitude was significantly decreased from awake levels in early night Stage 2, SWS, REM, and late night Stage 2. There was a nonsignificant recovery in N1 amplitude during REM. In contrast, P2 amplitude was larger in both early Stage 2 and SWS than during awake or REM. The results, with the observation that processing negativities are larger in NREM than REM, were said to support a conclusion that information processing capacity is greater in REM than NREM sleep.

It is likely the case that insufficient statistical power kept the recovery in N1 amplitude during REM from reaching significance. Utilizing a sample size much larger than the seven subjects used in the above study could have improved the design.

In a study focusing primarily on the P300 component, Wesensten and Badia (1988) recorded ten subjects (age range = 18–29; three male, seven female) across two nonconsecutive nights in the laboratory. The study employed two auditory oddball designs, with target/nontarget probabilities of 30/70 and 50/50. The target and nontarget stimuli were, respectively, 1400 Hz and 1000 Hz tones, both of 50 msec. duration and presented through earphones.

The authors of this study reported that the P300 amplitude was significantly higher during sleep to target tones than to either nontarget tones or nontones (control recording periods during which no stimuli were presented). The authors stated that this was evidence of intact differential processing of task-relevant and task-irrelevant stimuli during sleep. However, they failed to find a significant main effect of sleep stage on P300 amplitude. Also, there was a significant main effect of state, where P300

amplitudes were larger during wakefulness than when subjects were asleep. In contrast, N200 amplitude was significantly larger during sleep versus awake.

Latency to P300 also differed as a function of arousal state. There was a significant main effect of sleep/wake state, with latencies shorter during wakefulness than sleep. Mean P300 latency during awake was 302.72 msec., versus 762.49 msec. during sleep. Latency to N200 was similarly significantly longer during sleep than awake.

Problems with this study are numerous. First, the authors reported having scored the sleep according to Rechtschaffen and Kales (1968) criteria, yet did not have the required C3 or C4 electrode placement to do so. Second, in light of the fact that the study focused almost entirely around the P300 component, the authors failed to record from Fz and Pz, which would have enabled them to examine commonly-observed differences in P300 amplitude and morphology (Donchin, 1978). Additionally, to compare and contrast their results with those of waking P300 studies, they should have recorded from Fz, Cz, and Pz, to verify that the purported sleep-P300 had the same scalp distribution as is found in awake subjects. Furthermore, the subjects were required to both count and behaviorally respond to the odd auditory stimuli. Thus, the generalizability of these results are limited to statements concerning P300 in nonstandard sleeping conditions. Finally, all of the statistical analyses should be viewed as preliminary, in light of the small sample size and, hence, limited statistical power.

In a study reporting 10 subjects (age range = 18–37; eight males, two females), Nielsen-Bohlman, Knight, Woods, and Woodward (1991) examined the amplitudes and latencies of the late ERP components in an oddball design, comparing recordings made during awake, Stage 2, and Stages 3 and 4 (SWS). For the Stage 2 data from the frequently-presented stimuli, they found that central P1 latency increased from awake to Stage 2, while amplitude was unchanged. N1b amplitude decreased from awake to Stage 2, but latency remained stable. Similarly, the amplitudes of temporal N1a and N1c were

significantly decreased in Stage 2. For the late components, there was a significant increase in central P2 amplitude, with latency unchanged. For the frequent stimuli in the oddball paradigm, there was a significant increase in central N340 amplitude during Stage 2 sleep versus awake.

In four subjects for whom Stages 3 and 4 data (combined into SWS) were available, frontal P1 amplitude decreased compared to awake presentations. As above, central N1b amplitude but not latency was changed (smaller) during SWS. Data for N1a and N1c were equivocal, depending on the hemisphere of recording. As compared to awake, P2 amplitude was greater parietally. Central N340 amplitude was also increased relative to awake.

For the infrequent versus the frequent stimuli, P1 latency was increased during Stage 2 versus awake. There was a significant decrease in N1c latency during Stage 2. For the later components, there was an increase in central P2 and P420 amplitudes. For the SWS recordings, there was an increase in frontal N340 amplitude. Surprisingly, the P420 component was not detected during the sampling window of 800 msec.

In a study of nine subjects (age range = 18–25; one male, eight females), Ogilvie, Simons, Kuderian, MacDonald, and Rustenburg (1991) examined the changes in late ERP components during the transition from wakefulness to sleep. They used behavioral measures (pressing a button to terminate a tone delivered through a hearing aid) to determine sleep onset; the first failure to press the button to terminate the tone defined sleep onset. Statistical analyses of the amplitudes of the ERP components showed significant changes in all but P2 (largest positive peak in the 350 - 500 msec window). As behaviorally-defined sleep onset approached, N1 (largest negative peak in the 75 - 150 msec window) became less negative, P1 (largest positive peak in the 150 - 250 msec window) became more positive, N2 (largest negative peak in the 250 - 350 msec window) became more negative, N3 (largest negative peak in the 500 - 750 msec

window) became more negative, P3 (largest positive peak in the 750 - 1000 msec window) become more positive, and P300 ("classic" P300; the largest positive peak in the 250 - 500 msec window) became less positive.

A major confound in this study involved the instructions given to the subjects prior to the testing. Subjects were told to continue to respond while awake, while trying to obtain a representative night's sleep. Thus, the behavioral definition of sleep onset, response failure, could have resulted simply from the subject's subjective perception of sleep onset rather than an objective change in arousal level or arousal state. Again, the small sample size made the results from the study preliminary, at best.

In a recent conference presentation, Winter, Kenemans, Kok, and Elton (1993) presented data on the late ERP components recorded in an afternoon nap protocol. Twelve subjects (age range unknown; six males, six females) were presented with standard, small deviant, and large deviant tones, at 65 dB. The stimuli were, respectively, 1000 Hz, 1200 Hz, and 2000 Hz tones. There were three counter-balanced conditions: attend, ignore, and drowsiness/sleep. The attend condition required subjects to count one or the other of the deviant tones. During the ignore condition, the subjects were instructed to read a book. The drowsiness condition was defined as NREM Stage 1 sleep, while the sleep condition consisted of Stage 2 sleep, defined by the presence of sleep spindles.

The results indicated that in the attend condition, both deviant stimuli were associated with larger N1 and P3 components. In the ignore condition, only the large deviant produced a significant difference from the standard stimulus, and only in P3a. In the drowsiness condition, both deviant stimuli evoked larger P210, N330 (which appeared to be N200), and P450 (which appeared to be P300 or P3b) components. The large deviant was found to be associated with a significantly enhanced N130 component. During Stage 2 sleep, there was again an enhancement of the P210 and N330 components

to the deviant stimuli. While the enhancement of the N130 component to the large deviant stimulus was absent, there was an enhancement of the P450 component, versus the other two conditions. In the limited data from Stages 3 and 4 sleep, only the large deviant stimulus evoked enhanced N330 and P450 components.

There were three main problems with this study design. First, the experimenters stated that they were scoring sleep as per the Rechtschaffen & Kales (1968) criteria, yet they did not consider Stage 1 to be sleep – in violation of the clear wording of the sleep scoring manual. Second, the interstimulus interval used was only one second. As is clear from the research above, the P3 component can extend up to or beyond 1000 msec. after a stimulus during sleep, or even immediately prior to sleep. Third, there were no statistics reported in the report, thus it is impossible to interpret the effect sizes. However, there were good design elements, such as the use of an oddball design and the inclusion of different deviant stimuli.

In a poster presentation from the same conference as the previous study, Sallinen, Kaartinen, and Lyytinen (1993) reported a comparison of mismatch negativity to the K-complex during Stage 2 sleep. The stimuli were frequent, 1000 Hz tones and infrequent, 1200 Hz tones. Mismatch negativities were observed only in trials which also elicited a K-complex, not in those with only an EEG response or no EEG change. In their discussion section, the authors raised the point that the failure to find a mismatch negativity during the other trials could have been a result of variable latency to the negativity. The authors could have used latency adjustment procedures, such as Woody filtering, to verify this possibility.

### 3.3.4 General criticisms and summary

As indicated above, almost all of the research on ERPs and sleep have used very small sample sizes, usually less than ten. The use of larger sample sizes would permit not only firmer conclusions, but the examination of effects such as gender differences and light versus heavy sleepers. Additionally, many studies utilized only a single night of sleep recording, a criticism commonly leveled against any study involving the recording of sleep in human subjects. The commonly observed "first night effect", where subjects take longer to fall asleep and skip the first REM period of the night, could limit the generalizability of these findings to "normal" human sleep. It is possible that subjects are hypervigilant during the first night of polysomnography, both due to sleeping in a new environment and because of having numerous electrodes affixed to their scalps and faces.

It is also of concern that most studies did not report data on NREM Stage 1 sleep. One reason for this may have been the difficulty in getting subjects to sleep through the stimuli necessary to evoke the brain responses of the ERP. Regardless of the difficulties in obtaining ERPs during Stage 1, the area remains unstudied. Another area which appears not to have been examined concerns the sleep ERPs of older adults. As indicated above, most studies used subjects below age 40. The use of different age groups in the same study, or an all-inclusive group containing a broader age range would allow for observation of age effects.

Further research might benefit from studying populations thought to be more vigilant or aroused during sleep, such as subjective insomniacs or alpha-delta sleepers. Perhaps there would be amplitude and/or latency differences in the late, endogenous components of the ERP that would distinguish these groups from normal sleepers.

What is clear from the findings reviewed above is that 1) the early components of the ERP appear unaltered during sleep, 2) though research is very limited, some of the

middle-latency components are unaltered during sleep while others have slight changes, 3) the late components evidence the greatest changes from wakefulness to sleep and change as a function of sleep stage. What remains unstudied is how late components elicited by unmeaningful and semantically meaningful stimuli change in response to sleep, or at the transition from wakefulness to sleep.

### 3.4 Conclusions and Present Study

As mentioned in the introduction, the purpose of this introduction is to provide the background material from event-related potential and memory research necessary to apply this technology to the study of memory functioning during the sleep onset transition. Given the information presented above, the present study draws from the previous work in these areas.

A design to study memory at the wake-sleep transition (Wyatt et al., 1994) in a compromise with the Bentin & Moscovitch (1990) ERP methodology might prove worthwhile. While keeping the basic presleep design from our past study, the present study incorporated the ERP and the subject response time measures, as employed in Bentin & Moscovitch's work. The present study also included ERP measures to address the shortcomings of our past research, as mentioned above. We hypothesized that the present study would replicate the earlier findings of amnesia for stimuli presented immediately prior to sleep onset, using a different stimulus presentation design. It was hypothesized that stimuli might not be encoded sufficiently in the few minutes immediately prior to sleep onset to produce a von Restorff effect. Alternatively, perhaps if the amount of post-stimulus sleep was kept brief (30 seconds), the effect would still be observable. It was also hypothesized that a repetition priming effect would be observed in the ERP data collected during post-sleep recognition testing.

In general, the use of ERPs should provide many useful and unique contributions to the study of memory at sleep onset. They could provide an index of on-line attention, perhaps varying as a function of proximity to sleep onset. The later components, such as P3 (referred to as the Late Positive Component, or LPC), will allow inferences about differences in strength of encoding or updating the contents of working memory as sleep onset approached. Finally, it is possible, as shown in the repetition priming and ERP studies reviewed above, that ERPs could provide differential information about explicit and implicit memory functioning during the sleep onset transition.

## 4. METHODS

### 4.1 Subject Selection

Thirty undergraduate subjects enrolled in Introductory Psychology courses at the University of Arizona in the Fall semester of 1993 were recruited by a poster advertisement (see Appendix I) in the Psychology building. Inclusionary criteria were:

1. English as first or primary language (self-report)
2. No self-report of major sleep pathology (ie., chronic insomnia, sleep apnea, narcolepsy)
3. Average reported nocturnal sleep latency of 20 minutes or less
4. Average sleep latency of 20 minutes or less during afternoon naps
5. Average reported total sleep time of greater than five hours and less than nine hours
6. Right-handedness (self-report)
7. Self-reported interest in participating in a laboratory sleep experiment

Exclusionary criteria were as follows:

1. Current nicotine use of any amount, by any route of administration
2. Average reported weeknight bedtime before 9:00 pm. or after 1:00 am.
3. Self-report of past major head injury or concussion
4. Self-report of hearing deficiency
5. Self-report of memory deficit

6. Current use of over-the-counter (OTC) or prescription medication(s) possessing stimulant, depressant, or amnestic properties (ie., certain antidepressants, antihistamines, anxiolytics, and benzodiazepine hypnotics)

Trained research assistants gave all respondents who satisfied all criteria for participation a brief description of the research protocol and asked the respondents to participate in the study. Those consenting were scheduled for two appointments: one for memory testing (described below) and another on a separate day for the laboratory study (also described below). Subjects were given a research consent form (see Appendix II) to sign at the first appointment and were asked to conform to the following rules for the laboratory study.

1. No alcohol or illicit drug use during the three days prior to the laboratory appointment
2. No napping during the three days prior to the laboratory appointment
3. Bedtimes must be between 10:00 pm. and 1:00 am. and waketimes between 6:00 am. and 9:00 am. on the three days prior to the laboratory appointment
4. Less than the equivalent of 100 mg. of caffeine (one cup of moderately strong coffee or two caffeinated sodas) on the morning of the laboratory appointment and absolutely no caffeine intake after 10:00 am. that day

## 4.2 Apparatus

### 4.2.1 Auditory stimulus material

The target (or rare) auditory stimuli for the study were single-syllable, concrete nouns words culled from Carol, Davies, & Richman's (1971) book of word frequencies. Words were chosen to have moderate-to-high frequency of occurrence in the normative sample of ninth-graders. Words were divided into nine matched lists, which allowed for three trials per subject; of three lists, one served as a target or repeated words for the presentation phase and the words from the other two lists served as distracters or unrepeated words during the recognition task. The "frequent" auditory stimuli for the presentation phase were 1000 Hz, sine wave tones of 50 msec duration. These tones were generated from an Apple Macintosh PowerBook 170 computer with Farallon's SoundEdit software (version 2.0.5), using the "Tone Generator" function (rise time and fall time were each 10 msec).

### 4.2.2 Audio recording and playback equipment

The word stimuli were recorded onto a personal computer, using a custom program. Words were converted from analog to digital signals with an AdLib Gold 1000 audio card resident in the personal computer. Individual word recordings were parsed to be less than 1500 msec and saved as individual files. Custom software programs controlled the presentation phase and the subsequent recognition testing.

All auditory stimuli were presented to the subjects from the personal computer via two, small, bookshelf speakers (Radio Shack #40-2039B), amplified by a 20-watt P.A. system (Radio Shack #32-2033A). The speakers were located at the headboard of the bed, on each side of the subject's head, at a distance of approximately two feet.

#### 4.2.3 Electrophysiological monitoring equipment

All electrophysiological signals, the auditory stimuli, response button presses, and an event marker (for auditory stimuli) were simultaneously written to chart paper by a Grass Instruments Company Model 8-16E electroencephalograph (hereby referred to as the "polygraph") and digitized to computer for later spectral and ERP analyses. Analog signals were recorded with AC differential amplifiers with low and high frequency filters set to 0.3 Hz and 35 Hz, respectively. Analog signals were digitized by a Dell 310 personal computer with a Data Translation DT-2801 A-D board, governed by Stellate System's Rhythm software (Version 8.0). The analog information was sampled at 128 Hz and stored to magnetic tape (DC-6150) after each testing session.

The experimenters applied numerous electrodes to the subjects in order to obtain the physiological data. Scalp electroencephalogram (EEG) electrodes were placed at sites necessary to conduct both polysomnographic (PSG) and event-related potential (ERP) recordings, using scalp locations measured according to the 10-20 System (Jasper, 1958). These sites were Fz, C3, Cz, C4, Pz, and O1. All scalp electrodes were the gold-plated silver cup-type and were affixed with collodion-soaked gauze squares. The scalp sites, pre-cleaned with acetone, used HP Redux paste for a conductant. Gold-plated silver disk-type electrodes were placed for ground (ISO), linked-earlobe reference (A1-A2, for Fz, Cz, and Pz), individual mastoid reference (A1 and A2, for horizontal EOG and C3, C4, and O1) left and right horizontal electro-oculogram (EOG), right vertical-to-horizontal EOG, and bipolar submental electromyogram (EMG). These face electrodes were held in place with double-sided adhesive tape collars and covered with Micropore surgical tape. These sites were pre-cleaned with isopropyl alcohol and also used HP Redux paste for a conductant. (See Appendix III for polygraph amplification and filter settings.)

#### 4.2.4 Response press equipment

For obtaining behavioral responses during the recognition memory testing, the subjects had miniature, momentary switches (Radio Shack #275-1571) taped to their thumbs, in such a position as to be easily pressed by their index fingers. The response buttons were connected to a personal computer in the laboratory's Control Room, via 18-gauge speaker wire. At the Control Room, the speaker wires terminated into a custom-made interface box, including batteries and resistors to produce the signals. The interface box included optical-isolation circuitry, to ensure the safety of the subjects. Signals from the interface box were fed into the personal computer running a program that, 1) recorded the onset of the each stimulus word, 2) timed the delay until response, and, 3) scored the accuracy of each behavioral response.

#### 4.3 Baseline Memory Assessment

A measure of the subjects' baseline, explicit memory performance was obtained from scores on the Wechsler Memory Scale - Revised (or WMS-R, Wechsler, 1987). The entire battery was given to each subject, which required approximately one hour to complete. The experimenter or a trained research assistant administered the test and the experimenter evaluated all test results. The scales of primary relevance to the study were the measures of attention and concentration, immediate verbal memory, and delayed verbal memory. Subjects' performance was not utilized as a screening measure, but was collected to explore correlations with memory performance during the laboratory testing sessions.

All WMS-R administrations took place between the hours of 1:00 pm. and 6:00 pm., the same time period as the laboratory testing sessions, to control for possible circadian and time-of-day fluctuations in memory performance and subjective and objective arousal. Baseline memory testing occurred at least 2 days prior to the sleep trials, but not earlier than one week.

#### 4.4 Laboratory Procedures

##### 4.4.1 Orientation and electrode application

On a given study day, the subject arrived at the laboratory at 12:00 pm. or 3:00 pm. for orientation. At approximately 12:15 pm. (or 3:15 pm.), either the experimenter or a research assistant began electrode application. At approximately 1:00 pm. (or 4:00 pm.), the subject, after having electrode impedances checked (all below 10K Ohms) and being allowed to use the laboratory's bathroom, was assisted to the bedroom for the testing session. The subject remained lying in bed in the dimly-lit bedroom for the three trials, lasting a total of approximately 2 hours.

##### 4.4.2 Pre-trial procedures

After the subject was in bed, the experimenter returned to the Control Room and began standard subject calibrations (see Appendix IV). Following the subject calibrations, the subject gave verbal feedback about the volume of sample auditory presentations. The volume level was adjusted until the subject rated the level as loud enough to hear the words clearly, but quiet enough so that he/she could fall asleep during the stimuli. Then, the subject was read set of instructions for the trials (see Table 2

below). If there were no questions, the experimenter started all recordings and activated the computer program for the presentation phase of the first trial.

TABLE 2. Trial Instructions

<b>TRIAL INSTRUCTIONS: PRESLEEP</b>	
•	"Now we are ready to begin. I would like you to get into your favorite body position for falling asleep."
<b>PAUSE FOR SUBJECT TO GET COMFORTABLE</b>	
•	"I am going to play you some words over the speaker now and ask you to help me set the appropriate volume. The words should be loud enough so you can hear them clearly, but quiet enough for you to be able to fall asleep."
<b>PROCEED WITH VOLUME CALIBRATION</b>	
•	"In a few minutes, I will turn out the light and begin playing you words and beeps. You should try to listen to them, but you don't need to concentrate on remembering them. Your primary task is just to relax and not to resist the urge to fall asleep."
•	"At some point, at which you may or may not be asleep, I will call you over the intercom and ask you some questions. You will answer some of the questions verbally, and some with the buttons taped to your thumbs. You won't need the buttons until then."
•	"Do you have any questions?"
•	"In order for us to get a good recording, please lie quietly, with your eyes closed, and try to fall asleep."
•	"Good night."
<b>LIGHTS OUT</b>	
<b>WAIT 15 SECONDS</b>	
<b>START RHYTHM RECORDING</b>	
<b>PROCEED WITH PRESENTATION</b>	

#### 4.4.3 Stimulus presentation

During the presentation phase, the target stimuli were presented at a rate of one target word per ten-second block, for a total of six target words per minute. Frequents (beeps) were presented four times per ten-second block, for 24 frequents per minute. The ordering of the frequents and the target within each ten-second block was random (see Table 3 below).

TABLE 3. Stimulus Presentation

Example of presentation word/tone ordering  
for an arbitrary one-minute period

**First 10-second period...**

beep	beep	beep	beep	TARGET 1
<2 sec>	<2 sec>	<2 sec>	<2 sec>	<2 sec>

**Second 10-second period**

beep	TARGET 2	beep	beep	beep
<2 sec>	<2 sec>	<2 sec>	<2 sec>	<2 sec>

**Third 10-second period**

beep	beep	TARGET 3	beep	beep
<2 sec>	<2 sec>	<2 sec>	<2 sec>	<2 sec>

**Fourth 10-second period**

beep	beep	TARGET 4	beep	beep
<2 sec>	<2 sec>	<2 sec>	<2 sec>	<2 sec>

**Fifth 10-second period**

TARGET 5	beep	beep	beep	beep
<2 sec>	<2 sec>	<2 sec>	<2 sec>	<2 sec>

**...Last 10-second period**

beep	beep	beep	TARGET 6	beep
<2 sec>	<2 sec>	<2 sec>	<2 sec>	<2 sec>

Each presentation phase was continued until the experimenter observed fifteen seconds of continuous, sleep EEG on the polygraph. At that point, the subject was allowed to accumulate up to either 30 seconds or ten minutes of sleep (random selection). Following the sleep period, the experimenter called the subject's name over the intercom loudly and repeatedly, until a verbal response was obtained from the subject.

#### 4.4.4 Memory testing

Immediately after obtaining a verbal response from the subject, he/she was asked over the intercom to recall any words heard during the trial (see Table 4 below). After the subject stated that no further words were recallable, the recognition task instructions were given (also see Table 4). During the recognition task, a computer program presented the subject with the last five minutes of target words presented prior to sleep onset, plus twice as many matched distracters (1:2 ratio), at a rate of one word per four seconds. Subjects were instructed to press the button strapped to their right hand when they heard a word that was presented before (repeated) and to press the button on the left hand after they heard a novel, unrepresented word (unrepeated). Subjects were asked to respond as quickly as possible, but without sacrificing accuracy. The computer program recorded the response latencies and accuracies, as described above. Following this task, the subjects were informed that another trial would begin after a brief delay (approximately 1 minute), during which the experimenter reset the computer audio program and EEG acquisition program for another trial. Testing was repeated for a maximum of three trials (presentation–sleep–memory testing), depending on the time the subject needed to leave the laboratory for other obligations.

TABLE 4. Memory Testing Instructions

TRIAL INSTRUCTIONS: POSTSLEEP	
FREE RECALL	
•	"Speaking slowly and clearly, please tell me any words you remember hearing during this trial."
RECOGNITION	
•	"You are now going to hear more words. Some of these you've heard before — others are new. When you hear a word you've heard before, please press the button in your right hand. When you hear a new word, please press the button in your left hand."
•	"When responding to the words, please press the buttons as quickly as possible, but without sacrificing accuracy."
END	
•	"OK. You will not hear these words again, so you do not need to remember them."

#### 4.4.5 Debriefing

Following the third trial, a research assistant escorted the subject to the hookup room and removed all recording electrodes. During this procedure, the experimenter briefly described the design of the experiment, covering such topics as the free recall task, the recognition task, and ERP recordings (see Appendix V). He also explained some of the primary purposes of the study — 1) to validate this ERP/memory procedure in the auditory domain; and, 2) to assess ERP indices of on-line processing associated with memory functioning during the sleep onset transition.

## 5. RESULTS

### 5.1 Memory Performance

#### 5.1.1 Baseline memory performance

In order to assess the generalizability of the results from this study to the general population, all subjects were given the Wechsler Memory Scale-Revised (WMS-R) during the week prior to the sleep study, as a test of waking memory performance. Based on the means and standard deviations for the Verbal Memory (99.43 AND 11.99) and Delayed Recall indices (106.03 and 12.42), it appeared that our subjects were representative of the WMS-R's normative sample (see Appendix VI for complete WMS-R results).

#### 5.1.2 Free recall task

For the free recall task, word stimuli were coded with a score of "1" for correct responses. Correct responses were scored when the subject replied with the exact target word or any form of the word. Word stimuli were coded with a score of "0" for incorrect responses. Incorrect responses consisted of either the subject failing to free recall a given target word or recalling a word not presented during that trial. In order to analyze the free recall responses in terms of proximity to sleep onset, six-word bins were created by numbering the words backward, based on the six words presented in the minute prior to sleep onset. For example, for a subject who fell asleep after 30 words, the words presented 25th through 30th would be considered the stimuli presented in Minute 1 prior to sleep onset. Words presented 1st through 6th would be considered the stimuli

presented in Minute 5 prior to sleep onset. Data were omitted for all subjects ( $n=2$ ) who did not have data for each sleep condition.

In order to determine whether the results from this study replicated our earlier findings, a within-subjects  $2 \times 5$  ANOVA (Sleep Condition, Minute) was conducted on the free recall data. There was a significant effect of Sleep Condition, with subjects recalling significantly more words following 30 seconds of sleep, versus ten minutes ( $F[1,27] = 6.718, p = .0152$ ; 30 second mean = 5.25%, 10 minute mean = 2.03%). Below is a graph (Figure 6) showing the subject's recall of words from each of the five minutes prior to sleep onset. The interaction of Sleep Condition and Minute was not significant. However, it appeared that subjects had impaired recall of the words presented in the four minutes prior to sleep onset when tested following ten minutes of sleep, versus 30 seconds. As an exploratory measure, paired t-tests were conducted on each minute prior to sleep onset; subjects' free recall performance was significantly better in the 30 second sleep condition versus the ten minute condition for three of the four minutes prior to sleep onset (see Table 5 below). Thus, these results replicate our earlier findings.

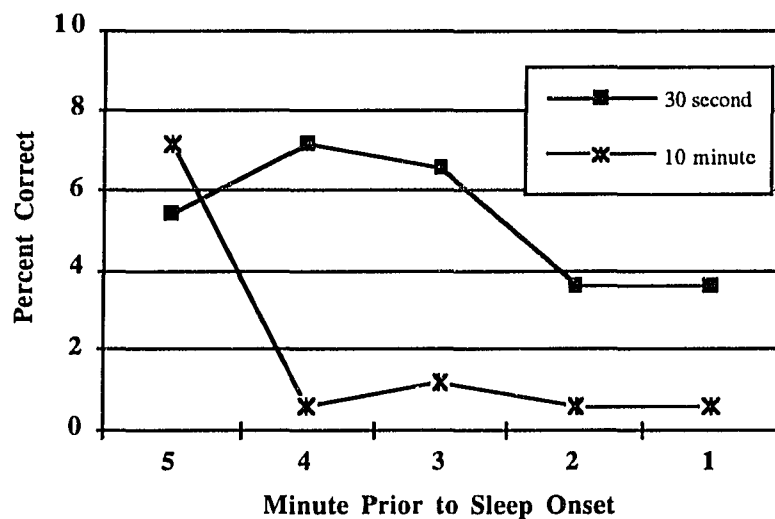


FIGURE 6. Free Recall

TABLE 5. Paired-t tests

	Minute 5	Minute 4	Minute 3	Minute 2	Minute 1
df	21	26	27	27	27
paired-t	-.746	2.275	2.353	1.728	2.423
probability	p = .4638	p = .0314	p = .0261	p = .0953	p = .0224

### 5.1.3 Recognition task: Explicit responses

For the recognition task, each target word was presented in a 3-word group with two randomly-selected distractor words. For data analysis, the target word responses were reordered according to the target words' presleep presentation order. Target words were binned in groups of six, to permit an analysis of recognition performance relative to the timing of sleep onset (as described above for free recall task).

Each pair of distractor-words was kept with its target word, in order to permit an analysis of response bias across the recognition testing session. Target words were given a score of "1" if the subject pressed the button indicating that the word was presented during the trial. Distractor words were also given a score of "1" if the subject responded that the word had been presented in that trial. All words were given a score of "0" if the subject pressed the button indicating that the word was not presented during that trial. A score of "0" was also assigned if the subject did not press either response button within the allotted time.

An average of each pair of distractor-word scores was calculated. An adjusted score for each target word was calculated by subtracting the average score of the matched distractor-words from the target word score, thus controlling for response bias (the tendency of a given subject to press the button indicating that the stimulus was a presented word).

A within-subjects 2x5 ANOVA (Sleep Condition, Minute) was conducted on the recognition data, adjusted as explained above. There were no significant effects. However, based on a visual analysis of the data, a further analysis was conducted, limited to the three minute window prior to sleep onset. A within-subjects 2x3 ANOVA (Sleep Condition, Minute) was conducted, adjusted as explained above. Subjects for whom there was not complete data for each sleep condition (n=3) were omitted from this analysis. There was only a significant main effect of Minute, with recognition performance being worse for words presented closer to sleep onset (see means in the Table 6 below). To further explore the recognition data, a within-subjects 2x3 ANOVA (Sleep Condition, Minute) was conducted on the data unadjusted for false positive rate. Again, there was only a significant main effect of Minute, as in the adjusted data (see means in the Table 6 below; see also Figure 7 for unadjusted data).

TABLE 6. Main Effect of Minute Prior to Sleep Onset: Percent Correct

			Minute 3	Minute 2	Minute 1
Adjusted Data	$F(1,26) = 7.425$	$p = .0015$	20.06%	6.79%	5.09%
Unadjusted Data	$F(1,26) = 7.5636$	$p = .0013$	49.69%	39.81%	36.73%

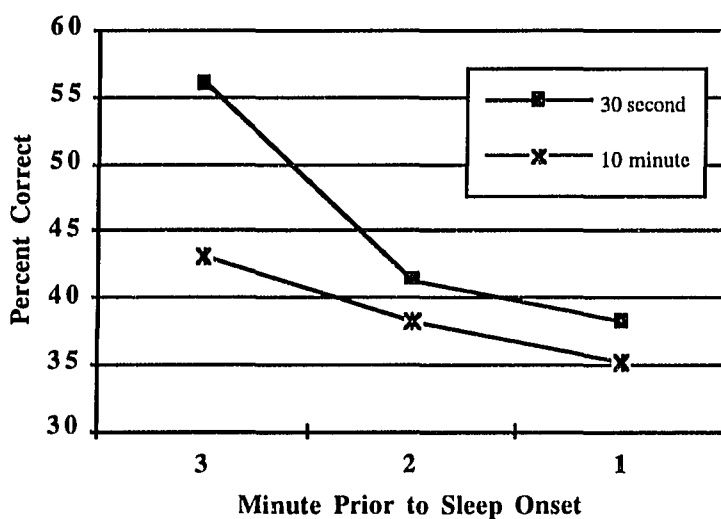


FIGURE 7. Recognition

#### 5.1.4 Recognition task: Reaction time

Recognition task reaction times were calculated on-line by a computer, based on the time between the onset of the word presentation and the onset of the subject's first response button press. Similar procedures (as described above) were followed to reorder the response latencies to target stimuli based on initial presentation order, while keeping each target response latency matched with the latencies to the two distractor words tested proximal to each target. Response times outside the 2500 msec. window allowable by the protocol were eliminated from the statistical analyses. Response latencies below 300 msec were discarded from the analyses based on two hypotheses. First, these extremely fast responses could have actually been delayed responses to the preceding stimulus word. Second, a response shorter than 300 msec could have been a premature response or a twitch made by the subjects prior to having heard the stimulus word.

A within-subjects 2x2x2 ANOVA (Sleep Condition, Repetition, Accuracy) was conducted on the data from the reaction times. There was a trend in the main effect of Sleep Condition ( $F[1,27] = 3.328, p = .0792$ ), with faster recognition times in the 30 second trials (see Figure 8 below). There was a significant main effect of Repetition ( $F[1,27] = 13.406, p = .0011$ ), with faster reaction times for repeated words (see Figure 9 below). There was a significant main effect for Accuracy ( $F[1,27] = 13.191, p = .0012$ ), with faster reaction times for correct responses (see Figure 10 below). There was a trend in the 2-way interaction of Sleep Condition by Repetition ( $F[1,27] = 4.144, p = .0517$ ). As can be seen in the Figure 11 below, the effect of repetition was greater in the 30 second sleep trials. Finally, there was a significant 3-way interaction ( $F[1,27] = 8.349, p = .0075$ ). As can be seen in the Figure 12 below, the effect of accuracy is due mainly to performance in the 30 second trials. The facilitated response for correct responses to repeated words suggests that in these trials, subjects had good explicit memory of these words.

To further examine the 3-way interaction, two post hoc 2x2 ANOVAs were conducted, to separate explicit from implicit effects. To examine for effects of explicit memory, a within-subjects 2x2 ANOVA (Sleep Condition, Response) was conducted on the data from the reaction times. There was a significant interaction of Sleep Condition with Response ( $F[1,27] = 12.396, p = .0015$ ). As stated above (see also Figure 12), only in the 30 second condition did the subjects have shorter response times to the hits than to the correct rejections. Thus, it was only in the 30 second trials that the response times showed an effect of explicit memory.

To examine for effects of implicit memory, a within-subjects 2x2 ANOVA (Sleep Condition, "No" responses) was conducted on the data from the reaction times. Contrary to prediction, subjects were not significantly faster in their incorrect responses to repeated

words than in their correct rejections of unrepeated words. There were no significant effects in this analysis.

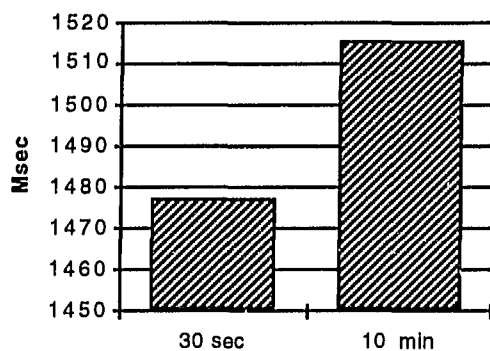


FIGURE 8. Sleep Condition

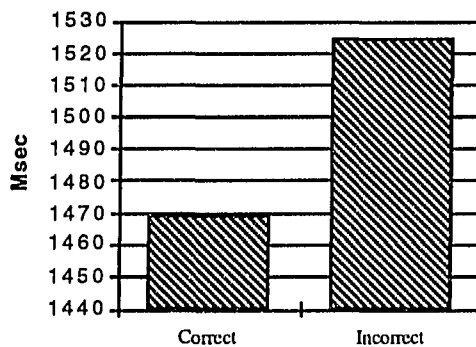


FIGURE 10. Accuracy

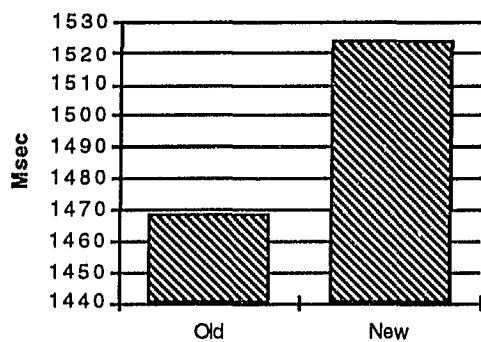


FIGURE 9. Repetition

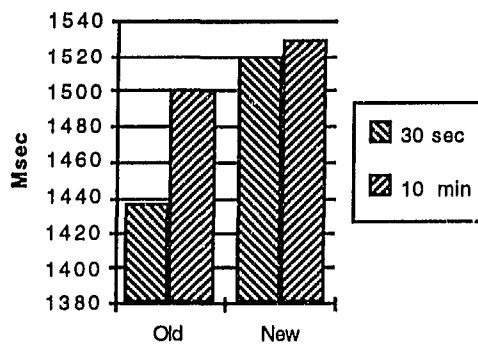


FIGURE 11. Sleep Condition by Repetition

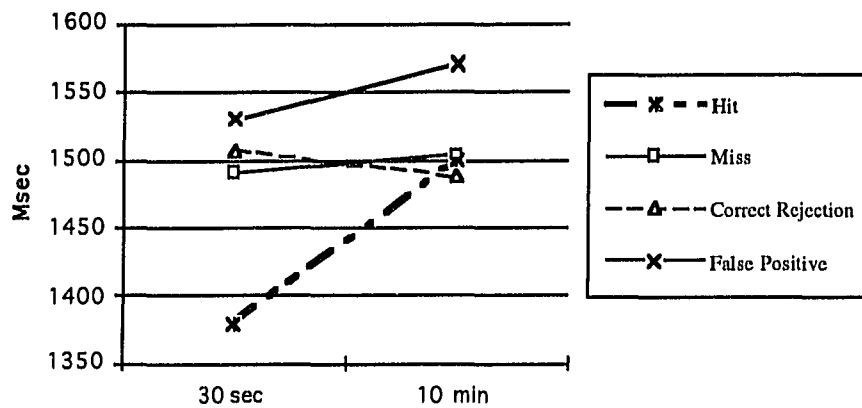


FIGURE 12. 3-Way Interaction

## 5.2 Sleep Data

### 5.2.1 Polysomnography

All sleep data from the 30-second and 10-minute trials were staged in 30-second epochs, according to standard criteria (Rechtschaffen & Kales, 1967). Sleep scoring was performed by a single, trained sleep researcher – the experimenter (JKW).

Each of the 30 subjects had at least one ten minute sleep trial that met the criterion of at least one half of the 30-second epochs being scored as sleep. Within the ten minute sleep condition, averaged across subjects, 17.07% of the epochs were scored as wakefulness. Within the first three minutes of sleep, this percentage of epoch scored as wakefulness rose to 25.56%. Within the 30 second sleep condition, across subjects, 100% of the single 30-second sleep epochs were scored as sleep.

The average sleep latency for the 30 second trials was 8.8 minutes, versus 8.4 minutes for the ten minute sleep trials. The sleep latencies were not significantly different across the sleep conditions (paired t-test;  $df=27$ , paired- $t = 1.39$ ,  $p = .176$ ).

### 5.2.2 Spectral analysis of the EEG

The EEG data from the 30-second and 10-minute trials were spectrally analyzed in 4-second, non-overlapping epochs. Sections of the EEG containing muscle or other artifacts, based on a visual inspection on the computer monitor, were rejected from the analyses. Relative spectral power was calculated on the following bandwidths: delta (0.75 - 2.75 Hz), theta (2.75 - 7.50 Hz), alpha (7.75 - 11.75 Hz), sigma (12.00 - 14.00 Hz), and beta (14.25 - 30.00 Hz).

Within the 10 minute and 30 second sleep conditions, power spectral analyses were conducted on the sleep EEG from sites C3, C4, O1, Fz, Cz, and Pz. For the 10 minute condition, the spectral analyses were limited to the data from the first three minutes of sleep. This time period was selected ad hoc, based on the duration of amnesia observed in previous research (Wyatt, 1994). Data were eliminated from the analyses if the subject did not have a valid trial of that sleep condition or if sweat artifact skewed the delta band power. Below are graphs (Figures 13 and 14) of the relative power, across each electrode site, for each frequency band.

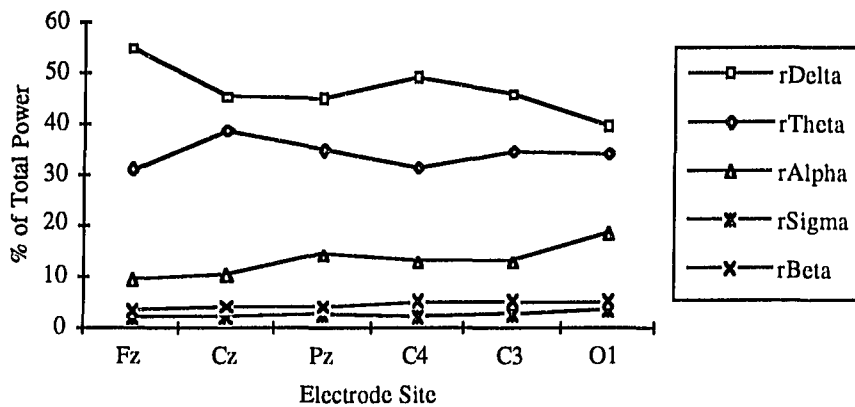


FIGURE 13. Relative EEG Power: 10 minute condition

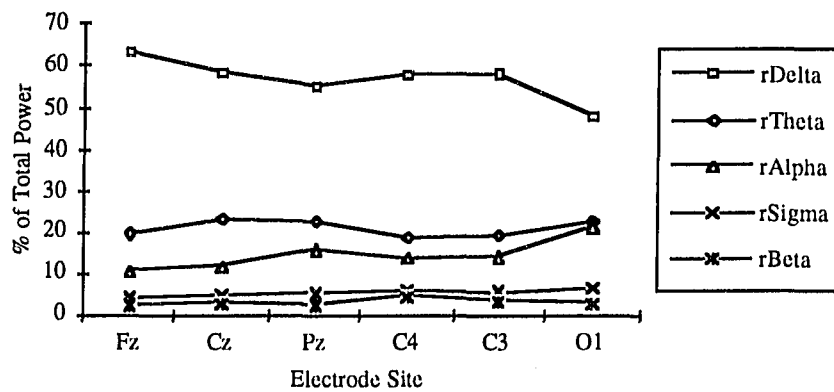


FIGURE 14. Relative EEG Power: 30 second condition

### 5.3 Event-Related Potential Data

#### 5.3.1 Initial stimulus presentation: LPC

For event-related potential analysis from the presleep period, 2400 msec EEG sections were cut by a program from the continuous EEG, including 500 msec prior to and 1900 msec following the stimulus onset. An amplitude scan was run and epochs where the average amplitude of any two continuous samples differed from the average

amplitude of the entire epoch by 70  $\mu\text{V}$  or more were rejected (based on the amplitude of visually-detected eyeblinks and other artifacts in the raw EEG signals). The segments were then digitally lowpass filtered (72 point, finite impulse response [FIR], with a half-amplitude frequency of 7 Hz). Waveforms were then linearly detrended. As the target stimuli were of variable length, the late components varied greatly in latency. Hence, simple averaging of the epoch yielded grand average waveforms that were not well-defined and appeared to have latency jitter. In order to correct for this confound, each EEG segment was shifted with a Woody adaptive filter (Woody, 1967), based initially on a positive potential occurring 500 - 1000 msec after the stimulus onset, with a sine wave template, for a maximum of 15 iterations or until the change in the correlation coefficient was less than .01. To determine the amplitude of the Late Positive Component (LPC), the highest positive potential within the search window on which the template was formed was selected. LPC amplitudes of the six word stimuli per minute were averaged, within each of the five minutes prior to sleep onset. Similarly, LPC amplitudes for each of the 24 beep stimuli were averaged, within each of the five minutes prior to sleep onset.

For the analyses in this section and the following section, data from subjects who did not have a valid trial from each Sleep Condition were eliminated from the analyses. Data from subjects who did not have ERP data for all five minutes prior to sleep onset were also not included in the analyses.

In a within-subjects 2x2x5 ANOVA (Sleep Condition by Stimulus Type by Minute Prior to Sleep Onset), there was not a significant difference in LPC amplitude for the main effect of Sleep Condition. This, taken with the nonsignificant interaction of Sleep Condition with Stimulus Type suggests that the subjects were processing the auditory stimuli similarly in the trials where they were later allowed either up to 30 seconds or ten minutes of sleep.

There was a significant main effect of Stimulus Type ( $F[1,18] = 24.804$ ,  $p = .0001$ ), with word stimuli being associated with larger amplitude LPCs than the beeps. The main effect of Minute was nearly significant ( $F[4,72] = 2.419$ ,  $p = .0563$ ), with the stimuli producing larger amplitude LPCs as sleep onset neared. These effects are shown in the Figures 15 and 16 below.

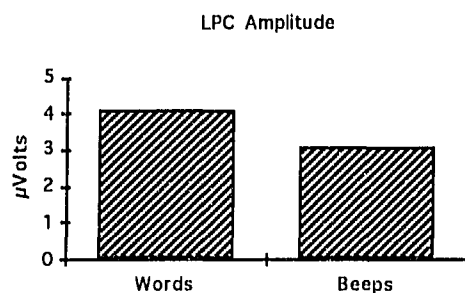


FIGURE 15. Stimulus Type

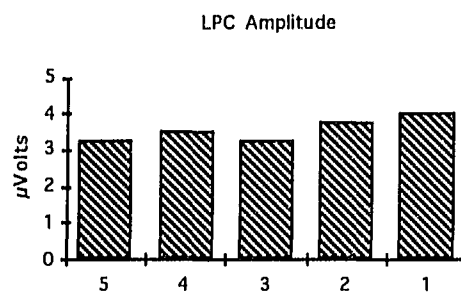


FIGURE 16. Minute

### 5.3.2 Initial stimulus presentation: N4

To process the presleep N4 components, similar procedures were followed (7-Hz digital and Woody filtering), as listed above. Based on a visual analysis of the ERPs without Woody filtering, the Woody adaptive filter was set for a negative potential, within the 575 to 875 msec window, with an initial sine wave template. The six word stimuli and 24 beep stimuli were averaged for each of the five minutes prior to sleep onset. The peak amplitudes of the N4 components were selected based on the highest negative peak within the search window on which the template was formed.

In a within-subjects 2x2x5 ANOVA (Sleep Condition by Stimulus Type by Minute Prior to Sleep Onset), as with the LPC amplitudes, there was not a significant difference in N4 amplitude for the main effect of Sleep Condition. Again, this with the

nonsignificant interaction of Sleep Condition with Stimulus Type suggests that the subjects were processing the auditory stimuli similarly in each delay condition.

There was a significant main effect of Stimulus Type ( $F[1,18] = 8.983, p = .0077$ ), with word stimuli being associated with larger amplitude N4s than the beeps. The main effect of Minute was significant ( $F[4,72] = 3.519, p = .0111$ ), with the stimuli closer to sleep onset producing larger amplitude N4s. These effects are shown in Figures 17 and 18 below. These results suggest that the subjects continued to differentially process the stimuli during the sleep onset transition.

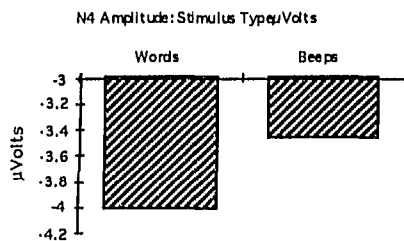


FIGURE 17. Stimulus Type

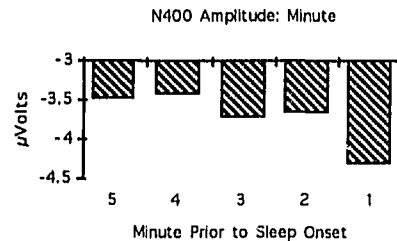


FIGURE 18. Minute

### 5.3.3 Von Restorff / Dm effect

An analysis was conducted to explore the hypothesis that words correctly recognized would be associated with larger amplitude LPCs at initial presentation than those later not recognized. Subjects who did not have data from both sleep conditions ( $n = 4$ ) were eliminated from the analysis. Presleep LPC amplitudes outside of 2 standard deviations from the group mean ( $n = 4$ ) were considered artifactual and eliminated from the analysis. During the recognition test, correctly recognized words were associated

with significantly larger amplitude LPCs at presentation (within-subjects 2x2 ANOVA [Sleep Condition by Recognition];  $F(1,21) = 6.765, p = .0167$ ). Figure 19 below displays the mean amplitudes at initial presentation of the LPCs elicited by the target words later recognized and not recognized.

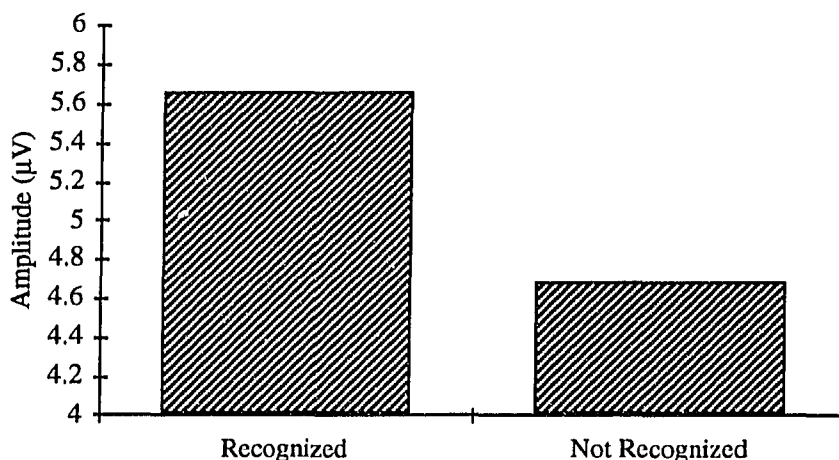


FIGURE 19. Encoding Strength: Presleep LPCs

#### 5.3.4 Recognition phase event-related potentials :LPC

Also as described above for the presleep ERPs, the recognition-phase LPCs were obtained from amplitude scanned, 7-Hz digital filtered and linear detrended, and Woody filtered segments of EEG. The EEG segments were cut from the ongoing EEG files, containing 500 msec prior to and 2500 msec following the target and distractor word stimuli. Peak amplitudes were picked based on a 500 to 1000 msec post-stimulus search window. ERP segments were sorted to correspond to the order of initial, presleep presentation (as described above). Due to the limited number of stimuli presented per minute and the associated inability to adequately increase the signal-to-noise ratio, it was

not possible to separate the analysis by minute prior to sleep onset. Thus, the target word ERPs were averaged across the five minutes prior to sleep onset.

To look for repetition priming, a within-subjects 2x2 (Sleep Condition by Repetition) ANOVA was conducted on the ERPs from the recognition testing. As hypothesized, there was a significant main effect of Repetition ( $F[1,21] = 8.147, p = .0095$ ; see Figure 20 below), with old (repeated) words eliciting larger amplitude LPCs than new (unrepeated) words. No other effects were significant in this ANOVA.



FIGURE 20. Repetition Priming

In order to provide a measure of explicit memory performance that would be parallel to the measure of implicit memory described below, a within-subjects 2x2 ANOVA (Sleep Condition by Response) was conducted, comparing the LPCs elicited to the old words judged to be old (hits) and the LPCs elicited to the new words judged to be new (correct rejections). There was a significant main effect of Repetition, with hits being associated with larger amplitude LPCs than correct rejections ( $F[1,20] = 7.144, p =$

.0146; hit mean = 6.2601  $\mu$ V, correct rejection mean = 5.0102  $\mu$ V). Thus, explicit memory was reflected in the amplitude of the LPC component during the recognition test.

In order to assess implicit memory performance in this data, a within-subjects 2x2 ANOVA (Sleep Condition by Response) was conducted, comparing the LPCs elicited to the old words judged to be new (misses) and the LPCs elicited to the new words judged to be new (correct rejections). Had there been significantly larger LPCs associated with the Miss than the Correct Rejection responses, this would have suggested an effect of implicit memory. However, no effects were significant in this ANOVA.

## 5.4 Combined Results

### 5.4.1 Polysomnography as a predictor of free recall performance

As the results of a previous version of this protocol had found a three minute window of amnesia in the 10 minute sleep condition, it was hypothesized that effective memory consolidation might require approximately three minutes of a sufficiently high level of arousal following the stimuli. To explore the relationship between impaired recall of words presented immediately prior to sleep onset (the four-minute amnesia window) and the subsequent arousal state, the percentage of 30-second epochs during the first three minutes of the 10 minute sleep period scored as wakefulness were correlated with the recall percentages of words presented in the four-minute window prior to sleep. The correlation was significant; a higher percentage of epochs scored as wakefulness was associated with better recall of the words from the window ( $r = .428$   $p = .0182$ ).

#### 5.4.2 EEG spectral power as a predictor of free recall performance

Based on the correlation reported above between wakefulness during the first three minutes of the 10 minute sleep period and free recall performance for words presented in the four minutes prior to sleep onset, similar correlations were performed with the spectral EEG data. Only relative power in the beta band, across EEG sites, correlated significantly with recall from the four minute window (see Table 7 below). In all cases, higher relative beta activity was associated with better recall of words from the four minutes prior to sleep onset. Also, within subjects, higher relative beta power correlated significantly with free recall percentage for all words presented in that trial.

TABLE 7. Correlations of Relative EEG Power (beta) and Free Recall Performance

Free Recall:	EEG Sites					
	Fz	Cz	Pz	C4	C3	O1
average for all words	r = .451 p = .0140	r = .447 p = .0150	r = .487 p = .0074	r = .525 p = .0035	r = .431 p = .0196	r = .482 p = .0081
4 minutes prior to sleep	r = .368 p = .0498	r = .497 p = .0061	r = .559 p = .0016	r = .479 p = .0085	r = .493 p = .0066	r = .523 p = .0036

#### 5.4.3 Baseline memory performance as a predictor of free recall performance

Only a small minority of subjects were able to recall any words presented during the four minutes prior to the 10 minutes of sleep. To assess whether these subjects had recall of material from this period due to above-average waking memory performance, a stepwise regression was conducted. Each of the WMS-R subscale scores as well as the raw and index scale scores were entered as predictor variables, against the percentage of words recalled from the four minutes prior to sleep onset. The subtest of delayed visual reproduction could not be entered, as the variance was zero. The only variable to enter

the regression was delayed verbal paired associates ( $F(1,27) = 13.99$ ,  $R\text{-squared} = .341$ ). However, the direction of this relationship was counter to expectation; the higher the delayed verbal paired associates score, the lower the recall of material from the four minutes prior to sleep onset.

Another stepwise regression was run with the same predictor variables, against the percentage of words recalled overall in the 10 minute sleep condition. None of the variables were entered into the regression. Thus, the data failed to support a conclusion that memory effects observed in the 10 minute sleep condition were related to baseline memory performance.

## 6. DISCUSSION

### 6.1 Summary of Major Findings

The results of the present study replicate earlier findings in this area. It was found that in trials where subjects were allowed to sleep for a ten-minute period following the presentation of auditory stimuli, performance was significantly worse on free recall and recognition tasks for these stimuli than when tested after being allowed to sleep for up to 30 seconds. In this study, we found that subjects were almost completely amnesic on free recall of words presented during the four-minute window prior to sleep onset. This compares favorably with previous results showing a three-minute window of amnesia.

Differences in the designs between studies could have been responsible for the difference in the length of the window of amnesia. This study entailed presenting the subjects with far more stimuli, more frequently (one word per ten-second window versus one pair of words per minute in the previous study). In addition, subjects in the present study were not required to repeat the words aloud. It is possible that this was associated with more shallow encoding of the stimuli. However, in the present study, there was also evidence that the subjects continued to process the meaningful auditory stimuli (the words) up until sleep onset. There was not a significant decrease, but rather an increase in the amplitudes of the LPC and N4 ERP components during the five minutes prior to sleep onset. The target stimuli (words) also elicited LPC and N4 components of larger amplitude than those elicited by the frequent stimuli (beeps). This suggests that the subjects were able to differentially process auditory stimuli up until sleep onset.

There was also evidence that individual stimuli that elicited larger-amplitude LPCs during initial presentation were more likely to be correctly recognized during post-

sleep memory testing. This result parallels findings in awake subjects, as described in the Introduction.

There were also significant correlations between the sleep parameters and memory performance within the ten-minute sleep condition. The subjects who were able to recall stimuli during the window of amnesia had more indications of wakefulness during the first three minutes of their sleep. Higher relative EEG power in the beta band, which is usually indicative of shallower sleep, was associated with better free recall performance. Similarly, subjects whose initial sleep period was fragmented, as judged by visual scoring of the polysomnograms, had better free recall performance.

Finally, repetition priming effects were observed in the ERP data collected during the recognition task. Repeated words, without respect to the accuracy of the subjects' behavioral responses, elicited higher amplitude LPCs than did new words. The recognition task LPCs also showed evidence of an explicit effect, with hits eliciting larger amplitude LPCs than correct rejections.

## 6.2 Stimulus Encoding During the Sleep Onset Transition

### 6.2.1 Differential EEG responses to meaningful and nonmeaningful stimuli: LPC and N4

As discussed in the Introduction, the amplitude of the LPC can be viewed as an index of the process whereby information is updated in working or short-term memory. In the present study, there was not a significant difference in the amplitude of the LPC components between the 30-second and ten-minute sleep conditions. Thus, a conclusion that differential amounts of processing during the presleep period could account for the different free recall performance between the sleep conditions, is not supported. A conclusion that is supported by this study is that subjects can continue to process auditory

stimuli until sleep onset. Both LPC and N4 amplitudes increased proximal to sleep onset. At least two explanations for this are possible. It could have been the case that the strength of the odd-ball paradigm increased proximal to sleep onset; target stimuli might have appeared subjectively less probably and/or more task relevant toward sleep onset. Second, the increases in amplitude could have been correlated with an overall trend toward synchronous EEG activity seen during and after the transition from wakefulness to NREM sleep. In addition to this increase in late component amplitude, subjects were able to differentially process meaningful and nonmeaningful stimuli during the five minutes prior to sleep onset, as evidenced by the significantly larger LPC and N4 components elicited by words versus beeps.

It is important to note that the latencies to the late components were highly variable in this study. As such, it was not possible to assess whether what is referred to in this study as N4 met the criteria set forth in prior research (latency of occurrence, relationship to degree of semantic mismatch). However, several lines of evidence from the present study suggested that what was observed could have been the traditional N4. First, the search window was picked based on a visual inspection of the ERP waveforms prior to latency adjustments. The presence of a large negative waveform between the 500 and 875 msec poststimulus window was evident in these waveforms, clearly outside the window of where one would expect to find N200. Second, it is also possible that the component referred to in this study as N4 corresponded to N200 for the beeps and N400 for the words. However, a component that looked to be N200 was evident in the raw ERP waveforms, at approximately the correct latency, without latency adjustment. Finally, that larger amplitude negative components were elicited by word than beeps supports a conclusion that the component is N4.

### 6.2.2 Arousal level: Sleep latency

The sleep latencies in both sleep conditions were not significantly different. This supports a conclusion that the subjects had comparable levels of sleepiness in the 30-second and ten-minute trials. Thus, arousal level during stimulus presentation cannot account for the difference in free recall performance between the sleep conditions.

### 6.2.3 Von Restorff / Dm effect

Previous results reported in the ERP literature have shown that stimuli for which subjects have better subsequent recall or recognition elicited larger amplitude LPC components during initial presentation. If one views the amplitude of the LPC component as an index of the amount and/or extent of stimulus processing, then it follows that stimuli that are recallable or recognizable were associated with more cognitive processing initially. In the present study, target words that were subsequently correctly recognized elicited larger amplitude LPCs than target words that were not recognized. It is important to note that this effect did not vary across the sleep conditions. It is also important to note this type of effect, as reported in studies of awake subjects, is present only when subjects engage in rote memorization. In contrast, when subjects utilize elaborative encoding strategies, the amplitude of a frontally-maximal slow wave correlates positively with subsequent memory performance. It is possible that subjects in the present study used rote memorization if they used any encoding strategy, which would not provide broad retrieval cues. This could account for the low performance observed in the free recall task. It would be important for subsequent research could utilize the present protocol with subjects given the instruction to use elaborative encoding techniques, in order to explore this issue further.

### 6.3 Consolidation

#### 6.3.1 Free recall data

In agreement with previous findings, subjects in this study were nearly completely amnesic for words presented in the four-minute window prior to sleep onset, when stimulus presentation was followed by a ten-minute sleep period. As described above, there is no evidence that initial encoding deficits alone, or at least deficits in rote memorization, can account for the deficit of free recall performance in the ten-minute condition, versus the 30-second condition.

#### 6.3.2 Delay between presentation and testing: Previous findings

Although not specifically addressed in this study, previous results from a similar protocol (Wyatt et al., 1994) supported the conclusion that the difference in free recall performance between the sleep conditions could not be attributed to the different delays between presentation and testing. Free recall performance was not significantly different in trials when subjects remained awake for either 30 seconds or ten minutes following stimulus presentation. Thus, it is unlikely that delay or simple "forgetting" due to elapsed time could account for the impairment observed in the free recall performance in the ten-minute trials from the present study.

#### 6.3.3 EEG spectral power versus memory performance

Within the ten-minute sleep condition, there were strong positive correlations between relative EEG power in the beta band during the first three minutes of sleep and

free recall performance, both for all words presented prior to sleep and for words presented during the four-minute window prior to sleep onset. Stated another way, subjects who evidenced a lesser degree of free recall impairment had more EEG indications of wakefulness during the first part of their sleep period. It could be hypothesized that this higher level of wakefulness allowed for better consolidation of information from short-term to long-term memory.

#### 6.3.4 Sleep fragmentation versus memory performance

Within the ten-minute sleep condition, there were also significant positive correlations between the number of 30-second epochs during the first three minutes of sleep scored as awake and subsequent free recall performance for the words presented in the four-minute window prior to sleep onset. As with the finding for beta EEG power reported above, this result suggests that lighter or more fragmented sleep allows consolidation to occur. Conversely, deeper or less-fragmented sleep that immediately follows the presentation of auditory stimuli is associated with poorer memory for those stimuli. It is important to note that this result and the one discussed immediately prior represent the use of a single-trait (EEG), multiple method design (FFT and sleep staging). Further research could benefit from broadening the protocol to measure other indices of arousal and attention, thereby using a multiple-trait, multiple method design. For example, convergent evidence from measurements of other signs of arousal level (such as galvanic skin response, or heart rate changes) could strengthen conclusions.

#### 6.3.5 Response latencies

The significant three-way interaction of sleep condition, repetition, and response accuracy, showed that in the 30-second sleep trials, subjects' response times were facilitated for repeated words that were correctly recognized as being repeated. This effect was absent in the 10-minute sleep trials. It is unlikely that sleep inertia, and hence a retrieval deficit, was responsible for this finding, as the subjects did not accumulate slow wave sleep during these brief trials. Again, it could be hypothesized that a certain duration and/or depth of sleep interrupted the consolidation of explicit memory, though further research is needed to examine this hypothesis.

#### 6.4 Implicit Memory

Previous research has been unable to make firm conclusions about implicit memory functioning, in this paradigm (Wyatt et al., 1994). The behavioral measure of implicit memory used in this study was the difference between the subjects' response times during the recognition task to repeated items judged to be unrepeated (misses) and to unrepeated items judged to be unrepeated (correct rejections). Contrary to prediction, subjects' response times for missed responses were not significantly faster than their correct rejections of unrepeated stimuli. It is likely that evidence of a significant implicit memory finding in this study was limited by two factors. First, the recognition memory task was phrased as an explicit memory task. Using an implicit task, such as a lexical decision task might have decreased the subjects' reliance upon conscious recollection. Second, it was clear in the accuracy of the subjects' responses that they had relatively intact explicit memory at the level of recognition testing (with correct responses to approximately half of the up to 90 words presented in a given trial). Therefore, it would have been unlikely to have been able to dissociate explicit and implicit memory in this protocol.

The ERP data collected during the recognition task failed to show an effect of implicit memory; repeated words judged to be new words ("misses") did not elicit significantly higher amplitude LPCs than new words judged to be new words ("correct rejections"). It is also possible that this negative finding resulted from the use of an explicit memory task. Perhaps the use of a semantic or lexical decision task, thus reducing the amount of interference from explicit memory, would have been associated with a different pattern of results.

## 6.5 Confounding Variables

### 6.5.1 Medication status

It has been well documented elsewhere that many prescription and over-the-counter medications can affect memory and/or arousal level. These include psychotropic medications, the hypnotics, anxiolytics, and antihistamines. In order to avoid confounding influences of these medications in this study, all subjects were screened to be free from any of these medications, on self-report. Similarly, subjects were instructed to abstain from alcohol and illicit drugs for the three days prior to the sleep study. Although subjects were not given objective tests to verify their drug-free status, it was decided that the within-subjects design and the reliance on valid self-reports from subjects adequately addressed this issue.

### 6.5.2 History of head injury

Much of what is known about memory functioning in normal individuals has involved comparison with individuals with known CNS lesions, including insults obtained during traumatic brain injury. Similarly, abnormal sleep patterns have been

observed in individuals who have suffered closed head injuries. Therefore, subjects were screened to have a negative self-report of head injuries, to screen out organic causes of memory or sleep abnormalities.

### 6.5.3 Baseline waking memory performance

In order to examine the generalizability of the results of the present study to the population, baseline memory performance was assessed in all subjects prior to the sleep study. As described above, our sample appeared representative of the age group in the WMS-R's normative sample, both in terms of the index score means and standard deviations. Furthermore, there were no significant correlations between the measures of the degree of deficit observed in the post-sleep memory testing and baseline memory scores. This suggests that the amnesia observed was not merely a function of pre-existing memory ability or impairment, but was instead related to the experimental manipulations.

### 6.5.4 Circadian fluctuations in short-term memory

As with many physiological and endocrine functions, short-term memory has been shown to follow a circadian pattern. Short-term memory performance has been shown to be at average daytime levels during the time window the subjects slept in the present study, and declines in successive hours (Monk, 1994) This makes the memory deficits observed in this study more impressive, as they occurred during the time of day when short-term memory has been shown to be relatively intact. One could hypothesize that further research utilizing the present protocol at nocturnal sleep onset might find

even greater memory deficits, due to time-of-day or circadian effects on short-term memory.

## 6.6 Conclusions

The presleep ERP results described above support the conclusion that short-term or working memory functioning is intact up until sleep onset. Therefore, observable memory deficits could be attributed to poor use of encoding strategies, impaired transfer of information from short-term to long-term memory, retrieval deficits, or a combination of the three factors.

With regard to the issue of memory consolidation, several explanations are possible. It could be that memory consolidation was impaired in a graded fashion, as a function of arousal level. In this way, the functioning of consolidation could be viewed along a continuum between fully awake/intact consolidation, and deep sleep/no consolidation. Alternatively, memory consolidation may have been blocked after arousal level fell below a certain level critical threshold. This threshold may have occurred during the sleep onset transition, at sleep onset, or at a certain depth of sleep. The linear correlations between beta EEG power while asleep and subsequent recall of presleep information supports the continuum hypothesis. Previous findings that memory performance is worse following just 30 seconds of sleep, versus 30 seconds of wakefulness, suggests that the downward slope of the function begins prior to sleep onset, as many researchers would not consider 30 seconds of sleep to be a "true" sleep onset. Support for the threshold hypothesis comes from the correlation observed between the percentage of awake epochs in the first few minutes of sleep and free recall for the stimuli presented in the four minute window prior to sleep onset. It could be the case that a certain duration of unfragmented sleep is required to completely block memory

consolidation from occurring. Further research could vary the length of the sleep conditions, to look for a dose-response relationship to memory impairment.

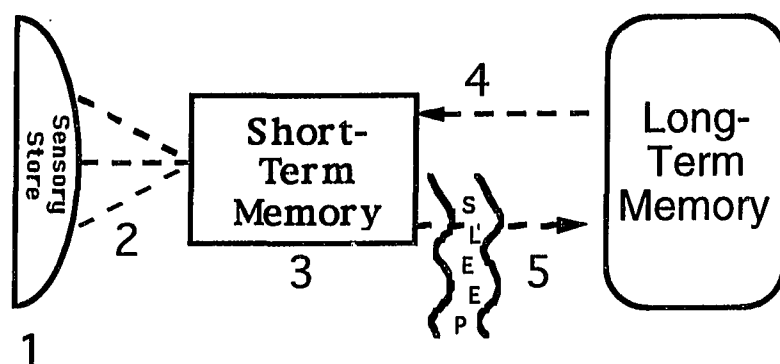
In order to compare these findings with others from cognitive neuroscience, the terms anterograde and retrograde amnesia become relevant. In keeping with the continuum hypothesis, anterograde amnesia would be said to have occurred in this protocol. As the subjects arousal level dropped, so did consolidation for subsequent stimuli. Alternatively, one could consider a certain depth or duration of sleep critical in blocking consolidation of material presented previously. This would indicate retrograde amnesia for the stimuli. As the results of this study support both hypotheses, a new term is needed to describe this amnesia – "mesograde amnesia".

The concept introduced here, mesograde amnesia, encompasses all aspects of normal memory functioning — encoding, consolidation, and retrieval. Several factors can lead to deficient encoding, including impaired attention and concentration, both of which might be mediated by arousal level. Arousal level likely decreases in a gradual function during the sleep onset transition, as the midbrain and forebrain receive less activation from the brainstem reticular activating system. Similarly, consolidation (beyond working memory) of ongoing stimuli might require a sufficient duration and level of arousal. Finally, if stimuli receive poor encoding and consolidation is weakened or blocked due to an insufficient level of arousal, then subsequent spontaneous retrieval would be nearly impossible. As described below, this model has applications beyond the study of memory during the sleep onset transition.

## 6.7 Broad Implications

One classic model of explicit memory functioning from cognitive neuropsychology utilizes the constructs of sensory store, short-term memory, and long-

term memory (Atkinson & Shiffrin, 1968). Applying the results of this study, as well as drawing findings from other sleep studies, it is possible to model how memory functioning differs in sleep versus wakefulness. The arrows in the figure (Figure 21 below) represent pathways of information transmission. In intact, awake individuals, it is assumed that all pathways are active. During sleep, numerous studies have shown that stimuli can reach the level of short-term memory (arrow #1 and #2; for review of this and following points, see Wyatt & Bootzin, 1994). Also, that subjects awaken preferentially to their own name versus someone else's name suggests the ability to draw upon information from long-term memory during sleep (arrow #4). That dream reports typically contain information from one's past also suggests that information can be accessed from long-term during sleep (also arrow #4). However, not a single study utilizing objective measures of sleep has shown convincing evidence that sleeping subjects can store new verbal information presented during sleep (arrow #5). Taken with the results of this study, it appears that the consolidation of new information is prevented during uninterrupted sleep.



- 1 Stimuli transduced to sensory impulses
- 2 Afferent transmission to STM
- 3 Reconstruction into unitary whole
- 4 LTM accessed for comparative information
- 5 Consolidation of new information to LTM

Diagram Elements: Support from previous research

Early ERP components: 1-2  
 Sleep mentation: 3  
 Differential arousability from sleep: 1-4  
 Sleep learning: failure at 5

FIGURE 21. Model of Memory and Sleep

Recent results from animal research strongly support the conclusion that certain limbic system structures become reactivated during sleep in patterns resembling those seen during learning tasks from the day prior to that sleep period (Wilson & McNaughton, 1994). Thus, one hypothesis could be that structures involved in the

processing of explicit memory become a dedicated system during sleep, with the task of reprocessing and consolidating information presented during prior wakefulness. The results of the present study could represent the time period when this system switches from processing ongoing information to reprocessing only information presented well before the transition to sleep, during full wakefulness.

The protocol used in the present study could also be applied to study clinical disorders of sleep. It has been frequently reported that insomniacs typically overestimate the amount of wakefulness they have during the night. One hypothesis that could be tested with this protocol is that the explicit memory systems of insomniacs do not block out the consolidation of new stimuli during the transition to sleep onset, and perhaps even into sleep itself. This would permit them to have memory for presleep and sleep mentation, which could be mistaken for periods of wakefulness. The present study's protocol could also be used to examine automatic behavior, a common symptom of disorders of excessive daytime somnolence, such as Narcolepsy or Obstructive Sleep Apnea. These patients often report that they are amnesic for events and activities they perform while presumably awake during the daytime. The clinical sleep literature commonly states that this memory deficit is the result of brief sleep periods called microsleeps, which are isolated periods (typically less than 15 seconds) where an individual's EEG goes from wakefulness, to sleep frequencies, and back to wakefulness. Further research with the protocol from the present study could test this hypothesis. It could also be hypothesized that extended declines in arousal level, but not including EEG-verified sleep onsets, could correlate significantly with the degree of memory impairment reported in these populations.

## APPENDIX I.

# LIKE TO NAP ?

Here's your chance! Nap for science! Nap for 4 credits! (Experiment #39)

The **Sleep Research Laboratory** is conducting a study of sleep and memory functioning. The protocol involves:

- taking a brief (1 hour) memory test, and,
- an afternoon sleep study, from 12pm to 3pm or 3pm to 6pm on either a Tuesday, Thursday, Saturday, or Sunday afternoon
- the sleep study involves listening to lots of words and taking several, short naps

As we are studying sleep and memory, all subjects must meet the following criteria to be eligible for participation (no exceptions):

- no history of major head injury
- no history of memory impairment
- English as first language
- right handed
- no history of (or current) sleep disorders
- ability to nap
- burning interest in having wires attached to you (temporarily), so we can record your sleep

If you meet the above criteria and are interested in participating, please call the Psychology Department's Sleep Research Laboratory, at **621-5127**, and leave a message with your name and telephone number. A research assistant will contact you to gather additional information, if appropriate, to schedule an appointment.

## APPENDIX II.

**RESEARCH CONSENT FORM**

**Title: MEMORY FOR EVENTS OCCURRING PRIOR TO SLEEP ONSET**

YOU ARE BEING ASKED TO READ THE FOLLOWING MATERIAL TO ENSURE THAT YOU ARE INFORMED OF THE NATURE OF THIS RESEARCH STUDY AND OF HOW YOU WILL PARTICIPATE IN IT, IF YOU CONSENT TO DO SO. SIGNING THIS FORM WILL INDICATE THAT YOU HAVE BEEN SO INFORMED AND THAT YOU GIVE YOUR CONSENT. FEDERAL REGULATIONS REQUIRE WRITTEN INFORMED CONSENT PRIOR TO PARTICIPATION IN THIS RESEARCH STUDY SO THAT YOU KNOW THE NATURE AND THE RISKS OF YOUR PARTICIPATION AND CAN DECIDE TO PARTICIPATE OR NOT PARTICIPATE IN A FREE AND INFORMED MANNER.

We would like to invite you to volunteer to take part in the research project named above. The purpose is to investigate memory for events that occur prior to the onset of sleep. If you agree to participate, you will have sensors attached to your face and scalp to measure eye movements, chin muscle activity, and brain waves during a series of brief, afternoon naps. These naps will take place during a single afternoon, in the Psychology Department's Sleep Research Laboratory. Before each nap, you will be listening to simple, verbal material, played over stereo speakers. After you are awakened from each nap, you will complete two, simple tests of memory. The total time you will be in the laboratory that afternoon will be approximately five hours.

To stabilize your sleep/wake cycle and to facilitate napping on the day of the study, you will be asked to maintain a sleep schedule for the three nights preceding the study: eight (8) hours of sleep for the third and second nights prior to the study and six (6) hours of sleep on the night prior to the study. You will also be asked to refrain from napping on those days.

On an afternoon during the week (seven days) prior to your laboratory afternoon, you will be given a memory assessment, lasting approximately one hour. This assessment consists of pencil-and-paper tasks as well as verbal questions.

We do not foresee any psychological or mental harm in participating in this study. You may experience slight skin discomfort after sensor/electrode removal. However, this risk is both infrequent and, when it does occur, quite transient.

The benefits of your participation will be primarily indirect. You will receive four credits toward your experimental participation requirement for Psychology 101. In

addition, you will be helping advance what is known about the functioning of memory at the transition from wakefulness to sleep. You will also get a chance to see what your sleep looks like on an EEG machine. You will also receive a verbal report about your scores on the memory assessment.

If you have any questions concerning your rights as a research subject, you may call the Human Subjects Committee office at 626-6721.

IN GIVING MY CONSENT BY SIGNING THIS FORM, I AGREE THAT THE METHODS, INCONVENIENCES, RISKS, AND BENEFITS HAVE BEEN EXPLAINED TO ME AND MY QUESTIONS HAVE BEEN ANSWERED. I UNDERSTAND THAT I MAY ASK QUESTIONS AT ANY TIME AND THAT I AM FREE TO WITHDRAW FROM THE PROJECT AT ANY TIME WITHOUT CAUSING BAD FEELINGS OR AFFECTING MY MEDICAL CARE. MY PARTICIPATION IN THIS PROJECT MAY BE ENDED BY THE INVESTIGATOR OR BY THE SPONSOR FOR REASONS THAT WOULD BE EXPLAINED. NEW INFORMATION DEVELOPED DURING THE COURSE OF THIS STUDY WHICH MAY AFFECT MY WILLINGNESS TO CONTINUE IN THIS RESEARCH PROJECT WILL BE GIVEN TO ME AS IT BECOMES AVAILABLE. I UNDERSTAND THAT THIS CONSENT FORM WILL BE FILED IN AN AREA DESIGNATED BY THE HUMAN SUBJECTS COMMITTEE WITH ACCESS RESTRICTED TO THE PRINCIPAL INVESTIGATOR, RICHARD BOOTZIN, PH.D., OR AN AUTHORIZED REPRESENTATIVE OF THE DEPARTMENT. I UNDERSTAND THAT I DO NOT GIVE UP ANY OF MY LEGAL RIGHTS BY SIGNING THIS FORM. A COPY OF THIS SIGNED CONSENT FORM WILL BE GIVEN TO ME.

\_\_\_\_\_  
Subject's Signature

\_\_\_\_\_  
Date

I have carefully explained to the subject the nature of the above project. I hereby certify that to the best of my knowledge the person who is signing this consent form understands clearly the nature, demands, benefits, and risks involved in his/her participation and his/her signature is legally valid. A medical problem or language or educational barrier has not precluded this understanding.

\_\_\_\_\_  
Investigator's Signature

\_\_\_\_\_  
Date

## APPENDIX III.

## POLYGRAPH SETTINGS

<u>Pen</u>	<u>Recording</u>	<u>Grid</u>	<u>M/μV</u>	<u>Sens</u>	<u>LF</u>	<u>HF</u>	<u>60</u>	<u>Pen</u>
1.	Paginator	-- (J6) --	μV	5	.3	35	in	1
2.	Frontal EEG (Fz)	Fz/A1-A2	μV	5	.3	35	in	2
3.	Central EEG (Cz)	Cz/A1-A2	μV	5	.3	35	in	3n
4.	Parietal EEG (Pz)	Pz/A1-A2	μV	5	.3	35	in	4
5.	Central EEG (C4)	C4/A1-A2	μV	5	.3	35	in	5
6.	Eye correction	Fp2/Y	μV	5	.3	35	in	6
7.	Central EEG (C3)	C3/A1-A2	μV	5	.3	35	in	7
8.	Occipital EEG (O1)	O1/A1-A2	μV	5	.3	35	in	8
9.	Right Eye (ROC)	Y/A1	μV	5	.3	35	in	9
10.	Left Eye (LOC)	X/A2	μV	5	.3	35	in	10
11.	Chin EMG	27/28/29	μV	2	10	70	in	11
12.	-----	-----	-	-	-	-	-	-
13.	Button #1	-- (J6) --	μV	-	-	-	in	13
14.	Button #2	-- (J6) -	μV	-	-	-	in	14
15.	Audio Marker	-- (J6) --	μV	-	-	-	in	15

## APPENDIX IV.

## Subject Calibrations Script

**"NOW please lie quietly... with your arms at your sides... keep your eyes open... and look straight ahead"**

- tech marks "EYES OPEN" on polygraph
- wait 30 seconds
- tech then performs 60-cycle checks on each active polygraph channel

**"Now please close your eyes"**

- tech marks "EYES CLOSED" on polygraph
- wait 30 seconds (making sure subject does not fall asleep)
- tech then switches to backup leads, performs 60-cycle checks, then switches back to primary leads

**"Now please open your eyes"**

- tech marks "EYES OPEN" on polygraph
- check video screen for compliance

**"Now without moving your head but just moving your eyes, please look to the left and hold it... right and hold it... left... right... and straight ahead"**

- check video and mark "L" near EOG channels on polygraph, then "R"... "L"... "R"... "STR"

**"Now please look up and hold it... down and hold it...up...down... and straight ahead"**

- check video and mark "U" near EOG channels on polygraph, then "D"... "U"... "D"... "STR"

**"Now please blink your eyes slowly five times"**

- mark "5 BLINKS" near EOG channels and underscore each blink

**"Now please grit your teeth... and relax"**

- mark "GRIT" below EMG channel

**"And now, how about a big yawn... and relax... GREAT!"**

- mark "YAWN" below EMG channel

-----  

The tech then proceeds with the Trial Instructions
--

APPENDIX V.  
SUBJECT DEBRIEFING SAMPLE

In this study, we are looking at the association between sleep and memory. We have already found that the decline in alertness that happens as someone falls asleep is associated with impaired memory for events occurring during that time. Also, we have found that a sleep itself can add to this memory impairment.

In today's experiment, we had you listening to words and beeps, until you fell asleep. After you fell asleep, we allowed you to obtain 30 seconds of sleep on some trials and 10 minutes of sleep on other trials. Then we awakened you and gave you two tests of memory -- a free recall test, and a recognition test. During the time you were falling asleep and during the memory tests, we were recording your brain wave activity. We are going to analyze these recordings to try to explain why we observe this memory impairment for events occurring just before someone falls asleep. The recordings will give us information about your level of alertness, your level of attention, and whether or not the words and beeps were making it into your short-term memory (also called working memory).

We also tested your memory ability with the Wechsler Memory Scale-Revised, a standard test of memory. On this task, you scored at the \_\_\_\_\_ percentile overall, which means that your performance is \_\_\_\_\_. We also got information about your ability to recall information after a delay; your scores indicated \_\_\_\_\_. Your memory for verbal information was \_\_\_\_\_. Your memory for visual information was \_\_\_\_\_.

After we have collected all of the data, we will spend several months looking at them. So, if you want to find out how you did on the memory tests, or what the overall study found, please drop by the lab next semester (sometime from February onward).

Finally, we ask that you wait until the end of this semester to tell your friends the details of this experiment. We don't want to bias anyone who might be a subject in the study. However, feel free to tell them everything we told you prior to your beginning the study.

That's it. Thanks again for participating.

## APPENDIX VI.

## BASELINE MEMORY PERFORMANCE

## WMS-R Scores

S#	Age	Sex	Info	MC	Fig	Mem	LM1	PI	PI	VR1	Dig	Vis	Span	Span	LM2	P2	P2	VR2
1	18	1	14	4	9	23	17	22	40	14	21	18	6	8	38			
2	20	2	14	4	9	33	18	24	41	24	18	31	6	8	41			
3	21	1	14	6	8	22	11	22	35	23	17	17	6	8	39			
4	18	2	14	5	8	36	18	20	41	11	17	36	6	8	41			
5	19	2	14	5	9	26	13	23	39	14	14	18	6	8	38			
6	18	2	14	4	9	32	18	22	38	21	22	27	6	8	40			
7	18	1	14	5	7	18	17	24	39	16	19	16	6	8	32			
8	18	1	14	6	10	36	17	24	40	21	17	33	6	8	39			
9	23	1	14	5	10	25	18	20	38	16	13	23	6	8	31			
10	19	1	14	4	8	21	15	19	41	21	18	15	6	8	41			
11	18	2	14	4	8	27	16	23	37	18	23	23	6	8	35			
12	19	1	14	6	10	21	15	23	39	17	20	12	6	8	36			
13	18	2	14	4	6	24	15	19	29	14	11	21	6	7	22			
14	18	1	14	6	9	29	17	23	37	22	24	21	6	8	39			
15	17	2	14	4	6	29	16	20	41	19	12	30	6	8	33			
16	18	2	14	4	7	28	16	23	39	20	22	23	6	8	36			
17	18	2	14	4	7	24	18	24	41	14	17	19	6	8	40			
18	18	1	14	4	5	19	17	23	34	11	14	18	6	8	33			
19	19	1	13	4	10	38	17	22	40	10	17	30	6	8	38			
20	18	2	14	5	9	27	18	23	41	23	22	23	6	8	38			
21	18	2	14	5	9	28	17	23	40	20	16	26	6	8	40			
22	18	2	14	4	10	31	17	24	39	15	19	31	6	8	35			
23	21	1	14	6	8	30	17	23	37	22	19	31	6	8	29			
24	18	2	14	5	9	29	18	24	33	16	14	28	6	8	26			
25	18	2	14	4	6	17	16	20	37	13	17	14	6	8	36			
26	20	1	14	4	7	34	18	24	37	18	15	30	6	8	38			
27	18	1	14	6	9	23	17	24	37	23	22	18	6	8	25			
28	19	2	14	4	6	34	18	23	40	15	17	30	6	8	39			
29	18	2	14	4	7	29	18	20	39	20	14	33	6	8	34			
30	18	2	14	4	7	28	17	21	36	16	21	27	6	8	34			
mean	18.63		13.97	4.63	8.07	27.37	16.67	22.3	38.17	17.57	17.73	24.07	6	7.97	35.53			
S.D.	1.25		0.18	0.81	1.44	5.51	1.6	1.64	2.76	4	3.43	6.6	0	0.18	4.94			
max.	23		14	6	10	38	18	24	41	24	24	36	6	8	41			
min.	17		13	4	5	17	11	19	29	10	11	12	6	7	22			

## WMS-R Scores (Cont.)

Raw Scores						=	Index Scores				
S#	Verbal	Visual	Delay	Attn/ Conc	Delayed	=	Verbal	Visual	General	Attn/ Conc	Delayed
1	68	66	134	74	84	=	90	124	99	102	100
2	90	68	158	88	100	=	114	138	124	118	130
3	66	54	120	86	84	=	91	95	93	114	103
4	92	67	159	61	105	=	112	125	120	86	98
5	75	61	136	61	84	=	98	109	100	85	100
6	86	65	151	90	95	=	108	121	115	120	117
7	60	63	123	75	76	=	83	115	92	103	90
8	96	67	163	82	100	=	118	129	126	110	127
9	70	66	136	63	82	=	95	91	102	87	100
10	61	64	125	82	84	=	84	119	93	110	100
11	77	61	138	86	86	=	99	109	102	115	103
12	65	64	129	80	76	=	88	119	96	108	90
13	67	50	117	54	69	=	90	84	87	75	83
14	81	63	144	98	88	=	103	115	108	•	106
15	78	63	141	66	91	=	78	114	102	92	107
16	79	62	141	88	87	=	101	112	105	117	104
17	72	66	138	66	87	=	94	124	102	92	104
18	61	56	117	54	79	=	85	97	87	75	94
19	98	67	165	58	96	=	120	129	127	80	119
20	77	68	145	95	89	=	99	134	109	127	107
21	79	66	145	77	94	=	101	124	109	105	115
22	86	66	152	72	94	=	108	124	116	100	115
23	83	62	145	88	88	=	108	113	112	118	110
24	82	60	142	65	82	=	104	106	106	91	98
25	54	59	113	64	78	=	78	104	84	90	93
26	92	62	154	70	96	=	117	113	121	97	124
27	70	63	133	96	71	=	92	115	98	128	85
28	91	64	155	68	97	=	113	119	119	94	121
29	78	64	142	72	95	=	100	119	106	100	117
30	77	60	137	78	89	=	112	138	123	108	121
						=					
mean	77.03	62.9	139.93	75.23	87.53	=	99.43	115.93	106.1	101.62	106.03
S.D.	11.4	4.13	13.87	12.68	8.75	=	11.99	12.95	12.23	14.83	12.42
max.	98	68	165	98	105	=	120	138	127	128	130
min.	54	50	113	54	69	=	78	84	84	75	83

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