BEHAVIORAL, PHYSIOLOGICAL AND SUBJECTIVE CORRELATES OF SLEEP ONSET

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ABSTRACT

The standard criterion used to judge a lapse of wakefulness is the disruption of electroencephalogram (EEG) alpha activity. Recent comparisons of alpha loss with functional abilities are found only in studies of sleep deprivation and vigilance detection. Earlier investigations of sleep onset in normal subjects were incompletely reported and left several questions unanswered about the general applicability of sleep onset criteria.

Twenty young adults, including seven females, were recruited from the university community to participate in two 2-hour polygraph sessions studying selected behavioral, physiological and subjective correlates of sleep onset. Experiment one compared passive closure of a telegraph key with changes in EEG alpha and theta activity, electro-oculogram deflections, functions of thoracic and abdominal breathing components and subjective sleepiness ratings. Experiment two monitored the same physiological measures during a modified Wilkinson auditory vigilance test.

Subjects who produced abundant wakeful alpha activity had a mean latency of four sec from alpha loss to passive lapse (key closure) and a strong association of alpha level (present/absent) with key level (up/closed). In these subjects alpha loss was also associated with significantly greater incidence of slow eye movements, reduced abdominal breathing, a larger ratio of thoracic to abdominal (TA) excursions and a longer expiratory time with respect to inspiratory amplitude. All subjects, regardless of either alpha abundance or sex, showed significant and similar associations of behavioral lapse with all of the above changes. Significant sex differences in breathing amplitudes, and overall results with respiratory variables, were discussed with reference to the history of experimental techniques and findings.

Results from use of the Stanford Sleepiness Scale (SSS) were inconclusive because of faulty data collection. A replication attempt is indicated to confirm significantly sleepier mean ratings for both the key closed condition and the second half of the session, and an interaction in which SSS ratings were indistinguishably high during the second half.

In the second session the Wilkinson auditory vigilance test was administered at two levels of difficulty to different subjects. Misses were distinguished from correct detections (hits) by having a higher-numbered sleep stage during the four sec prior to tone delivery and both a lower abdominal amplitude and higher TA ratio in the preceding breath cycle. Only the subgroup that had the easy (400-500 msec) discrimination produced significantly more theta activity during the 1 sec prior to missed tones. Other variables failed to discriminate between hits and misses, and none of the variables distinguished hits from incorrect "detections" (false alarms), whether in component subgroups or for all subjects combined.

Analysis of differences in sensitivity, based on signal detection techniques and expressed as p(A), showed main effects for both task difficulty and sleep stage (W vs 1). An interaction also appeared in which the effect of stage upon sensitivity appeared only under the difficult discrimination (420-500 msec). The implications of this difference were discussed in terms of previous findings about rate of performance decrement across time as a function of signal intensity. Also discussed were the role of threshold changes as a limiting factor on easy discriminations and a generally reduced discriminatory capacity with the progress of sleep onset.

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DEDICATION

This thesis is dedicated to those who provided the data, especially fellow grad students in Psychology who generously made themselves available toward the end of a busy semester to transfer records of their functioning onto reels of tape and reams of polygraph paper.

ORIENTATION

Slumbering in the archives of imaginative escapades contrived by the first wave of TV variety shows is a program dealing with attempts to sleep in noisy surroundings. Arranged as a spectacle and containing several confoundings, the event was never intended as a serious test of whether the truly tired could fall asleep anywhere at any time. But it did highlight continuing questions about sleep: its definition, onset, characteristics and measurement.

The situation involved a contestant, a challenge and a prize. The specified performance: fall asleep on a bench by an appointed time during rush hour in Grand Central Station. No medication before then, no privacy, and on the night before, no sleep either. On arrival at the station the contestant was wired for polygraph recording, given a blanket and pillow and moulded into a position convenient for the lighting and camera crew.

At the appointed time, to no one's surprise, the polygrapher declared him to be awake. The contestant promptly agreed, yet just a moment before he certainly seemed to be sleeping. With closed eyes, sprawling posture and rhythmic breathing, he looked to me as I must have looked to my parents on the many occassions when I (possibly) deceived them into assuming that I was really asleep. So how did the polygrapher know that the contestant was awake? When I asked my mother she said that the polygrapher's machine could tell the difference. When I asked her how the machine knew I received that most perpetual of all possible replies to inquisitive kids: some day when I grew up I would find out all about it.

That was decades ago, and I'm still not sure. Behind every machine, however, are humans and humanly determined criteria of waking and sleeping that machines are designed to monitor. What, then, do we mean when we ask if someone is awake or sleeping?

Defining and Measuring Sleep

Questions about how sleep differs from waking were once popular but are now seldom asked and even less frequently answered. One could do worse than defer to Kleitman (1963), whose treatment of this issue is judged by many to be still incapable of further improvement. Kleitman had inherited an emphasis on physical immobility described by Pieron as an "internally determined, periodic and reversible suspension of activity". Kleitman added a functional dimension, stressing one's relative ability "to utilize the past and contribute to the future" (pg. 5). Kleitman thus emphasized the primacy of behavioral indicators of one's critical reactivity to the environment, which implies an analysis of sensory stimuli with respect to prior experience and an elaboration of appropriate responses.

Today sleep research is well developed, rapidly expanding and remarkably interdisciplinary. Sleep studies continue to provide useful and provocative information to those who work in biomedical and behavioral fields. And because sleep study techniques extend the range of inquiry into human and animal behavior - from wakefulness to sleep and dreaming - they are widely used by experimentalists and clinicians in a variety of investigations.

Sleep and its measurement are clearly inseparable subjects. In order to establish substantive reliability about the nature of sleep and procedural reliability about its measurement, sleep and its functional attributes had to be clearly described in terms of a monitoring technique that didn't seriously interfere with the process being measured but was strongly correlated with other more invasive methods of judging its presence and depth.

Early discoveries showed that sleep was not a unitary phenomenon, but a behavioral, physiological and experiential state that differed qualitatively and quantitatively across the night. Not surprisingly, considerable emphasis was placed on all-night recording sessions during which behavioral responsiveness and subjective reports were compared with physiological measures. More all-night sessions were then necessary to standardize recording techniques and scoring procedures, establish normative standards for human subjects and develop computer scoring methods to deal with the mass of data that accumulated in the course of a night.

As matters turned out, sleep research and electroencephalography grew up together, each contributing to the refinement and elaboration of the other. In contrast with other measures of sleep, the electroencephalogram (EEG) shows characteristic phasic as well as distinct tonic changes that lend themselves to an EEG staging system with which to describe the progress of sleep. The eventual discovery of regularly recurring rapid eye movement periods and their association with dream reports intensified research with the EEG. Supplemented with additional instrumentation, it is capable of continuous non-invasive measurement of sleep by stage criteria that can be judged with an overall scoring agreement of 90 percent (Karacan, 1978). As an added advantage, the EEG is especially reactive; stimuli in all sensory modalities produce stage changes and other transient events in the continuous EEG trace as well as in average evoked potential (AEP) data. In short, the EEG is a versatile and expressive instrument that enjoys widespread use in studies of the sleeping brain.

But underlying and ultimately validating these EEG criteria are still the kinds of functional behavioral relationships implicated by Kleitman. These behavioral phenomena are among the principal concerns here. This thesis examines the manner in which various behavioral measures, including spontaneous and discriminative motor responses and ratings of subjective state, correlate with the more primary electroencephalographic criteria, and inquires into the relations of both behavioral and EEG measures with activity in peripheral physiological systems.

In view of the central role of the EEG in the study of sleep and its pivotal position in evaluating other behavioral and physiological measures, the next section will review the basic properties of the EEG during wakefulness and sleep. Section three will then review the literature concerning relations among EEG, behavioral and peripheral physiological measures relevant to sleep onset.

THE EEG AND SLEEP

The overall appearance of an EEG tracing is characteristic of a subject in much the same way that a fingerprint is. For most purposes the EEG can be comprehensively described in terms of the frequencies of its component waves, their amplitude (voltage), the dominant frequency in the record and the proportion of record time occupied by the dominant frequency. It has long been known that these properties are relatively constant across time when the EEG is recorded under similar conditions from the same site (Davis and Davis, 1936). When at rest with closed eyes, 95 percent of adult humans have a dominant frequency in the 8 to 13 Hz (alpha) range (Brazier and Finesinger, 1943).

The term "high alpha" appears frequently in the literature. It refers to a record in which an alpha frequency is not only the dominant frequency, but also occupies a substantial percentage of the record. Using the quartile subdivisions of percent alpha time proposed by the above research groups, Table 1 shows the percentage of subjects in each group.

TABLE 1

Distribution of subjects according to alpha abundance

Research group	Percent of subjects in each quartile			
	< 25	25 to 50	50 to 75	> 75
Davis (100ss)	25	21	34	20
Brazier (500ss)	5	20	48	27

The alpha rhythm can be considered as an intermediate state of cortical activation (Oswald, 1962).* Such activities as problem solving or stimulation in any sensory modality will abruptly terminate (block) alpha (Mackworth, 1969), which is replaced by faster (beta) frequencies. But if a subject is in a drowsy, non-alpha condition, the return of activity in the alpha range accompanies partial alerting and signals of "awakening" (Anch et al, 1982).

As a relaxed subject with closed eyes descends into drowsiness, Davis et al (1937) observed the following changes.

1. Voltage of the alpha rhythm decreases and may undergo amplitude modulation.

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* Slow frequencies are generally related to a depressed cortex in sleep, anesthesia and coma, and faster frequencies correspond to activation produced by problem solving, anxiety and stimulant drugs (Gibbs and Gibbs, 1950).

2. Alpha activity may be disrupted and even completely interrupted for periods of 1 to 5 sec with replacement by low voltage mixed frequencies.
3. As cycles of alpha interruption continue, gaps become longer and theta is often observed between alpha trains.
4. Alpha abundance continues to be reduced and its frequency is 10 to 20 percent slower.

These observations were essentially corroborated by Kellaway et al (1967) who attempted to quantify the order in which various EEG and physiological changes occurred during sleep onset in a normative study of 200 aviators.

The EEG may be analyzed into a variety of sleep stages depending on the intent of the investigators. The most commonly used system for all-night recording is that designed by Rechtschaffen and Kales (1968), hereafter referred to as R&K, and containing 6 stages (W, 1, 2, 3, 4, and REM).

Relaxed wakefulness (stage W) with closed eyes is associated with more than 50 percent of an easily recognized alpha rhythm in a given EEG epoch of "high alpha" subjects. As sleep onset progresses the alpha is more frequently interrupted, decreases in amplitude, slows by 1 to 2 cps and is progressively eliminated. When alpha abundance falls below

50 percent in a given epoch (usually 30 sec) stage 1 is scored. Stage 2 corresponds to a low or medium voltage mixed frequency trace of predominately theta activity without organized alpha. Slow eye movements (SEM) appear and may continue for several minutes during the transition from alpha to low voltage mixed frequencies. Transient activity (vertex waves, sleep spindles and K complexes) is also observed during this time. The EEG pattern progressively becomes dominated by low frequency, high voltage waves (stages 3 and 4). A reversal of this sleep descent phenomenon is observed in transition to REM periods. Stages 4 and 3 EEG patterns revert to the lower voltage, faster frequencies of stages 2 and 1. REM periods are characterized by a stage 1 EEG, a sudden transition to low muscle tension and bursts of conjugate Rapid Eye Movements (by definition) that are usually accompanied by other phasic phenomena, like twitching of the extremities (Baldridge et al, 1965).

Electrophysiological measures have become nearly indispensible to the study of sleep. The EEG in particular has long since provided the standard criteria for sleep onset and sleep stages, assuming properties of an independent variable against which other physiological and subjective measures of sleep onset (SO) and sleep depth are compared. The comparison of these other non-EEG measures directly with behavioral criteria are rare. Magnussen's (1944) Studies on the Respiration During Sleep is the most recent example.

As a consequence the assessment of other than EEG measures of behaviorally- defined SO and sleep depth has been neglected. This is unfortunate for several reasons:

1. The use of alpha dynamics to determine sleep onset is difficult or impossible for "low alpha" subjects who comprise a quarter of the population.
2. An EEG determination may itself be difficult or impossible to obtain, especially with small children and other potentially uncooperative subjects.
3. Physiological measures other than EEG must be relied on to provide supplementary information. Even in high alpha subjects the significance of interruptions in alpha activity is ambiguous. At least two explanations are possible: It could be a lapse resulting from lowered alertness or a classical alpha block attributable to inceased alertness. Another measure, like SEM, is needed in order to make an appropriate assignment (Oswald, 1962).
4. For that matter, peripheral physiological measures may provide important information about the organism's functional state that isn't available in the EEG record. For example, slight sounds that show no

EEG reactivity have been observed to change respiratory patterns (Loomis et al, 1937).

Principal aims of the present research were therefore to replicate selected behavior-EEG studies, to determine the relations of peripheral physiological measures with EEG and behavioral variables and to explore the generality of their application as monitors of sleep onset.

REVIEW OF THE LITERATURE

Basic literature concerned with the transition between wakefulness and sleep is easily divided into two major areas of interest. Those studies which explore the relation of behavioral and peripheral physiological variables to EEG are of greatest relevance to this thesis and are discussed first under the heading Objective Measures. A separate section is devoted to a consideration of studies that extend the inquiry to subjective aspects of drowsiness and sleep onset. The chapter concludes with closer consideration of physiological variables considered for use as dependent measures in the proposed research.

OBJECTIVE MEASURES

Investigation into the behavioral and physiological correlates of sleep onset (SO) may be classified into two basic styles. Those explicitly conducted as studies of SO are discussed first, followed by those approached as studies of wakeful performance.

Sleep Onset Studies

To better organize the discussion, the various studies can be further subdivided according to whether subject behaviors are spontaneously emitted or cued by the experimenter.

Spontaneous Behaviors.

The Davis group (1938) asked subjects to signal, by pressing a rubber bulb, occassions when they noticed that they "had just drifted or floated off for a moment". The relation between EEG and float signals was reported to be clearest in the records of six subjects who showed a waking alpha rhythm 70 percent or more of the time. Of 156 float signals, 150 were recorded after a depression of the alpha waves which lasted from 1.5 to 40 seconds. Thirty nine similar alpha depressions weren't signalled. This isn't surprising, if only because - as already mentioned - the significance of an alpha interruption can't be determined without supplementary information.

The minimum duration of alpha loss that could be signalled was characteristic of the individual and tended to increase across the session. The alpha rhythm almost always reappeared before subjects signalled. The signal latency from reappearance of occipital alpha (0 to 5 sec) was also characteristic of the subject but showed no pattern across the session.

That some subjects were able to signal before alpha returned was discussed as an indicator that the awareness of floating may be possible in the absence of alpha. Alpha "may return because the subject rouses himself further by the act of signalling" (pg. 30).

The Blake group (1939) approached SO with a passive behavioral test. Eight subjects held a light spool between two fingers. In time the spool fell and the subject was aroused and asked whether or not s/he had been aware of dropping it. Regrettably, subject variance and group variance weren't reported, only the latency range of 0.5 to 25 sec after disappearance of the alpha rhythm. If the spool fell within 0.5 to 1.5 sec (usually the case), the subject was aware of its fall. But if the fall was delayed until 6.5 to 25 sec after alpha loss, the subject couldn't recall having dropped it. The authors conclude: "(Muscle) tone, therefore, diminishes soon after the alpha rhythm is lost, but consciousness does not disappear for some seconds more" (pg. 54).

It's unfortunate that within-subjects variability wasn't reported. Experimental error arising from inconsistent spool-holding and hand posture may have produced the larger latencies. Spool dropping, however, was only one element of an extensive investigation. In this and many early studies, observations of SO phenomena were largely serendipitous on route to an assessment of all-night sleep characteristics. As a result SO events weren't always systematically investigated.

A similar passive behavioral test was attempted during a pilot study this summer. Behavioral, physiological and subjective correlates of afternoon naps were studied with six volunteers. Among them were 4 high alpha subjects who showed a positive correlation between passive closure of a response key and decreased alpha, whether scored as a decrease in integrated alpha or as a visually detected alpha loss in the raw EEG record. A within-subjects ANOVA comparing alpha level (pg.resent/absent) with key level (up/closed) showed key closure significantly related to alpha loss $F(1,5) = 14$, $p < 0.01$, with 64 percent of shared variance.

In support of the Blake results, pilot high alpha subjects usually allowed the key to close between 0.5 and 1.5 sec after alpha loss (which was always judged in the occipital derivation). A comparison of latencies from alpha lapse to key depression with the alpha lapse times necessary to result in float reports wasn't attempted in this study.

Anliker (1963) conducted a time-estimation task, asking good alpha generators to tap a telegraph key every 3 sec during two 4 hour sessions. If subjects didn't tap for 90 sec they were alerted, asked to tap once, and then continue with the session. Graphs were recorded for each subject session. The mean integrated amplitude of dominant (awake) alpha frequency was cumulated on the ordinate against time on the abscissa. Each record was later analyzed and the data replotted so that elapsed time between responses could be compared with average slope of the corresponding alpha voltage during that interval. As subjects became drowsy, indicated by reduced amplitude means, their inter-response intervals increased.

Operant responding to avoid or escape a negative reinforcer has also been tried. Lindsley (1957) used subjects who were sleep deprived for 15 to 38 hours. By pressing a microswitch during all-night sessions, the subjects were able to control the volume of a tone that would otherwise increase to an intensity of 30 dB. A cumulative recorder was used to record response rates. Rates usually dropped after the first 30 min and fell to zero about 15 min after that. In a later session without the tone, subjects were instructed to press at the same rate. They continued to respond for the first half hour as before, but then stopped entirely. The extra 15 min can be considered as a numerical value for the difference between the effect of negative reinforcement and conditioned responding with instructions.

Kamiya (1961) attempted a replication with EEG control. He reported that an optimum rate of 30 presses per min was observed early in the session. But "as alpha is replaced by the low voltage pattern of drowsiness, the rate of pressing declines often to zero" (pg. 157).

Carskadon and Dement (1979) used a Serial Alteration task in which subjects tapped two microswitches "regularly and alternately at a steady pace". An illustration and caption of one record segment shows that after two nights of sleep deprivation the subject performed well during unbroken alpha trains, even in the presence of slow eye movements that are thought to precede or accompany drowsiness. "When the EEG changed to stage 1 sleep, however, the subject was unable to tap the switches" (pg. 501). (The meaning of "stage 1" isn't explicit, but seems to denote alpha-lapse periods.)

The main conclusion to draw from studies of spontaneous behaviors is that a behavioral lapse always coincides with or follows an alpha lapse. This is even the case in Anliker's study. Although his analysis indicates impoverished responding to be associated with decreased alpha voltage, the cumulative record shows that response lapses are accompanied by a drastic reduction in slope, indicating that only background alpha within the low voltage mixed frequencies is being integrated.

Unable to be determined from the available data is the extent to which experiences of floating coincide with lapses of spontaneous responding. A within-subjects study would be needed to test this possibility since the minimum duration of alpha lapse perceived as a float seems to vary across subjects.

Cued Behaviors.

The Loomis group (1937) presented 10-sec tones * to 6 subjects who were instructed to open their eyes at tone onset and close them at tone offset. Detailed results weren't

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* Stimulus intensity is seldom reported in early studies. This review reports stimulus parameters whenever they are known.

reported but rather: "If the subject is asked to respond, if awake, to a tone signal sent in every minute, we often observe the A state (containing alpha) to continue for 5 to 10 minutes after the subject has ceased to respond to the signal" (pg. 137). Under the Loomis scoring system, epochs containing any amount of alpha are scored as stage A. Prior to an entire epoch of non-alpha (Loomis stage B) are many periods of alpha-loss that could have coincided with tones. If these tones were of low intensity, responses might not have occurred during low-alpha epochs.

Fischgold and Schwartz (1960) delivered single flashes from a strobelight during various EEG conditions and required two button presses as a correct response. Data for 7 subjects asleep under Nembutal show that responding is still reasonably accurate during stage B (a low voltage mixed trace) and isn't disrupted until stage C (sleep spindles on a background of predominately theta activity). Responding from stage B seems inconsistent with the Loomis results and also with results from studies of spontaneous behaviors. But an inspection of the Fischgold data reveals that the positive responses from stage B were preceded by EEG activation responses to the stimulus. Their strobelight was probably a photic stimulator used routinely during clinical EEG recordings. These are designed to elicit EEG responses from drowsy or lightly sleeping subjects.

Magnussen (1944) used both spontaneous pressing and pressing to a verbal cue during long day sessions with shift workers and medical students. Much of his spirographic recording was done with apparatus that offered a slight resistance to expiration. Under these conditions very clear respiratory patterns were noticed. He discovered that disruption of cued rather than spontaneous pressing coincided better with transitions from regular respirations to those in which prolonged expirations or post-expiratory pauses occurred. These respiratory patterns would probably be associated with EEG-defined SO transitions under similar conditions. Respiration will be discussed further at the end of this chapter.

Simon and Emmons (1956) were the first investigators of sleep learning with EEG control. They used two behavioral measures: immediate response and subsequent recall of questions and answers. The authors compared EEG patterns that corresponded to detected vs undetected and recalled vs unrecalled material to measure the degree of learning at various R&K sleep stages.

Ninety-six questions of general information were given as a pretest to 21 experimental subjects who had a persistent wakeful alpha rhythm. A control group matched on means and variances of IQ, age and pretest scores also took the pretest. During the training session, experimental subjects were instructed to call out their name if they heard any of the 96 questions and answers being delivered 5 min. apart throughout the night.

Both unaided recall and multiple-choice posttests were administered to both groups. The percentage of items that were signalled as heard and the percentage correctly answered on the post-test were referred to sleep levels that prevailed during delivery of the answers. These levels were refined subdivisions of the Loomis stages. Predictably, the percentage of items reported heard during the session declined with the appearance of deeper sleep levels, as did the percentage of answers correctly recalled and correctly recognized on the exams. Answers presented during any alpha-containing level resulted in significantly higher percentage scores for the experimental group. Table 2 shows the percentage of correctly recalled items during alpha-containing levels:

TABLE 2

Percent recall as a function of alpha level

Level	Description	% correct
0	High amplitude continuous alpha rhythm at normal (waking) frequency.	80
A+	> 50% alpha rhythm that shows normal alpha blocking to stimuli, then returns.	75
A	< 50% alpha present but containing at least 3 cycles at normal frequency.	55
A-	Lower frequency alpha (or no alpha, but having alpha within 30 sec either before or after stimulus delivery).	30

Okuma et al (1966) investigated two behavioral measures with all-night subjects. Ten subjects received photic stimulation of 3 to 6 pulses at the rate of one per sec during various sleep stages and were asked to press a switch the corresponding number of times. They did so accurately from stages 1 to 3 which contain various amounts of alpha activity and correspond to R&K stages W and 1. But activation effects on the EEG were noted even in the deepest stages. Once a signal is given the subject is unavoidably alerted. Considering that a minimum of 3 flashes were presented in as many seconds it is likely that perception, and certainly response, begins from a much higher level of activation than that existing before the first flash. In support of this contention, incorrect responding from Okuma's deepest sleep stages were usually 1 or 2 less than the number of stimuli.

An auditory reaction time task was combined with photic stimulation in the sessions of 5 of these subjects. Subjects were asked to press a button whenever they heard a continuous 800 cps, 90 dB tone. Both reaction times and standard deviations increased from lighter to deeper sleep levels, showing that response latency to a single flash is a better discriminator of Okuma's stages.

In a more sensitive test, (Okuma, 1966) pairs of words were presented to subjects during various sleep stages. Fifty four percent of words presented during continuous alpha were identified the next morning, but only 18 percent of those words that were presented during theta activity while alpha was suppressed.

Liberson and Liberson (1966) used an RT procedure similar to that of Okuma. Twelve subjects pressed a key as rapidly as possible in response to an undefined signal that was given during alpha activity and at different times after the lapse of alpha activity. Subjects then reported their subjective experiences just before the signal was given. These experiences will be described elsewhere. Their response latencies were graphed as a function of time between alpha lapse and stimulus delivery. To compensate for the possible influence of session length on RT, the data included samples of alpha lapse both early and late during the session. A linear plot resulted in which RT latency was proportional to alpha lapse time.

Ozawa and Hirose (1979) studied simple reaction time (SRT) and choice reaction time (CRT) during both long-term memory (LTM) and short-term memory (STM tasks). 10 LTM subjects were first trained to respond selectively to the lowest and highest frequencies of four 70 dB tones, a CRT task. These tones were then presented in randomized interstimulus intervals (ISI) of 10, 20 or 30 seconds. Subjects were given directions for SRT (respond to all tones) and CRT (respond only to highest and lowest tones) on separate days.

Reaction times to non-alpha stimuli were longer than those to alpha stimuli, and reaction times in both EEG conditions were longer with CRT directions. Choice time (CRT - SRT) did not differ across EEG conditions, nor did the percentage of false alarms. Error percentages of misses were also non-significant across EEG conditions and response directions.

Another 10 subjects participated in the STM task. Experimental conditions were identical except for the directions to respond only to a tone of higher frequency than the one last heard. STM reaction times were longer than LTM times and the STM task differentiated sharply between alpha and stage 1 stimulus conditions. In addition to longer RT for stage 1 presentations, the error percentages of misses and false alarms were also significantly greater than for alpha presentations. ISI also exerted an influence; as ISI increased, reaction times were longer and errors increased. Finally, STM choice time, probably calculated as (STM CRT) - (LTM SRT), increased in stage 1. Further information wasn't available in this abstract.

Two comments about studies using cued behaviors are in order. First, sufficiently intense stimuli, especially when continued for several seconds, will almost invariably change even relatively deep sleep levels. Okuma's photic bursts and the Simon & Emmons verbal stimuli are in this category. EEG correlates of their behavioral measures will accordingly need to be considered by SO stages during, or degree of activation resulting from, the stimulus delivery. In addition to the activation effect, scoring by long epochs can also yield ambiguous results because in R&K stage 1, for example, both alpha lapses and reductions in alpha frequency occur, and theta abundance increases. To the extent that one might want to explore the EEG correlates of stimuli in different sensory modes and of various behavioral responses, scoring the precise kind and degree of EEG activity near stimulus onset is essential.

Conclusions and implications for the present study

A fair summary of objective behavioral correlates of sleep onset would note a consistency between findings of those studies employing cued behaviors and those using spontaneous behaviors. In both cases a change in performance is at least indicated by, if not dependent upon, a prior or simultaneous EEG alteration.

The present research included the use of two different behavioral variables in two experiments carried out in two successive sessions. For the first experiment it seemed appropriate to extend the pilot study, which used passive depression of a telegraph key as a spontaneous indicator of sleep onset. A cued behavior in the form of responding to a vigilance task was used in the second experiment. Both experiments attempted to correlate changes in behavior with prior or parallel physiological alterations and to investigate these as potential non-invasive indicators of sleep onset. The degree of association between alpha dynamics and the behavioral indicator, and the latency between alpha loss and behavioral lapse deserved special attention. Also important were appropriate tests to determine the suitability of the behavioral measure as an indicator of sleep onset for other than high alpha subjects.

Performance Studies

The term "performance studies" is intended to denote a paradigm that differs from that of sleep onset studies in set, setting and stimulation. SO subjects are literally couched in a sensorially diminished environment with instructions to drop off to sleep. Performance subjects are oriented to specific tasks, often in a psychophysical or simulated operational setting, typically encounter higher levels or rates of stimulation and are under at least an implicit demand to remain awake.

Performance research most relevant to this thesis are vigilance studies of signal detection and determinations of reaction time in both normal and sleep-deprived subjects. The experimenter measures the detection of a signal or speed of a response and relates these performance scores to one or more physiological or subjective correlates. Peformance decrements characteristically observed across session time are often correlated with specific EEG changes. In this section it is of interest to determine how well the inter-relations of behavioral and physiological variables in performance studies agree with those of SO studies. In the review to follow, performance studies of normal subjects are discussed first. Experimentation with sleep-deprived subjects and associated complexities are considered next.

Performance of Normal Subjects: Reaction time.

Reaction time studies measure behavioral response latency to easily detected or easily discriminated stimuli. Stimuli are usually grouped in blocks of about 10 min and several blocks, with intervening rest periods, comprise an experimental session. When mean reaction times are scored for each block, longer times are typically associated with the later blocks.

Much of the research on EEG correlates of RT decrements has been addressed to a pre-existing hypothesis that the cycle length of alpha rhythms is a basic temporal unit of event processing by the central nervous system (Lindsley, 1952). Surwillo (1961, 1963, 1964a) studied reaction times to tone stimuli of subjects ranging in age from 28 to 99 years. He attempted a two-part demonstration: that response latency differences between subjects who were maximally alert was a function of alpha frequency, and that within-subject data followed the same trend during individual sessions.

In each experiment the mean period of the EEG epoch between stimulus onset and response was calculated for each RT trial and compared with the associated response latency. Surwillo's 1961 results showed an inter-subject correlation of 0.81 between RT and alpha period (or -0.81 between RT and alpha frequency). In a 1963 replication, 93 of 99 intra-subject correlations were positive. When the mean of these Fisher-z transformed scores was reconverted to Pearson r, an average correlation of 0.31 between RT and alpha period resulted. Mean inter-subject data for the high-motivation block gave a correlation of 0.72.

In another study of 54 adults (Surwillo 1964a), individual subject means for both simple and choice reaction times and their associated EEG periods resulted in a correlation of 0.76 for both SRT and CRT tasks.

It was mentioned before that slowing of the alpha frequency develops with the progress of sleep onset. Results such as those of Surwillo are especially relevant to this thesis when evidence of drowsiness can be found during performance sessions. And it can. Surwillo (1963) presented and discussed portions of a representative record containing various levels of EEG activation, pointing out easily-observed systematic variations between response time and alpha frequency. Included was a response time over twice as long as the subject's mean to a stimulus that occurred about the time he reported dropping off to sleep. Morrell and Morrell (1965) have also emphasized recurrent EEG sleep episodes during performance studies.

The relation between alpha frequency and RT isn't unchallenged, however. The most serious criticism of Surwillo's procedure is the location he chose for sampling the EEG frequency. Positioned as it is immediately after stimulus onset, the EEG epoch can consist of various movement artifacts, an alpha-blocking response, alpha elicitation (common when alpha is already reduced in amplitude and frequency) and evoked potential components as well as the underlying EEG frequency itself. The influence of artifacts and alpha-blocking was largely eliminated by Surwillo's decision to score only well-defined waves. This removed one third of the total responses. Even then the EEG epoch seems too complexely determined to support a simple interpretation of results making use of it.

Boddy (1971) analyzed a 1-sec epoch immediately preceding signals for both visual and auditory trials. His experiment, which is essentially a replication attempt differing only in epoch location, failed to find a significant correlation between EEG period and RT of alert subjects in either inter- or intra-subject comparisons.

Other findings are also negative with respect to frequency shifts within the alpha range. Lansing et al (1957) found no correlation between visual RT and either the period or amplitude of alpha at stimulus delivery. Birrin (1965) also tested the influence of both alpha frequency and amplitude on RT at the onset of a tone. In his young male subjects "no relation was found between response speed and alpha frequency or voltage either within or between subjects". But he did find significantly longer response times to signals delivered during non-alpha compared with alpha times. Morrell (1966) established three pre-stimulus categories - alpha, low voltage and slow waves - and discovered correspondingly longer RT scores across these frequency bands. (Among Boddy's 1971 results is an extension of this correlation into the beta range. Reactions times became longer as frequency bands changed from beta to alpha and from alpha to low voltage and then slow waves.)

It's anyone's guess what Surwillo's results imply. In addition to questionable EEG sampling his study also differed in the extreme age range of subjects he used. Perhaps a factor that correlates imperfectly with age, like cerebrovascular disease, might produce the same experimental results. An extensive screening process would be necessary to exclude potential confoundings of age with health.

Performance of Normal Subjects: Detection studies.

Detection studies are concerned with responses to target stimuli. These may be presented alone at low intensity or in combination with one or more other stimuli as a discrimination task. Traditional vigilance tasks reported the probability of target detection across blocks of a session. More recently signal detection analysis has been used; subject performance is reported in statistical decision theory terms based on correct and incorrect responses about the presence or absence of a target. Such an analysis addresses the fact that variations in a subject's performance may reflect changes in sensitivity to the signal or changes in a subject's criterion of what constitutes a reportable signal (i.e., changes in the subject's willingness to report the presence of a target). Appropriate parameters for determining both sensitivity and criterion are based on percent hits (correct detections of a target signal) and percent false alarms (incorrect "detections" when in fact no target is present). Reaction time can be used as an additional measure of vigilance and is essential in unlimited-hold experiments where the signal remains until it is detected.

Results of vigilance studies generally show declining performance during the session accompanied by decreasing electrocortical activation. As representative examples, Davies and Krkovic (1965) found that both alpha abundance and target detection rate declined across blocks of trials during a 90 min auditory task. Groll's (1966) visual discrimination task showed that the number of correct detections and a subject's dominant alpha frequency reliably decreased during 90 min sessions. O'Hanlon and Beatty (1977) conducted a simulated radar monitoring with results reminiscent of the Morrell-Boddy correlation of frequency bands with response times. In the radar task, longer detection latencies were positively correlated with percent theta (0.44) and percent alpha (0.68)* and negatively with percent beta (-0.67), indicating decreased activation during the 120 min session.

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* In a visual task alpha is initially sparse and becomes more abundant as the session proceeds.

When EEG activity near individual signals is analyzed, theta activity predominates when the subject misses signals. This was first demonstrated qualitatively by Oswald (1962) and later confirmed. Groll (1966) examined 1-sec intervals before signals and found that an average frequency of 8.9 cps preceeded correct detections compared with 5.6 cps before misses. The Horvath group (1975) reported that misses were preceeded by higher levels of theta activity than were observed before correct detections (Davies & Parasuraman, 1982).

Daniel (1967) found the opposite - decreased theta production during missed signals. His task, however, was locating a specfic 3-digit sequence in pre-recorded numbers presented at a rate of 1 per sec. Comparatively more theta during correct detections in such a task could easily be attributed to perceptual processing or selective attention. A related study (Haslum & Gale, 1973) shows greater integrated theta abundance during auditory presentation of 4-digit sequences than during rest periods. And as reviewed by Schacter (1976), tasks involving concentration, problem solving and selective perception are associated with an increase of theta in frontal and central areas. Both of Daniels' bipolar electrode placements contained a vertex lead and therefore would have detected theta activity of that type as well as the more diffusely distributed theta of drowsiness. Daniels' observation of decreased theta in association with misses can therefore be understood as a lapse of serial concentration instead of a failure of EEG dynamics to account for performance decrements.

Further support for the similarity of relations among variables in both sleep onset and performance studies can be found in experimental investigations that alter the frequency or abundance of EEG activity. To be consistent with the correlational data obtained in both study types, increases in alpha abundance or frequency, and suppression of theta, should result in greater wakefulness (behavioral improvement), whereas less or slower alpha and enhancement of theta should yield a drowsier (behavioraly impaired) subject. This is generally the case.

Surwillo (1964b) attempted to modify alpha frequencies by entraining them with photic stimuli. Five of his 48 subjects were able to sustain frequency shifts between 6 and 15 Hz while participating in RT trials. They showed the expected positive correlation betwen RT and alpha period. Woodruff (1975) obtained similar results with biofeedback modification of alpha frequencies in 20 subjects. During slow-alpha trials subjects had longer reaction times than when they were performing fast-alpha trials. But Woodruff's rank-order correlation (0.40) of RT with EEG period across subjects was much smaller than Surwillo's, and her intra-subject correlations weren't significantly different from zero regardless of the session analyzed. Reflecting on these less than optimum results, Woodruff acknowledged the possibility that slow-alpha learning might be accompanied by more frequent alpha lapses and fast-alpha learning with fewer lapses. In short, a discontinuous EEG measure (alpha versus non-alpha or alpha versus theta), which correlates negatively with alpha frequency, could have explained her results.

Theta feedback conditions have been used in short and long sessions with both inexperienced volunteers and skilled observers. As reviewed by Davies and Parasuraman (1982), operant control of theta is generally effective when otherwise unregulated conditions show deterioration in performance. "When the observer is already fully alert, however, performance is unlikely to improve further as a result of theta suppression" (pg. 197). At the other end of the arousal continuum, attempts to improve performance with theta suppression (Morgon & Coates, 1975) or high alpha frequency enhancement (Hord et al, 1976) fail as sleep loss increases during sleep deprivation (SD) studies.

Other methods of experimentally modifying EEG frequencies might also be investigated. Obrist (1965) suggests sensory deprivation and prolonged body immobilization to reduce alpha frequency. The former is an attractive way to separate the alpha-lapse periods of drowsiness from the effect of frequency reduction. Subjects typically sleep during the initial hours of the session. Later, when they are presumably "refreshed", measurements show both decreases in performance and a progressive lowering of the dominant alpha frequency throughout the session , and even hours later during the recovery period (Heron, 1957).

In the absence of additional evidence the available findings tend to support the claim that performance of simple behavioral tasks correlates primarily with major shifts in the EEG frequency - beta to alpha and alpha to either alpha loss or replacement with theta.

There is yet another method that allows studying a subject's performance during the otherwise delicate, easily interrupted process of falling asleep. The method consists of making sleep onset less delicate (or drowsiness more durable), and is the topic to follow.

Sleep Deprivation Studies

A subject may be described as sleep deprived when s/he must exert extra effort to comply with socially-reinforced prolongations of wakefulness. Few attempts have been made to quantify this extra effort, although the various strategies are often extreme and no doubt contributed to Rechtschaffen's (1979) quip that the function of sleep is to avoid the symptoms of sleeplessness. The Naitoh alliance (1971) used a pedometer to show significantly higher walking mileage during SD, compared with baseline, as a strategy for remaining awake. Greater muscular activity has also correlated with improved performance during SD (Wilkinson et al, 1962).

The extra effort on one side and increasing sleepiness on the other tends to polarize the findings and interpretations of SD studies. Demands of task performance, the quality of interpersonal relationships and conditions of measurement (active or passive) often determine whether SD is conceptualized as a state of arousal or lassitude.

In terms of performance, however, SD subjects seem to show a general decline (Morgan et al, 1980). But early results of various behavioral experiments were inconsistent. Subjects would perform surprisingly well at times regardless of the nature of the task, extent of SD or time of day. Bills (1931) discovered the converse phenomenon - periods of non-responding that were later linked to EEG decreases in frequency and amplitude (Bjerner et al, 1949; Williams et al, 1959) and called microsleeps.

The Williams group predicted that the results of peformance tests would depend on whether tasks were paced by the experimenter or by the subject. In terms of a Lapse hypothesis popularized by Bjerner and Williams, when tasks were paced by the experimenter, some trials would occur during lapses, resulting in failures to respond. When tasks were paced by the subject, trials would not be attempted during lapses. Therefore error percentage would be higher for experimenter-paced tasks and rate of performance would be slower for subject-paced tasks. Their data and that of others support this hypothesis (Glenville et al, 1978).

In a study of vigilance and EEG changes under the influence of sleep loss or chlorpromazine, Mirsky and Cordon (1962) administered visual or auditory versions of the Continuous Performance Test * to each of 4 subjects. In agreement with previous studies, errors of omission were much more numerous than those of the unimpaired control session. For SD subjects more high voltage, slow EEG activity was present during omissions than during correct detections. The observed theta and delta activity and vertex waves supports the claim that behavioral lapses occur during microsleeps.

Concurrently recorded respiration and plethysmographic finger volume were even more sensitive to behavioral change. Behavioral omissions occurred when a combination of lower amplitude respirations with prolonged pauses and increases in both pulse volume (PV) and blood volume developed.

The Williams group (1962) followed with a study of auditory reaction time compared with EEG and PV. These investigators had previously observed fast as well as slow responding during alpha rhythm depressions in the same subjects. This isn't unusual considering that alpha depression may

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* This is a 10 min test during which 1 per sec presentations of random letters include a target stimulus to which the subject must respond.

represent either a decrease or an increase in alertness. An auditory CRT of 10 min was given to 7 subjects during baseline, SD and recovery days. EEG frequency was measured before the first second of each critical tone. Mean group correlations between CRT and EEG frequency were significantly higher during SD trials and distinctly bimodal trends were seen in the data. Subjects displaying an alpha rhythm of any frequency responded within half a second; during theta they missed the signal entirely. (The authors didn't report or discuss alpha-lapses unaccompanied by increased theta activity.)

Within-subjects comparisons depended on the development of a bimodal EEG distribution. Unless subjects exhibited a theta activity mode as well as an alpha rhythm mode, CRT omissions were minimal and intra-subject comparisons between EEG frequency and CRT were marginally significant, if at all. But although theta was necessary for omissions, it was not sufficient. During the first SD session one subject responded to 32 of 80 theta trials, yet stimulus intensity was described as "slightly above the subject's threshold". PV trend covaried significantly with CRT during SD trials, but to a lesser extent.

Naitoh (1975) reports the ability of a variety of autonomic measures as well as EEG to track the progress of a continuous manual task. Subjects performed cycles of pressing 9 numbered keys on a response panel in descending order and were instructed to respond to an infrequent click by pressing the tenth key. Results of these SD sessions were consistent with previous studies. EEG alpha prevailed during the pressing task on SD days and was replaced by a low voltage trace with occasional vertex waves a fraction of a second before behavioral lapse. Also observed during nonpressing times were lowered heart rate, increased pulse volume amplitude and slower, shallower breathing.

Of special interest to the proposed research is that these same changes could be observed during baseline as well as SD sessions. On the basis of this study a subject during SD differs from his or her undeprived condition only in the degree and lability of arousal level, which is indicated by the relative magnitude of EEG changes and by the greater rapidity with which behavioral sleep onset develops. One could predict that patterns of similar behavioral and peripheral physiological measures that occur during SD would also be found under normal conditions provided that the subject were well adapted and the session prolonged. The Mirsky and Williams experiments couldn't address this issue because of the short (10 min) testing sessions they used.

Conclusions and implications for the present study

This much, at most, might be concluded from the results of performance studies: Just as the performance of SD subjects is a function of condition change (normal vs sleep-deprived), so the performance of normal subjects is a function of stage change (alpha vs alpha-lapse or theta). The comparison is compelling and invites analysis, with normal subjects, of the relation between performance decrements and signs of lowered alertness in both EEG and peripheral physiological measures.

Data from vigilance tasks show characteristic performance decrements across long sessions. Several studies show errors of omission correlated with brief periods of reduced EEG activation (Davies & Krkovic, 1965; Groll, 1966; Horvath et al, 1975), and others demonstrate performance decrements under sleep deprivation (Beatty et al, 1977; Deaton et al, 1971; Wilkenson, 1968). There is even evidence that experimental alteration of EEG variables affects performance in the expected direction (Woodruff, 1975; Morgan & Coates, 1975). But although performance decrements over time and those attributed to sleep deprivation have been documented with signal detection analyses, the EEG - performance relation has not. Thus we do not know to what extent correlations between EEG and vigilance performance actually reflect an EEG influence on the subject's discriminatory capacity (sensitivity). Nor have the perpiheral physiological variables of experiment one been studied as possible correlates of performance decrements in unimpaired subjects. Experiment two was allocated to these determinations with a suitably modified version of the Wilkinson auditory vigilance test.

The experimental intent behind experiment two consisted of three parts:

1. A replication attempt of previous findings that detection errors were associated with indications of reduced wakefulness in the EEG.
2. Further tests to determine whether detection errors were associated with indications of reduced wakefulness in other dependent measures.
3. A signal detection analysis to test for differential responding at selected EEG levels of wakefulness.

From the section on Performance Studies it was concluded that performance decrements were related to stage shifts (alpha vs alpha-lapse or theta) of normal subjects and to condition shifts (normal vs sleep-deprived) of SD subjects. Because the general EEG appearance of an SD subject approximates that seen during normal sleep onset (Horne, 1978), it is a safe conclusion that decrements in simple performance tasks can be related to a decreased activation level of the EEG in terms of shifts to lower frequency bands. Whether task decrements of both normal and sleep-deprived subjects occur for the same reason is still unknown. Good evidence exists that SD subjects are both less sensitive and less cautious than unimpaired subjects during vigilance tasks (Glenville et al, 1978; Polzells, 1978).

The Wilkinson auditory vigilance test was selected because it showed reliable performance decrements across time and produced lower sensitivity scores as a function of increasing degree of sleep deprivation (Wilkinson, 1969; Deaton et al, 1971). It was modified by reducing the event rate and making a compensatory increase in the ratio of target to non-target events in order to generate enough false alarms to allow determinations of both sensitivity and criterion level across sleep onset levels. These were intended to be alpha without SEM activity, alpha with SEM and alpha loss periods.

SUBJECTIVE MEASURES

As already mentioned, Davis et al (1938) reported that subjects described their "float" episodes as distinct from real sleep. These subjects were equally unanimous in reporting floats as depressions of awareness (in terms of a diminution of sensory stimuli and forgetting of the experimental setting).

Such observations were also reported by Dement and Kleitman (1957) who elicited SO reports from 10 subjects during stage 1. Subjects invariably stated that "they were not asleep and that mental imagery was not as organized or real as that occurring in dreams". A variety of images and sensations were described that might be called hypnogogic such as floating, drifting, flashing lights, etc..

Lehman et al (1979) compared alpha with non-alpha experiences. Their 30 subjects were asked to report from two conditions: when 4 sec of low voltage was followed by 20 sec of alpha, and when 20 sec of alpha was replaced by a sustained low voltage trace for 10 sec. The former (alpha) condition was associated with significantly more non-visual thinking, and the latter with visual experiences. A comparison of the first two and the last two reports of the session showed a similar agreement of report type with EEG category, indicating no apparent interaction of mentation type with session length.

Zilberg's (1977) dissertation abstract reported a positive correlation of sleep stage (W versus 1) with mental contents categorized in terms of a Primary Process Scale. (He also reported stable intra-subject shifts in hemispheric laterality from stage W to stage 1, but doesn't specify the direction.)

Liberson and Liberson (1966) elicited mentation reports during alpha and at various times after alpha-lapse. Decreasing reality-orientation accompanied increasing time between alpha-lapse and report cue.

Foulkes and Vogel (1965) chose to explore subjective factors in several distinct physiological stages of sleep onset. They conducted extensive interviews during awakenings from their 4 SO levels that ended with an estimate of the subject's degree of wakefulness (on a descending 5-point scale of 0 to 4). Reports corresponding to these levels were collected from 9 subjects each of whom contributed 6 awakening reports during each of 4 sessions. A comparison of the 3 early with the 3 late awakening reports shows substantially the same depth rating for a given level, reflecting little if any time-of-night effect. Data Table 3 shows subjective depth at different SO levels.

TABLE 3

Subjective sleep depth as a function of stage

SO level	Depth estimate	
	Medians of subject means	range of subject means
alpha (no SEM)	.80	.2 - 2.1
alpha SEM	1.50	.5 - 2.7
stage 1	2.00	1.3 - 2.7
stage 2	2.25	1.7 - 2.7

Notice the extensive range in both alpha levels.

This decrease in wakefulness with deeper levels was also observed in individual subjects. But individual graphs of depth versus level had varying slopes as well as different absolute values. One could conclude from this that subjective estimates of sleepiness are less sensitive indicators of SO level than the EEG. But it is also possible that with sufficient practise use of an expanded and well-defined interval scale might correlate better with EEG criteria and perhaps show a strong relation with one or more behavioral measures as well.

The Stanford Sleepines Scale (SSS) consists of 7 numbered statements arranged in equal-appearing intervals of increasing sleepiness. Hoddes et al (1975) compared the SSS ratings and performance measures from baseline to SD conditions. SSS correlations with the three performance measures were:

Wilkinson Vigilance test 0.70
Wilkinson Addition test 0.67
Williams Word Memory test ... 0.74

All of these were non-significant with a group of 5 subjects, but baseline SSS ratings were significantly lower (more wakeful) than corresponding SD ratings throughout the day.

Carskadon and Dement (1979) also studied behavioral and subjective correlates of SD. Performance on the Wilkinson addition test declined significantly during SD and scores on their serial alteration test decreased while the SSS, Mood scale and their Sleep Latency test depicted a sleepy and inert drudge who could fall asleep in less than 2 minutes after a day of SD.

Glenville et al (1978) used the SSS with a 1 hour Wilkinson Vigilance test and 4 short performance tests to study the effect of one day of SD. As might be predicted from the previous section, Vigilance fell during SD. Number of hits decreased by 50 percent and signal detection analysis showed decreases in both sensitivity and criterion level.* Of the four 10 min tasks, SRT and CRT data showed slower responding and more response times greater than 1 sec, but tests of short term memory and handwriting were unchanged. SSS scores corresponding in time to all tests increased significantly from baseline values of about 3 to SD values of about 5. EEG wasn't recorded.

Conclusions and implications for the present study

The case for subjective changes during sleep onset is not as clear as for the objective data. The SSS seems able to distinguish between sleep-deprived and unimpaired subjects but its "short-term" within-subjects use in normal sleep onset has not been reported. Of those subjective measures

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* Subjects were less sensitive to differences and less cautious in their judgement.

that have been tested in this way, changes do not appear to vary with the length of experimental sessions (Foulkes and Vogel, 1965; Liberson and Liberson, 1966; Lehman et al, 1979).

The Stanford Sleepiness Scale was used in experiment one to test the hypothesis that subjective sleepiness varies with the objective sleep onset level and not simply with time across the session. The intended sleep onset levels were the same as those planned for the Wilkinson test; alpha without SEM, alpha with SEM and alpha loss.

PERIPHERAL PHYSIOLOGICAL VARIABLES

The following physiological variables are proposed as important adjuncts to EEG monitoring, as possible substitute measures when EEG is inappropriate, and as variables that offer functionally significant information both during and on both sides of EEG- and behaviorally-defined SO transitions.

Slow Eye Movement

Sleep onset offers a unique opportunity for the eyes to be literally extravagant. They wander in ways that are impossible to duplicate intentionally, undergoing slow excursions that are invariably seen during the administration of anesthetics (Burford, 1941).

While awake with closed eyes, a subject is capable of saccadic fixational movements. These are rapid conjugate motions of low amplitude (small arc) sometimes observed when the eyes are closed (Shimazono et al, 1965). As confirmed by Aserinsky and Kleitman, 1953, 1955), an entirely different pattern emerges concurrently with the onset of sleep - slower and higher- amplitude excursions that are deflected in an oblique plane, sometimes asynchronously. Dunwoody and Edmonston (1974) define SEMs as slow horizontal eye movements having a frequency below 0.8 Hz; Kellaway et al (1967) estimate SEM frequency at 0.25 cps.

As reviewed by Dement (1964), SEM activity generally precedes the earliest alpha loss events and is usually terminated by an alerting stimulus during this time. If sleep onset continues unhindered, SEM activity usually continues through stage 1, becoming slower and less abundant, and quiescent for long periods during stages 3 and 4 sleep. At such times when sleep is well established, a body movement or a stimulus of sub-awakening intensity will initiate SEMs. In deep anesthesia SEM activity is entirely absent (Burford, 1941).

In a recent study by Hori (1982), 19 of his 20 subjects exhibited SEMs a few minutes before stage 1 onset. This temporal separation was advantageous to a study of subjective correlates of SO by Foulkes and Vogel (1965), allowing them to establish a category between alert wakefulness and stage 1. This intermediate category, called alpha-SEM, differed in subjective wakefulness from both higher and lower categories and served to identify a continuum on other subjective rating scales as well.

So far, the behavioral measures used during SO have not been directed to distinguishing SEM conditions from other sleep onset patterns. In fact SEM is rarely recorded during behavioral studies.

In a summer pilot study the extent of SEM activity was compared during alpha and alpha-loss times and also times scored as up versus closed with respect to a response key that indicated a passive behavioral lapse. SEM activity monitored during epochs containing SO transitions (gaps in alpha trains) was significantly more abundant during alpha loss and behavioral lapse in the 6 subjects studied, with shared variance of 35 and 27 percent respectively.

This served only to demonstrate that SEM might be a useful substitute predictor of SO for low alpha subjects. To assess the functional significance of SEM a more sensitive measure is needed that might discriminate between SEM and non-SEM conditions during stage W as well as distinguish between alpha-SEM and alpha-loss phases of SO.

Dement's (1965) subjects responded "awake and thinking" during sustained alpha without SEM, but reported visual experiences and diminished awareness of the environment when SEM accompanied continuous alpha. This content was included in subjective float reports described by the Davis group (1938) as "depressed awareness of immediate external stimuli or of self- consciousness, awareness of the experimental situation, etc.".

Little help in sorting out the contribution of SEM to the subjective aspects of SO is available in the rest of the literature. Mentation reports were made either by stage

(Dement and Kleitman, 1957) or by elapsed time after alpha loss (Liberson and Liberson, 1966).

The use of SEM to monitor performance of either sleep-deprived or unimpaired subjects has not been reported. Perhaps SEM activity has no qualitative behavioral significance. Kellaway (1967) included SEM activity among the first indicators of drowsiness only because it was so frequently observed during the transition form wakefulness to sleep. Aside from some modest utility in predicting a change in behavior, SEM onset may indicate little beyond the development of relaxation to the point where the level of cortical activity is too low to maintain equivalent (balancing) innervation of the extraocular muscles. This was proposed by Aserinsky and Kleitman (1955) to explain the slow excursions "which appeared to be passive drifts entirely lacking in fixation and frequently binocularly discoordinate" (pg. 8).

Reports of Dement's subjects nevertheless suggest that performance decrements could be found during SEM in a suitably designed vigilance study, and his (1965) contention that SEM activity is inappropriate to visual experiences was supported by Rechtschaffen and Foulkes (1965). Two of their subjects who fell asleep with taped-open eyes were presented with illuminated objects during alpha-SEM periods. When awakened 2 to 20 sec later they had no recall for the stimulus object. It is still an open question whether this sensory impairment is general or restricted to vision.* If general, then it's conceivable that a sensitive auditory vigilance task could distinguish awake alpha-SEM from alpha without SEM. These were two of the three sleep onset levels planned for obtaining SSS reports in experiment one and for determining performance parameters on the Wilkinson auditory vigilance test in experiment two.

Respiration

If poetic descriptors were ever admissable in research reports, studies of respiration would merit them most. The history of inquiries into the relation of respiration and sleep reads like a romance. In physiology it is a tantalizing parallelism and intimate association between cortex and brainstem; in methodology it is an intrigue of delicate reactivity and refined technology; and in results it is a tangle of tales from the jaded and jubilant.

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* The idea of attempting to determine whether different sensory modes were "differentially obtunded" during SO hasn't been proposed since 1830.

The following summary of respiratory control is a useful oversimplification to help clarify the rationale of various research strategies. It is based on reviews by Phillipson (1978) and Sullivan (1980).

Respiratory control can be exercised by either of two major control systems. The automatic (metabolic) center in the brainstem responds homeostatically to central and peripheral chemoreceptors that are sensitive to carbon dioxide, acidity and oxygen and also to chest wall mechanoreceptors. The voluntary (behavioral) system, mainly of cortical origin, allows the breathing apparatus to be used for non-respiratory purposes.

Unless respiratory pathology or exhorbitant oxygen demand supervenes, it is changes in carbon dioxide and acidity (via the intermediate carbonic acid) that determine basic respiratory control by the automatic system.* Increased carbon dioxide and acidity stimulate the respiratory center to produce deeper and/or faster breathing. The increased ventilation and resulting elimination of excess carbon dioxide reduces the source of stimulation and the activity level falls.

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* As expressed by one respiratory poet, "Thus carbonic acid spreads its protective wings over the oxygen needs of the body".

When the organism is awake and at rest, yet another respiratory stimulus exists quite apart from operation of the voluntary system. This wakefulness stimulus, as it is called (which appears to be of reticular origin), acts directly on spinal respiratory neurons and also increases the activity level of the automatic control center. As a result, carbon dioxide is kept below the concentration that would activate an increase in ventilation.* Its effectiveness is most dramatically demonstrated by patients who, because of defective automatic control, stop breathing when they fall asleep (Sullivan, 1980).

When the organism falls asleep two main changes occur. First, loss of the wakefulness stimulus results in decreased ventilation and a subsequent rise in carbon dioxide concentration. Second, the sensitivity of the automatic control center to carbon dioxide is reduced so that greater concentrations are tolerated without compensatory activity.

An increase in the partial pressure of alveolar carbon dioxide that closely tracks EEG changes at sleep onset has recently been confirmed. Naifeh and Kamiya (1981) used min-

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Expanding on the previous footnote, the wakefulness stimulus has even bigger wings.

imally intrusive measuring devices in a design that controlled for possible effects of habituation to the laboratory setting and incorporated an awake but supine control group. Using individual breaths as the scoring epoch, and sampling carbon dioxide at the end of each expiration, they observed that transient stage 1 activity was always preceeded by or simultaneous with carbon dioxide elevations. Alveolar carbon dioxide was also significantly higher in a within-subjects comparison of stage 1 compared with stage W subject means.

In this same experiment Naifeh and Kamiya obtained similar results measuring relative amplitudes of thoracic (T) and abdominal (A) excursions. Both higher TA ratios and increased alveolar carbon dioxide were associated with breath epochs scored as stage 1. Concern with the relative thoracic and abdominal contributions to respiration is a dominant tributary in the cascade of controversy about changes in the form of breathing that are reported to occur with reduced ventilation during sleep.

Changes in the amplitude ratio have been reported since Mosso's (1878) experiments with human and animal subjects. His results were supported by Shepard (1914); both found larger abdominal contributions to ventilation during wakefulness and thoracic preponderance during sleep.

In contrast, Pieron (1913) found the opposite in 6 of his 10 subjects, all of whom were female.*

Reed and Kleitman (1926) did observe a reduction in abdominal movement during sleep, but lower thoracic excursions were often observed as well. An increase in the TA ratio was observed in only half of the 30 trials on 9 subjects.

Magnussen (1944) made pneumograph recordings of 6 subjects during spirograph and non-spirogrpah sessions. He found frequent episodes of decreased breathing amplitude at SO but no lasting changes in the proportion between the two kinds of respiratory movement.

Goldie and Green (1961) presented examples of TA alteration associated with EEG changes in arousal, but no details about subjects or procedures.

A decade later Timmons et al (1972) also reported this association during explicitly stated conditions. During wakefulness 10 of their 11 subjects showed abdominal-dominant respiration. In 93 percent of SO transitions resulting in alpha loss for at least 10 sec a reduction of abdominal amplitude was observed. With resumption of alpha for 5 sec.

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* Is it possible that women of that time, "assumed to be inclined to thoracic respiration" (pg.ossibly learned for cosmetic reasons with the help of tight corsets) reverted to uninhibited breathing with the development of sleep?

abdominal enhancement appeared in 81 percent of the traces.

Liberson and Liberson (1966) noticed a general phasic reduction in ventilation during SO. They reported that high amplitude regular respirations decreased during the first few seconds after alpha loss.

The pilot study referred to earlier found a close association between SO transitions and abdominal amplitude. Several epochs showing alpha trains with gaps were selected for each subject record. Mean amplitudes were compared, in a within-subjects analysis, for alpha and alpha-loss breaths. A significant difference was found with 27 percent of shared variance. When abdominal amplitude was compared with up versus closed times of a telegraph key designed to show passive behavioral lapse, 39 percent of the variance was shared between key and abdominal amplitude.

Expiratory time (ET) has also been studied as a correlate of sleep onset. This is reasonable considering that the established reduction in ventilation during sleep onset (Bulow, 1963) can manifest as diminished volume or decreased rate. A post-expiratory pause associated with disruptions in cued behavior was seen by Magnussen (1944) during spirograph recordng and by Mirsky and Cordon (1962) when their sleep-deprived subjects made errors of omission.

During the pilot study expiratory time was found to be non-significantly longer during alpha loss epochs. A derivative variable was then created using abdominal amplitude (A) with its corresponding expiratory time (ET). When the quotient A/ET was calculated during SO transition epochs, smaller A/ET shared 50 and 58 percent of variance with alpha-lapse and key-lapse respectively, achieving significance at the 0.01 level with only 6 subjects. Tabachnik et al (1981) recorded similar measures during the sleep of adolescents. Their ET was also non-significantly longer (stage 2 compared with stage W). But inspiratory time (IT) did achieve significance. It was used with tidal volume (V) in the same way as above to yield a significantly smaller V/IT in stage 2 compared with stage W.

Thoracic amplitude changes weren't closely related to SO transitions in either the Timmons or pilot studies. But as SO progressed in both of them "a tonic decrease in abdominal breath cycle amplitude and concomitant increase in thoracic amplitude occurred" (Timmons et al, 1972, pg. 173). Following an initial abdominal reduction, 9 of the Timmons subjects showed equality of thoracic and abdominal excursions by the first 30 sec epoch of stage 1. TA results of Naifeh and Kamiya are consistent with those of Timmons. Sampling 1 to 5 min epochs containing SO transitions, Naifeh and Kamiya found both intra-subject increases in TA ratio during EEG-defined SO as well as a significantly higher group TA ratio in stage 1 compared with stage W. The present author's pilot results were marginal, partly because of high variability. Epochs that were scored also contained shorter durations of alpha loss than those of Naifeh and Kamiya. Maximum TA ratios didn't occur in the pilot study until alpha-loss had continued for several breaths. In terms of ventilatory responses, decreased abdominal amplitude relates to loss of the wakefulness stimulus whereas TA ratio change may result from eventual responses of the automatic system to increased carbon dioxide tension with differential innervation of the breathing musculature.

Sex differences may account for some experimental discrepancies in TA ratio change. The Timmons group, who had one female among their 11 subjects, found larger TA ratios after SO whereas Pieron found the opposite in 6 of his 10 subjects - all female. Four of eight subjects used by Naifeh and Kamiya were female. Compared with resting awake measurments of male subjects, their alveolar carbon dioxide tension was lower and their TA ratio higher. An analysis of respiratory measures by sex is definitely indicated. And although measures used in the present research can make no specific determination of differential sensitivity or neural control, well-documented results may contribute to the more thorough examination of sex differences urged by Sullivan "particularly in view of male predominance of some disorders of breathing, and the known respiratory stimulative effects of progesterone" (1980, pg. 235).

The available literature suggests that respiratory changes could be used to score SO transitions, at least in those subjects who exhibit them prominently. A reduction in ventilation over 1-3 breath cycles and an increase in expiratory time, both most easily seen in the abdominal trace, accompanies alpha-loss periods. If the alpha loss is prolonged, thoracic amplitude increases, giving rise to a significantly larger TA ratio. A large abdominal excursion and prompt resumption of waking respiratory values accompany the return of alpha. The present research used mercury strain gauges and procedures adopted by previous investigators. But it examined the change of respiratory variables relative to changes in two behavioral variables - passive lapse and vigilance performance - as well as in relation to EEG dynamics.

Muscle Activity

The electromyogram (EMG) has found some application in performance studies to indicate and warn against lowered vigilance (Kennedy and Travis, 1941). It has also been used to monitor decreases in muscle tonus attained as a result of various relaxation methods (Sime and DeGood, 1977).

In sleep studies EMG is most useful in defining one of the parameters of a REM period - greatly reduced muscle tonus. The first attempt to measure decreasing activity in relaxed muscles as one passes from wakefulness into sleep was first reported by Max (1935). His results with EMG from arm and leg placements were replicated on throat sites by Berger (1961) and on both throat and chin locations by Hishikowa (1965). All investigators reported that tonic EMG activity decreased gradually from relaxed wakefulness through sleep onset and into deeper sleep, showing its greatest change at REM onset. The gradual change in activity of passive musculature makes it an unlikely marker for sleep onset. EMG was not used in this thesis research.

EXPERIMENT ONE

Among the spontaneous behaviors monitored during SO, only one passive measure has been used - the spool drop of Blake et al (1939). As reported previously, both the Blake group and the summer pilot results show a behavioral lapse shortly after alpha loss. Experiment one included a replication attempt and addressed many related questions:

1. How do alpha dynamics and peripheral physiological measures vary with a passive behavioral indicator of sleep onset (Analysis I, below)?
2. Do the correlations between physiological and behavioral measures referred to in analysis I vary appreciably with alpha abundance, or can they be generalized to all young adults regardless of alpha abundance (Analysis II)?
3. How do various dependent measures vary with alpha level (Analysis III)?
4. Is there a low variability of latency between alpha loss and behavioral lapse within and between subjects (Analysis IV)?

5. Do physiological (especially respiratory) changes associated with alpha level vary with sex or are they non-specific measures of sleep onset for all young adults (Analysis V)?
6. How does a subjective measure of sleepiness vary with session time and objective indicators of sleep onset level (Analysis VI)?

A consideration of these questions was included in the first of two 90-min polygraph sessions. In order to combine behavioral, subjective and physiological measures in the same experimental session a minimally stimulating behavioral indicator was designed in an attempt to reduce the impact of sensory feedback on subjective judgements of sleepiness.

METHOD

Subjects

Volunteers for two 2-hour polygraph recording sessions were obtained in response to various forms of advertising on the university campus and as a result of referrals by former subjects. Applicants were young adults ranging in age between 19 and 39 years. By self report they were in good health and had neither sleeping difficulties nor any hearing impairment.

Volunteers were encouraged to select their own experimental days - preferably adjacent dates - and a starting time likely to favor rapid sleep onset. Their chosen starting time was constant for both days. Because of the possibility that variations in hormonal level might be a factor in the variability of data obtained from female subjects, females were scheduled during the luteal phase when their hormonal levels were likely to be high and stable. (For non-cycling females, all pregnacy and "pill days" are luteal days.)

Apparatus and Measurment Instruments

All physiological measures were recorded by a Grass Model VII polygraph aided by three auxiliary devices:

1. A General Radio Company 1952 Universal Filter used as a bandpass filter prior to integrating frequencies in both the theta (4-7 Hz) and alpha (8-13 Hz) ranges.
2. A Grass model 7P10B polygraph integrator to provide 1-sec integration values of filtered theta and alpha activity.
3. A Model 4 Vetter FM recording adapter to transform analog polygraph data to FM for storage on magnetic tape, then back again for filtering and processing of different frequency bands.

Continuous output from a Marietta model 24-21 white noise generator was delivered through a speaker located on the floor about 1 meter below the subject's head. Sound intensity measured from that distance was 48 dB (A).

Passive behavioral lapse was monitored with a modified telegraph key. Spring tension was adjusted so that a very slight upward (extensor) tension was needed to avoid switch contact by the third finger of the preferred hand. Loss of muscle tension at sleep onset initiated contact, generating a continuous signal on both the original chart and FM tape until the finger was lifted.

Monopolar EEG was obtained from C-Z and O-2 sites* of the Jasper 10-20 system (Spehlmann, 1981). These active electrodes were referenced to linked ears, and a mastoid ground was used. EEG was monitored by gold cup electrodes filled with Beckmann electrode adhesive paste and held in place with tape or an elastic bandage over gauze pads. Electrode pairs were checked with an impedence meter to insure a resistance below 10,000 ohms. Both of the EEG preamplifiers were externally calibrated with a wave generator at a sensitivity of 25 microvolts per cm, peak to peak. A time constant of 1 sec and low pass filtering at 75 cps was used.

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* These correspond to the vertex (C-Z) and the right occipital region (O-2).

The 8-13 Hz output from bandpass filtering of O-2 was cumulatively integrated over 1 sec reset intervals. To prepare for subsequent analyses, raw C-Z, O-2 alpha and integrated O-2 alpha were FM-transformed and recorded on tape.

A bipolar EOG was recorded from the outer canthus of each eye at a sensitivity of 0.2 millivolts per cm. DC output of eye movement activity allowed SEM to be distinguished from the comparatively shorter and more rapid saccadic motions that sometimes occur with closed eyes during the early part of a recording session. EOG was monitored with miniature silver/silver chloride electrodes filled with Beckman electrode electrolyte and secured with electrode collars.

Thoracic and abdominal breathing components were recorded with Parks Electronics mercury thread strain gauges about 25 cm long. These were powered by a 6 volt DC generator and balanced into the DC preamplifiers with wheatstone bridges using adjustable potentiometers. In use, expansion of the chest wall and abdomen stretches the silastic tube, and the resulting decrease in the diameter of the mercury capillary increases its resistance to an applied current. Prior to recording sessions both gauges were calibrated so that equal stretch produced equal voltage change at the polygraph amplifiers. (Linearity of performance was also observed over their normal operating range.) Gauges were then stretched to 30 cm and taped in place at the level of the third intercostal space and at the level of the navel.

The measuring instrument used to provide subjective depth reports of alertness-sleepiness level was the Stanford Sleepiness Scale (SSS) reproduced below.

1. Feeling active and vital; alert; wide awake.
2. Functioning at a high level, but not at peak; able to concentrate.
3. Relaxed; awake; not at full alertness; responsive.
4. A little foggy; not at peak; let down.
5. Fogginess; beginning to lose interest in remaining awake; slowed down.
6. Sleepiness; prefer to be lying down; fighting sleep; woozy.
7. Almost in reverie; sleep onset soon; lost struggle to remain awake.

Procedure

A day or two before the first session prospective subjects were given an instruction sheet. This contained a request to avoid stimulants for at least six hours before scheduled sessions. It also contained a copy of the Stanford Sleepiness Scale and a rating sheet. Volunteers were asked to rate and record several SSS values before their first session and to return the rating sheet when they arrived for experiment one.

As part of a general orientation to recording sessions, and immediately before they began, subjects were reminded that their individual contributions were valuable aids in clarifying and extending the SO literature. Special emphasis was placed on the need for an accurate record of events and patterns as they actually transpired. In particular, it was stressed that the telegraph key closure was to be a passive lapse; it was not to be emitted in response to a perceived cue. Subjective reports were, in like manner, to correspond to perceived sensations, not to anyone's preconceptions or expectations.

While subjects were prepared for polygraph recording the purpose of electrodes and transducers was explained. After EEG and EOG electrodes were applied the subject assumed a comfortable supine position on the bed in the electrically-shielded subject chamber. Respiration gauges were then taped in place and subjects were cautioned not to move their arms or roll over during the session, as such movements tended to disrupt the recording and necessitate recalibration of the circuitry. Lights were then dimmed and eye movements were calibrated by having the subject fixate alternately on two LEDs positioned on the ceiling at a visual angle of twenty degrees. Subjects were then instructed once more to refrain from deliberate key closure and to give an SSS number, when called by name, that corresponded to their perceived level of sleepiness immediately before hearing the report cue. While making final polygraph adjustments the experimenter provided practise trials for subjective reports and opportunities to test the behavior-lapse key. Before closing the door of the subject chamber, volunteers were assured of a secluded experience that would be interrupted only by the anticipated protocol of the experimental design. Subjects were also informed that a microphone adjacent to the bed allowed communication with the experimenter at low volume.

During the session SSS reports were solicited by either interrupting the white noise or calling the subject by name. Numerical replies to SSS cues were written directly on the chart, centered on the peak of the previous breath cycle. If at any time a report wasn't given within 5 sec from cue subjects were repeatedly called by name until a reply was obtained. Because recording episodes of sleep onset was of paramount importance communication with the subject during experiment one was minimized. As a consequence of this (and also of the experimenter's tendency to become engrossed in the unfolding record) subjective reports were often not obtained, as planned, from each third of the session.

Data Scoring and analysis

The original chart, recorded at 10 mm per sec, contained event markings representing key position, raw EEG from C-Z and O-2, 1-sec integrations of O-2 alpha, a DC EOG trace and DC thoracic and abdominal breathing records. Tape recordings contained FM-transformed signals of the event marker, raw C-Z, O-2 alpha and integrated O-2 alpha, all on one channel, and direct audio input from the subject chamber on the remaining channel. From the tape was produced a second chart containing event marks, O-2 alpha, integrated O-2 alpha, C-Z and integrated C-Z theta.

The first chart was used to measure excursions of integrated O-2 alpha, assess the presence or absence of SEM, to determine respiratory components of both derivations and to score 1-breath epochs with respect to the level of the event (key up or closed). A section of one such chart is reproduced in figure 1. The second chart was used to assess the presence or absence of alpha activity (scored blind as described below), to score latencies between alpha and event marks and to measure excursions of the integrator recording C-Z theta. See figure 2 for an example.

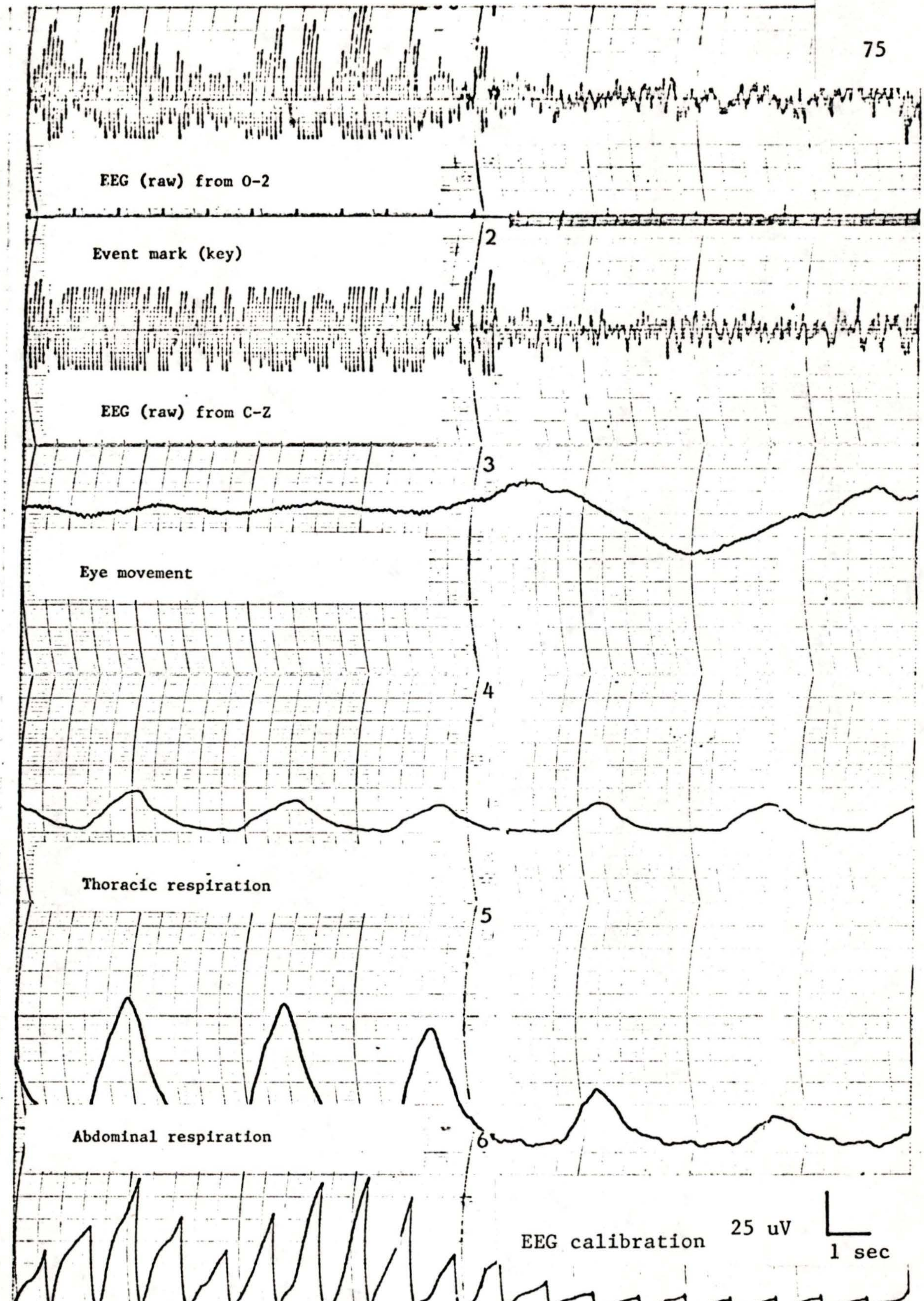

Figure 1: A sample of chart one selected to show well-defined physiological changes.

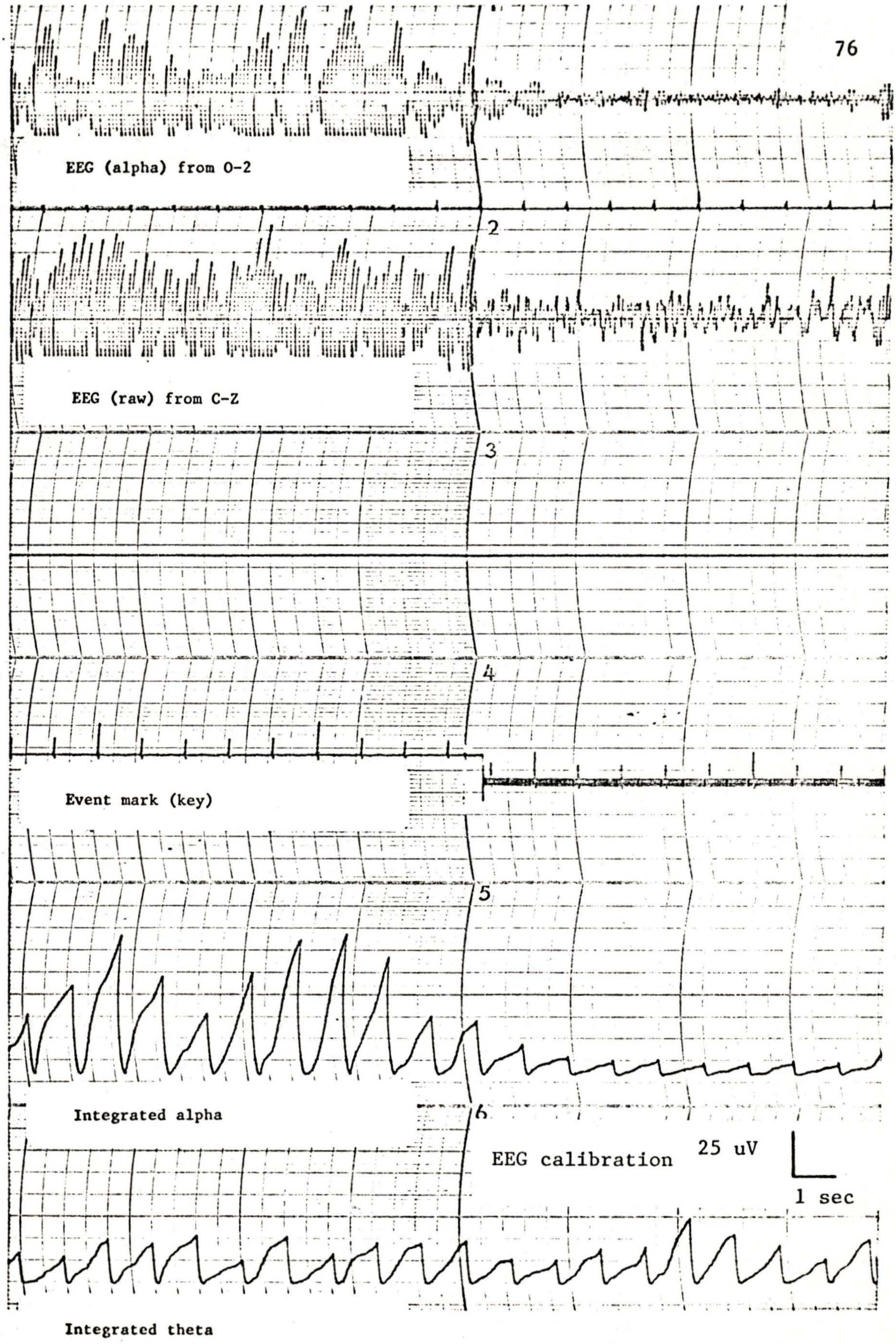

Figure 2: A sample of chart two for the same subject

Defining conditions for the purposes of this thesis are as follows:

1. Scorable alpha-loss events were periods during which the filtered 0-2 amplitude fell below 15 microvolts, peak to peak. To avoid scoring numerous momentary interruptions of alpha trains and periodic sections of amplitude modulations that fell below 15 microvolts, only alpha depressions in excess of 2 sec were scored.*
2. Scorable SEMs were defined as excursions greater than the equivalent of a 20 degree visual angle lasting 0.5 sec or longer. A deflection whose duration or amplitude was less than criterion during one breath cycle did not qualify unless it was imbedded in a SEM train flanked by scorable SEM in both adjacent breath cycles.

To determine how dependent measures varied with the behavioral variable, scoring epochs were chosen in the vicinity of a sustained behavioral lapse - which was invariably accompanied by a prolonged alpha loss - so that data could be gathered before, during and (when possible) after the beha-

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* Only one subject could not be scored in this way. Distinct, rhythmic alpha remained even at 10 microvolts; this was therefore considered as the limiting amplitude.

vioral change. To prepare for these analyses several scoring periods of 1-2 min that were free of artifacts and that contained one or more sustained (15-60 sec) key closures were first selected from representative sections of the subject's first chart. When the corresponding places were approximately located on the subject's second chart, all areas of that chart except the filtered alpha trace were covered so that alpha could be scored blind into segments judged present and absent.

To determine the relation of key level with alpha dynamics and other physiological variables, vectors of data were gathered at each breath. The following information was tabulated for each breath cycle during which the key was continually up or closed. If both key levels were present during a given breath cycle, data for that breath were ignored.

1. Observation number.
2. Key, scored as up (-1: "awake") or closed (+1: "asleep") during the breath.
3. O-2 alpha, scored as present (+1) or absent (-1) for at least 50 percent of the breath.
4. SEM, scored as present (+1) or absent (-1) for at least 50 percent of the breath.

5. Average value of 0-2 alpha integrations (intAL) during each breath.
6. An identically calculated value for integrated C-Z theta (intTH).
7. Thoracic amplitude (THOR) from peak of inspiration to trough of the previous expiration.
8. Abdominal amplitude (ABD) as above.
9. Abdominal expiratory time (ET) from peak of inspiration to the start of the next inspiration.
10. SSS rating ,when available, and time of occurrence.

Two derivative variables, ET/ABD and TA ratio, were generated by the SPSS program used to analyze each data set. ET/ABD was determined as indicated - the ratio of expiratory time to abdominal amplitude. In an attempt to avoid negative TA ratios, these were calculated in the manner of Naifeh and Kamiya (1981) as (3xTHOR - ABD) / (THOR + ABD).

In the presentations to follow, statistical tests of dependent measures were prepared by sampling several observations at each level of an independent variable and calculating a mean value at those levels for each subject. Individual subject means at each level of the independent variable were then compared with repeated measures Analyses of Variance (ANOVAs) to determine whether the difference in group means was significant with respect to an arbitrary confidence level.

The above procedure entails minor violations to ANOVA assumptions. Individual observations contributing to subject means were not independent. And because subject means were determined with an unequal number of observations, individual subject variances were different. The chief concern for this thesis was avoiding a type 1 error. Therefore, as a precautionary measure, in view of these violations, a relatively conservative confidence level of 99 percent was used throughout both experiments.

Analysis I: Key level and physiological measures.

For each subject mean values of each variable when the key was up (behaviorally awake) were compared with those in which the key was closed (behaviorally asleep). A within-subjects ANOVA was used with these subject means to test the predictions summarized in the review and supported by a pilot research: that key lapse would be associated with significantly more SEM, lower values of integrated O-2 alpha, higher values of integrated theta, lower abdominal amplitude, longer expiratory time, higher TA ratio and increased ET/ABD.

Analysis II: Interaction of alpha abundance and key level.

Among the many reasons for conducting this research was to compare high alpha subjects with low alpha subjects to determine differences, if any, in how dependent measures changed with levels of the behavioral variable. Such a test is unreported in the literature. It therefore seemed important to test the hypothesis that alpha abundance would not interact significantly with a behavioral criterion of SO. If, in general, the same relations between peripheral physiologial variables and the behavioral criterion obtained in subjects with lower as well as higher alpha abundance, then use of this behavioral indicator would be justified for subjects who didn't produce abundant wakeful alpha (for whom the use of alpha dynamics is difficult or impossible). For the purpose of this and subsequent analyses, 70 percent alpha time during the first few (wakeful) minutes of a subject's session was chosen as the lower boundary of the high alpha category.)

A 2x2 ANOVA with key and alpha abundance as independent variables was chosen to evaluate the presence of a significant behavior/alpha-abundance interaction with respect to each of the dependent variables.

Analysis III: Alpha level and dependent measures.

An analysis identical to that of analysis I was performed using visually scored 0-2 alpha as the independent variable and all other measures (excluding integrated alpha) as dependent variables. The same predictions were made, and again, only high alpha subjects were used.

Analysis IV: Latency from alpha loss to key closure.

The previous analyses focussed on selected 1-2 min scoring periods. To investigate the latency between alpha loss and key closures, all scorable alpha losses maintained with an accompanying key closure of any length were included wherever they occurred during the entire session.*

Mean latency data from alpha loss to key closure were first determined for each high alpha subject and then averaged across subjects. Data were then subjected to post-hoc scoring to minimize a possible carryover effect on latency from prior key closures. With this intent only those alpha/key conjunctions were scored that followed previous key closures by more than 10 sec.

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* If alpha reappeared for 2 sec in an extended stage 1 record this 2 sec break was considered to delimit separate alpha-loss events.

Analysis V: Interaction of key level and sex.

Because of the uncertainty about sex differences in respiratory changes a 2x2 ANOVA was planned on the original Analysis I data sets with alpha level and sex as independent variables. In order to obtain a larger number of subjects, and because most high alpha subjects were males, key level was substituted for alpha level. In view of results from the preceding two analyses this substitution seemed reasonable. SEM and EEG variables were not expected to show significant behavior/sex interactions . No definite prediction could be made about whether interactions would be found for respiratory variables of females in the luteal phase.

Analysis VI: SSS rating level as a function of time and condition.

A separate analysis was planned to determine the relation of SSS ratings with both polygraph pattern and with session time. SSS ratings were assumed to be equal-interval for application of a parametric test. It was predicted that SSS rating would show a significant trend with polygraph pattern (key up, key up with SEM and key closed) but not with time (successive thirds of the session). In order to make maximal use of the sparse data obtained, and in view of the strong relation between key and alpha shown by analysis III, key levels were considered as equivalent to alpha levels for this analysis.

A 3x3 ANOVA was planned with repeated measures on both time and polygraph pattern to test the predicted significant increase of SSS rating across polygraph patterns (conditions), the lack of a significant trend across time, and absence of a significant condition/time interaction. The paucity of data dictated reducing the analytic design to a 2x2 ANOVA - first and second halves of the session and key up vs key closed.

RESULTS AND DISCUSSION

To optimize clarity and cohesion, discussions accompany results of individual analyses as they are reported. Additional discussion follows at the end of each experiment.

One of the twenty people who volunteered for these two sessions reported discomfort during experiment one and showed no objective signs of entering stage 1 sleep throughout one hour of recording. This subject withdrew from further participation and his polygraph record was not scored.

Recording sessions lasted from 46 to 166 min with a mean of 91. Elapsed time from closure of the subject chamber to the first 30 sec epoch of stage 1 sleep varied between 3 and 53 (average 21) minutes. The mean latency to the first appearance of stage 1 for all-night recording sessions is 12 min. (Agnew and Webb, 1971).*

Four subjects did not contribute data to analyses I. EEG records of two subjects indicate membership in the 5 percent of the population for whom alpha is not the dominant waking frequency. They were both beta producers who occasionally showed sporadic alpha activity, often when they were behaviorally asleep. Their polygraph records were held aside and included among comparison records in the analysis III test for an alpha-abundance/key interaction.

Two other subjects were excluded from analysis I because they didn't produce key lapses. When asked about this, one mentioned having cultivated the ability to "lock" his body in place whereas the other's hand was positioned in such a way that loss of muscle tension could not result in key closure. Both claimed to have nodded off or napped during the session at times when EEG stage 1 sleep was present (alpha lost with low to moderate voltage theta and recognizable vertex waves).

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* This is the mean value of sessions 2 and 3 that began in the evening with preparations for undisturbed all-night recording.

It was generally unnecessary to adjust the tension of the telegraph key. Subjects were able to adopt comfortable juxtapositions of hand and key that were sensitive to loss of muscle tension. In retrospect, surprisingly few difficulties developed during experiment one. One subject exhibited several myoclonic jerks and seemed unable to assume the original orientation of hand and key, which is fairly critical. This was solved by taping that subject's finger to the key. Intentional loss of contact to rest the hand (with subsequent forgetfulness and sleep onset) was also discovered. These incidents were easily detected by alerting the subject whenever several minutes of stage 1 sleep appeared without a key closure. In all cases, with exceptions noted above, the subject confirmed loss of contact with the key and corresponding segments of the polygraph record were not scored.

After several such incidents the experimenter adopted the procedure of taping the subject's finger in place before the session began. Soon thereafter one subject displayed an extended key closure without anticipated signs of lowered alertness in the polygraph record. When questioned after the second such occurrence this subject replied that deliberate closure was effected to hasten the development of sleep onset. Instructions were then repeated and the session was resumed. Throughout the remaining sessions of experiment one the experimenter offered to tape the subject's finger in place if s/he had any doubt about maintaining unrestrained contact throughout the session. Three of the remaining eight subjects replied that this would be unnecessary and their refusal was affirmatively accepted.

Data scoring was as representative across the recording session as a given chart allowed. Some charts provided an abundance of useful data throughout the session. In other cases sleep onset was delayed or napping occurred only in the middle of a session. Whenever possible, though, data were gathered in continuous 1-2 minute scoring epochs containing sustained (15 - 60 sec) behavioral lapses. And in all cases each scored breath furnished data for all associated variables. The average record provided 100 such data vectors (range 62-157).

Analysis I: Key level and physiological measures

Table 4 is a summary of how physiological measures varied with the behavioral variable near and during SO transitions. The stated predictions, based on the available literature and supplemented by a pilot project, were supported by the results obtained with these fifteen subjects.

TABLE 4

Change in dependent measures at each level of key

Variable	Mean and S.D. key up		Mean and S.D. key closed		F (1,14)	p	Percent of variance
Alpha	0.21	(0.54)	-0.67	(0.41)	56	<0.0001	48
SEM	-0.24	(0.37)	0.25	(0.43)	21	0.0004	29
THOR	9.1	(3.3)	9.6	(3.5)	2	0.25	< 1
ABD	17.1	(4.9)	13.5	(4.2)	11	0.005	15
ET	23.4	(4.5)	24.9	(5.3)	6	0.028	2
intAL	11.6	(5.7)	5.8	(2.2)	20	0.0005	32
intTH	8.7	(3.8)	9.3	(3.4)	1	0.38	< 1
TA	0.41	(0.49)	0.69	(0.48)	33	<0.0001	8
ET/ABD	1.60	(0.72)	2.34	(1.55)	9	0.009	9

Key closures were associated with significantly less alpha activity, greater incidence of SEM, decreased abdominal amplitude, a higher TA ratio and larger ET/ABD. Neither thoracic breathing amplitude nor integrated theta (intTH) changed significantly with levels of the key variable, though both showed trends in the expected direction.

Because of an unfortunate oversight, values of integrated theta activity are misleading as written. The electronic filter used in this study had considerable "leakage", passing some of the activity in adjoining frequency bands. Set for a 4-7 cps bandpass, therefore, the resulting theta integration registered a decrease in level when alpha activity diminished. This partially offset the expected increase in level of integrated theta with the transition from alpha to alpha lapse, which tended to occur simultaneously in C-Z and O-2 derivations. In principle this factor could be corrected for individual subjects by duplicating the mean amplitude and frequency of their wakeful (key up) alpha activity with a wave generator, integrating its output from the same 4-7 cps bandpass and then subtracting this baseline value from the subject's wakeful theta values.

In terms of individual subject means at each key level, respiratory measures were remarkably consistent. Abdominal amplitudes were always lower during key closed periods, and ET/ABD was always higher. In many subjects these changes were dramatic and abrupt, tending to accompany key closure and alpha loss. Figure 1 exemplifies the decrease in abdominal amplitude and also shows a moderate increase in expiratory time. With one exception, TA ratios changed to higher values during key closed periods, but did so several breaths after lapse of alpha and key.

Analysis II: Interaction of alpha abundance and key level

Tests of analyses II and III required provisions for repeated measures with an unequal number of subjects per cell in the between-subjects factors. Data processing was accomplished as a series of successive multivariate tests transformed for repeated measures (Hull and Nie, 1981).

In analysis II a test of the key/alpha-abundance interaction was undertaken to determine whether this behavioral change resulted in systematic differences in selected physiological variables that were associated with the relative abundance of wakeful alpha.

The key/alpha-abundance interaction didn't approach significance for any of the dependent variables examined in this study. This is a fortuitous finding; it indicates that the key variable is an appropriate indicator of sleep onset in subjects who do not produce abundant wakeful alpha. (Of the two non alpha-dominant subjects studied here, one showed good agreement of changes in both behavioral and peripheral physiological measures and the other didn't.)

Analysis III: Alpha level and dependent measures

Nine of the 19 subjects who completed experiment one produced rhythmic alpha activity for at least 70 percent of their first few (wakeful) minutes. Table 5 shows results obtained with these high alpha subjects by using alpha as the independent variable and its visually determined presence (+) or absence (-) as levels of that factor.

TABLE 5

Change in dependent variables of high alpha subjects at each level of alpha

Variable	Means and S.D. alpha (+)		alpha (-)		F (1,8)	p	Percent of variance
KEY	-0.61	(0.30)	0.55	(0.36)	133	<0.0001	77
SEM	-0.15	(0.31)	0.28	(0.32)	17	0.004	34
THOR	8.4	(1.7)	9.4	(2.9)	4	0.09	5
ABD	20.3	(5.2)	14.8	(3.6)	13	0.007	29
ET	20.7	(3.8)	22.7	(4.0)	14	0.005	7
intTH	10.9	(3.7)	9.6	(3.5)	3	0.13	4
TA	0.20	(0.18)	0.56	(0.21)	24	0.001	49
ET/ABD	1.12	(0.26)	1.73	(0.46)	11	0.0097	43

Key level was strongly related to alpha level, and other physiological measures changed in the expected direction.

But again, both thoracic amplitude and integrated theta activity failed to attain significance.

Unanalyzed but frequently observed were simultaneous inceases in both alpha activity and abdominal amplitude, most of which signalled impending behavioral recovery.

Analysis IV: Latencies from alpha loss to key closure

Two latency determinations were performed. The original design provided for the inclusion of any key lapse with an accompanying scorable alpha loss (with the exception that an alpha appearance and subsequent loss during a key closure was ignored). Corresponding means and standard deviations are as listed in table 6

Discounting the first subject, results here are in accord with those of Blake et al (1938) who found that the usual latency between alpha disappearance and spool drop was 0.5 to 1.5 (average 1.1) sec. The main reason for determining latencies was to investigate the possibility that the Blake group's longer latencies - between 6.5 and 25 (average 14) sec - might reflect imprecision in the selected task. One subject of the present study also had a longer mean latency compared with others, and a correspondingly large standard deviation, with a range of 1 to 57 sec having no discernable

TABLE 6

Latencies from alpha loss to key closure in high alpha subjects

Subjects	latencies (sec) from alpha loss to key closure			latencies re-scored to avoid carryover effect		
	cases	Means and S.D.s		cases	Means and S.D.s	
1	20	18.4	(15.5)	19	19	(16)
2	29	5.5	(4.4)	22	6.6	(4.6)
3	20	4.2	(5.3)	14	5.3	(6.0)
4	22	2.2	(4.3)	8	4.5	(6.7)
5	37	1.0	(2.6)	15	1.7	(2.8)
6	21	0.7	(2.1)	19	0.8	(2.1)
7	24	0.3	(1.8)	14	0.3	(1.4)
8	23	-1.8	(4.7)	12	5.0	(2.1)
Means and S.D.s for all subjects		3.8	(6.3)		5.4	(6.0)
Means and S.D.s for 2 - 8 only		1.7	(2.5)		3.5	(2.5)

trend across the session.* This anomalous subject volunteered information about being extremely conscientious in regard to holding the key open as long as possible. With this one exception, within-subject variabilities were comparatively low as was the calculated between-subject variabil-

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* None of the records show an obvious trend across the session when latency is plotted against time.

ity. It can therefore be concluded that the key indicator is reasonably precise within subjects and reproducible across subjects. Perhaps the unusually longer mean latency (and larger standard deviation) reflects added influence from subject variables - greater motivation? - that extended the performance beyond normal limits, thereby resulting in more variable as well as longer latencies.

Negative latencies - key closure sustained until alpha loss - were most prevalent in subjects with the lower mean latencies. The records of subjects 5 - 8 contained upwards of 33 percent negative latencies.

While scoring these conjunctions it was noticed that prior key closures seemed to exert a carryover effect on the latencies of nearby alpha/key conjunctions, making them shorter and even negative. Latencies were then re-scored, omitting any conjunctions that followed within 10 sec of recovery from a key closure. This change in scoring probably provided the equivalent of a closer approximation to inter-trial conditions imposed on the Blake group subjects, who were undoubtedly alerted somewhat between trials to reposition the spool. As can be seen in the above table, re-scored latencies are longer but not, as anticipated, less variable.

Analysis V: Interaction of alpha level and sex

To determine whether sex interacted significantly with alpha level, key level was substituted for alpha level and the appropriate analysis was performed with the 8 male and 7 female subjects of analysis I. No significant key/sex interaction was found for any of the dependent measures examined in this study.

The sex main effect approached significance for abdominal amplitude $F(1,14) = 8$, $p = 0.014$. Re-analysis of the wakeful (key up) level shows significantly lower relative abdominal amplitude in female subjects $F(1,14) = 10$, $p = 0.007$, and a non-significant trend in the same direction at the key closed level.

Naifeh and Kamiya (1981) reported higher TA ratios in their female subjects, as is the case here. Present results indicate that this arose from reduced abdominal breathing relative to males. Other factors equal, pulmonary volumes are 20-25 percent lower in the female (Guyton, 1966), and this is reflected in lower breathing amplitudes. What is not so easily understood on purely anatomical grounds is why this difference would appear only in the abdominal amplitude. It is estimated that 70 percent of the expansion and contraction of the lungs is caused by anteroposterior movement of the chest cage (Guyton, 1966), yet males and females in this study had indistinguishable mean thoracic breathing excursions during wakefulness and sleep.

Analysis VI: SSS rating levels across time and condition

The intended analysis of SSS with repeated measures on both session time and polygraph pattern was defeated because of numerous missing observations in one or more cells. This was even the case with the design reduced, post-hoc, to two levels of each variable - first and last halves of the session and key up vs key closed. Separate tests on each of these factors were therefore devised that would use the available observations to best advantage.

To prepare for a test of how SSS report changed with session length, the chart time of each subject's last SSS rating was taken as the session length and divided into halves.

In an attempt to remove the influence of session length an ANOVA was performed on key level means that were matched on session half. The converse comparison was also made: session halves matched on the same key level. As a compacted prediction from the original hypothesis it was expected that key level would account for more difference in SSS report than would session level. Table 7 summarizes results of these two within-subjects ANOVAs.

TABLE 7

An improvised test of main effects for SSS ratings

variable	comparison		F	p	percent of variance
key matched on halves	up	closed	F(1,9)		
	5.7 (0.9)	6.5 (0.7)	12	0.0007	23
Halves matched on key	first	second	F(1,8)		
	5.2 (0.9)	6.3 (0.6)	14	0.004	37

Both time and condition were important contributors to SSS ratings. An estimation of the interaction magnitude was then sought by returning to the original 2x2 ANOVA and filling missing observations in each cell with that cell's mean. Results confirmed the significance of main effects and showed a significant interaction between session length and key level *F*(1,9) = 14, *p* = 0.005. The difference in SSS rating during the first half of the session (4.6 up vs 6.1 closed) was greatly reduced in the second half when both key levels were rated high (6.2 up vs 6.5 closed).

A test of physiological variables for similar interaction of key and session time was then performed. The first and last SO scoring periods of SSS subjects were chosen for reanalysis and a repeated measures ANOVA was performed for each variable using mean values at each key level from each scoring period. No significant interaction could be found for any of these variables. Nor were there significant main effects for time, but integrated alpha tended to be lower and integrated theta to be higher in the late scoring period.

Lack of significant physiological differences across the session, in spite of differences in SSS ratings, is not unusual in light of a recent report by Broughton et al (1982). Working with narcoleptic and normal control subjects, they discovered that subjective sleepiness was rated greater by the narcoleptics in spite of comparable task performance and EEG-defined level of wakefulness. Significant distinctions between these groups were visible only in some components of evoked potential data. It may be the case, therefore, that SSS ratings reflect, in part, a subject's awareness of the passage of time, perhaps as a function of physiological changes that weren't monitored in this study.

ADDITIONAL DISCUSSION: EXPERIMENT ONE

Behavioral lapse and the EEG

The telegraph key served as a reasonably precise and reproducible indicator of passive behavioral lapse despite known (and possibly unknown) incidents during which its liabilities were manifested. The most frequent sources of potential variability arose from accidental or deliberate loss of contact with the key. Direct inquiry after extended alpha lapse without key closure successfully identified such incidents, however. And premature (emitted) key closures were easily spotted as anomolous episodes since EEG and other changes almost invariably precede sustained key closures.

In retropect, though, an EMG record from the extensor of the third digit would have been a welcome supplement to the event marker. It could have provided an independent criterion of behavioral lapse and additional information about the dynamics of sustained muscle tension near sleep onset transitions.

The latency from alpha loss to behavioral lapse was focussed upon to the exclusion of another latency that could have been included within the scope of experiment one. The interval between alpha reappearance and key recovery wasn't determined; subjects were instructed only to raise the key whenever they noticed that it was closed, not to do so as quickly as possible. It was generally the case, however, in agreement with observed latencies between resumed alpha and "float report" signals (Davis et al, 1938), that subjects raised the key within two breaths after alpha returned.

Peripheral physiological measures

The progress of research with respiratory variables has been stalked by two major suspicions: first, that methods of measurement alter the outcome; second, that one or more sex-related effects contribute to inconsistencies in the literature.

The first of these issues once revolved about the technique of spirographic recording, which necessitated mouth breathing. This produced distortion in the pattern and amount of ventilation (Gilbert et al, 1972). The arrival of less invasive procedures brought improvements accompanied by still other drawbacks. Prolonged postural immobility, for example, is required when using mercury thread strain gauges, and use of these devices has generated several research reports based on supine subjects. The extent to which a supine position may have produced its own unique respiratory pattern or injected artifacts into the dependent measures of this thesis can't be known until some of the older studies are repeated with state-of-the-art instrumentation (Timmons, 1982).

The issue of a possible sex differential has not been satisfactorily addressed. As mentioned before, 6 of 10 female subjects studied by Pieron (1913) gave opposite TA ratio changes. Reed and Kleitman (1925), who set out to resolve several inconsistencies, presented mixed results that were highly variable within and between subjects with no clear indication of the source of variability. Some studies used only males, and the Timmons group (1972), who found definite abdominal decreases in close association with alpha loss, included only one female in their population of 11. Finally, Naifeh and Kamiya (1981) reported higher resting awake TA ratios in their female subjects, but without further comment.

It was considered a prerequisite of the present research to minimize sources of sex-related variability by scheduling female subjects at times of relative constancy. The luteal phase of the monthly cycle was selected because it offered several days of latitude for the convenience of subjects during which their hormonal levels were relatively constant and conducive to optimum ventilation (Skatrud, 1978).

Comparison of female with male values of each dependent variable at each level of the behavioral indicator resulted in the discovery of significant differences in abdominal respiration. Smaller wakeful abdominal excursions in females can explain the sex difference in TA ratios reported by Naifeh and Kamiya.

More importantly, no significant interaction with the behavioral indicator was found for any of the dependent measures. Results of experiment one therefore replicate similar studies with similar outcomes (e.g., Timmons et al, 1972) regardless of the sex composition of research populations. The present results can also provide reference material for studies that employ other independent variables, including sex-related factors.

A discussion on respiratory monitoring would be incomplete without reporting the more qualitative aspect of respiratory tracings from thoracic locations. Changes in topography with subject condition have been described and illustrated by Lugaresi (1975), Timmons et al, (1972) and Tusiewicz et al, (1977). Generally confirmed by this study was the association of a positively accelerated inspiratory slope with wakefulness, giving a peak-shaped appearance to the complete breath cycle. After alpha has disappeared the respiratory trace tended to be dome-shaped with a distinct negative acceleration of the inspiratory slope. (Whether a reliable index of sleep onset could be devised based on qualitative criteria is not explicitly addressed in the literature. Various authors implicitly extend the possibility, but don't develop it.) A characteristic flattening of the inspiratory slope always appears while snoring is audible. Extreme flattening and even a paradoxical negative excursion (with positive abdominal expansion) was observed in four sessions during sleep apnea episodes of one subject who does not appear in this study.

Stanford Sleepiness Scale

Previous results with the SSS have woven it into the descriptive tapestry of performance decrements and sleep loss (Hoddes et al, 1973; Glenville et al, 1978) and sleep tendency (Carskadon and Dement, 1979). Whether the SSS will be revised depends largely on its present and potential utility in clinical and experimental assessments. If a simple self-report measure can succeed in replacing an extensive or costly procedure, everyone gains.

As reported in this study, the present SSS shows strong main effects for session time as well as behavioral condition, a limitation not showed by the simpler sleepiness scale of Foulkes and Vogel (1965). Of course the latter study imposed no postural requirements and awakenings were made across an entire night. But a replication attempt could be devised that would enable the SSS to be tested under similar conditions. If a time effect is still found and if a time/condition interaction can be demonstrated for SSS report level but not for physiological variables, then the utility of this scale as a simple report measure of sleepiness level is questionable. Better results might be obtained using a numerical scale without verbal descriptors.

EXPERIMENT TWO

Performance on many vigilance detection tasks, including the Wilkinson auditory vigilance test, declines across the session. Among the more robust findings with these tests is that errors of omission are preceded by indications of reduced wakefulness in the EEG. Sleep deprivation studies have demonstrated that performance can be predicted with peripheral physiological variables as well, and many of these have been ignored in normal vigilance studies. Further, the implications of performance decrements across sleep onset levels have yet to be tested with a signal detection analysis. Experiment two was addressed to these investigations.

METHOD

Subjects

Sixteen of the nineteen subjects who completed experiment one participated in the second session. No new subjects were added.

Apparatus

In addition to devices employed in experiment one, equipment suitable for conducting the auditory test was included:

1. As before, continuous output from a Marietta model 24-21 white noise generator was delivered through a speaker located on the floor about 1 meter below the subject's head. Sound intensity measured from that distance was 48 dB (A).
2. All tones were produced by a Knight model KG 688 wave generator set at a frequency of 500 Hz and delivered through headphones located on the floor near the white noise source. Tone intensity, identically measured at the subject's head, was 63 dB (A).
3. Two interval timers were used: a Hunter Decade Interval Timer, model 111C, to deliver the 500 msec non-target tone and a Digital Millisecond Timer capable of being adjusted to the nearest 10 msec in the range being used.
4. Tone deliveries were controlled by tape loops with a manual override option for selectively presenting additional events or avoiding programmed ones.

Procedure

Subjects were prepared as before for polygraph recording and informed about the parameters of their detection task:

1. Tones would differ only in duration.
2. Subjects were to report the probable occurrence of the shorter (target) tone and make no response to the other.
3. Tones would appear at an average rate of 3 per min but range between 10 and 120 sec apart.
4. Fifty percent of these tone events were targets but their delivery would be randomized to prevent a tone's identity from being predicted on the basis of past presentations.

After the application of respiratory gauges and calibration of eye movements, subjects were given an ample opportunity to compare target and non-target tones. A practise period with feedback was also provided that continued until the subject had both attained 90 percent accuracy on 10 successive trials and also expressed willingness to proceed with the experimental session.

Changes in both task and procedure were made halfway through the data collection phase of experiment two. The first eight subjects were prepared as described above and given the task of discriminating a 400 msec target tone from the non- target tone of 500 msec. When it was noticed that they performed well at reduced levels of wakefulness and even offered unsolicited reports that the discrimination task was increasingly easy throughout the session, changes were introduced in order to make the task more difficult. The second group of subjects had a 420 msec target tone and were given practise trials with feedback until they agreed to continue with the session. (Practise was not continued to 90 percent accuracy.) They then received a 5 min period of adaptation to stabilize their responses to the target tone. Following this, additional discrimination practise was given until subjects agreed to begin the experiment.

Before the session began subjects were again instructed to signal only the probable occurrence of the shorter tone. Any vocal response was accepted, and they were encouraged to adopt one that required minimal activation.

Data Scoring and analysis

Several physiological variables used in experiment one were recorded at the 1-breath epoch whose peak preceded the tone. The following information was collected:

1. Observation number.

2. Class of response to each tone (hit, miss, correct rejection or false alarm).
3. Sleep stage (W, 1 or 2)* based on the EEG traces during the four seconds immediately prior to tone presentation.
4. SEM activity, measured as before, but evaluated over the same 4 sec interval.
5. Integration values for alpha and theta measured during the 1 sec prior to tone onset.
6. Respiratory values determined as before (except for expiratory time and ET/ABD which were eliminated to avoid introducing more lag time between the tone and values associated with the nearest breath.

Because of alterations to experiment two, corresponding differences in performance, among other indications of differential treatment, were anticipated. The most straightforeward approach to the resulting data was to carry out the intended analyses on both groups combined, using single factor ANOVAs with repeated measures at both levels of the com-

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* For subjects who didn't produce abundant wakeful alpha, stage 1 was scored if one of the EEG traces contained 3 successive waves in the theta range and neither trace showed organized fast (alpha or beta) activity. Stage 2 was scored for all subjects when the C-Z trace showed at least one sleep spindle (13-14 Hz) of 0.5 sec duration or one K-complex of at least 100 microvolts peak to peak since the last stimulus presentation with no evidence of resumed alpha or increases in frequency since then.

parison. Identical analyses were performed for each subgroup when they seemed warranted by a comparison of subgroup means. But 2x2 ANOVAs were substituted when the intent was primarily to detect possible interactions between independent variables and differential treatment. Finally, unequal-n ANOVAs were used as needed to test specific differences between subgroups at one level of a particular factor.

Analysis VII: Physiological indicators of performance decrements.

On the basis of existing performance studies of normal and sleep deprived subjects reviewed by Parasuraman and Davies (1980) and findings of the pilot study, it was predicted that misses would be preceded by depressed values of both EEG and peripheral physiological variables compared with their values immediately preceding hits:

1. Deeper (higher numbered) sleep stage rating.
2. Greater SEM abundance.
3. Greatly reduced alpha activity.
4. Increased theta activity.
5. Lower abdominal amplitude.
6. Higher thoracic amplitude.
7. Larger TA ratio.

Previous EEG findings focussed on correlates of omission errors. Correlates of false alarms have been reported only in evoked potential studies. If the subject is alert enough to emit a response, and that response is incorrect, it might be anticipated that false alarms would show an intermediate pattern between the two extremes of hits and misses.

Because the main intent was to distinguish each response category from the others, pairwise within-subject ANOVAs were performed to test differences between physiological measures of each focal category.*

Analysis VIII: Vigilance performance as a function of sleep onset level.

A standard signal detection analysis was planned for each subject. Sensitivity and criterion levels, based on the percentage of hits and false alarms, were to be calculated from responses to tones presented during alpha, alpha-SEM and alpha-loss periods. Sleep stage (W, 1, 2) was later substituted when it became obvious that these discrimination tasks were not sensitive enough for performance to be discriminable in terms of SEM activity. Unfortunately, sleep stage was not scored blind with respect to the response type. But subsequent blind-scoring of segments from eight

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* Correct rejections weren't scored because lack of a response has no clear implications regarding sleep onset level of origin.

representative records agreed to the extent of 85 percent (range 65-94) with the original scoring.

On the basis of subjective float reports and decreased responsivity during these times (Davis et al, 1938), alpha-loss periods were expected to show significantly lower sensitivity.

RESULTS AND DISCUSSION

Traditional vigilance tests concerned with performance decrements typically processed raw data (hit and false alarm rates) chronologically for several blocks of the session. The intent of experiment two, however, was to examine performance as a function of sleep onset level. And although an alternate analysis across session time might have been desirable (as for SSS rating), it was inappropriate. One or more experimenter interventions occurred in about half the sessions whenever subjects remained unresponsive for more than five minutes. They were then alerted and conversed with briefly before the session continued. Interrupting otherwise continuous task performance with rest breaks is widely recommended to offset performance decrements (Parasuraman and Davies, 1982).

Fourteen subjects provided scorable charts during experiment two. In the analyses to follow results of the entire group are reported first and followed by a comparison of subgroups.

Analysis VII: Physiological correlates of performance decrements

Table 8 presents means and average standard deviations for the three focal response categories of experiment two. The table is based on the 10 subjects who provided at least 4 cases to each category listed. It also includes data from one subject on the key variable. This subject displayed an unusual facility for falling asleep during experiment one and agreed to include the behavioral lapse key as a comparative measure with the variables of experiment two.

Pairwise comparisons of hits, false alarms and misses were performed, as before, via within-subject ANOVAs for each of the dependent variables. Individual subject means composed of 4 or more cases were included in the comparisons of table 9.

Comparing hits and misses.

Table 9 reveals the familiar pattern of decreased wakefulness in comparisons of hits and misses. When all sub-

TABLE 8

Mean values of dependent variables for the three focal response categories

Variable	Means and Standard Deviations		
	Hits	False alarms	Misses
Stage	0.58 (0.36)	0.72 (0.42)	1.28 (0.55)
SEM	-0.37 (0.31)	-0.54 (0.40)	-0.53 (0.36)
THOR	11.4 (6.0)	12.4 (6.5)	12.5 (6.6)
ABD	17.7 (5.0)	17.8 (5.0)	15.0 (3.9)
intAL	8.0 (4.1)	7.5 (3.3)	6.8 (4.3)
intTH	8.8 (2.0)	9.3 (2.2)	10.7 (2.7)
TA	0.56 (0.58)	0.62 (0.61)	0.76 (0.63)
Key for 1 subject	-0.41 (0.93)	0.33 (1.00)	0.66 (0.76)

TABLE 9

Comparisons of dependent variables for hits and misses

Variable	Means and S.D.s Hits	Misses	F(1,12)	p	percent of variance
Stage	0.54 (0.35)	1.13 (0.60)	28	0.0002	28
SEM	-0.36 (0.35)	-0.44 (0.39)	1	0.4	1
THOR	10.8 (5.5)	11.9 (6.0)	3	0.09	1
ABD	17.4 (4.7)	15.1 (3.5)	13	0.004	8
intTH	10.6 (5.2)	11.9 (4.3)	6	0.04	2
TA	0.52 (0.52)	0.72 (0.55)	13	0.003	4

jects were combined, values of sleep stage- where W is coded zero and stages 1 and 2 are coded accordingly - was the best predictor of misses. Abdominal amplitude and TA ratio also distinguished hits from misses.

Similar mean values of SEM can be understood with reference to the sleep stage from which hits and misses were recorded. Hits came predominately from stages W and 1, while the majority of misses occurred during stage 2 and late in stage 1 when SEM activity subsides.

Integrated alpha, determined for high alpha subjects, was also unable to distinguish hits from misses. Correct responding continued during some alpha loss periods and misses were sometimes recorded when alpha was present. There were also systematic differences in integrated alpha between the two component subgroups. High alpha subjects of the first group had a mean of 6.3 units during the one sec preceding misses, which is similar to its mean score during key closures of experiment one. High alpha subjects of the second subject group, who had the harder task, inflated the mean with their average of 15.6 units before misses.

Data were also analyzed for component subgroups of experiment two. For the first subgroup, sleep stage was again significantly deeper (larger number) before misses than before hits $\underline{F}(1,6) = 36$, $\underline{p} = 0.001$, and integrated theta was significantly higher $\underline{F}(1,6) = 24$, $\underline{p} = 0.003$. This is in complete agreement with previously found correlates of vigilance detection errors (Groll, 1966; O'Hanlon & Beatty, 1977; Morrell, 1966; Horvath et al, 1975; Gale et al, 1977). Lower integrated alpha before misses, however, was non-significant.

Sleep stage in group two barely missed achieving significance $\underline{F}(1,5) = 13$, $\underline{p} = 0.015$. Integrated theta didn't distinguish hits from misses in this subgroup. Performance deteriorated before major increases in theta abundance and amplitude could be expected (late stage 1 and stage 2).

Abdominal amplitude failed to distinguish hits from misses in the smaller component groups at the 99 percent confidence level, but they did claim 10 and 7 percent (respectively) of shared variance with the independent variable.

Comparing hits and false alarms.

Dependent measures of this study differed little in comparisons between hits and false alarms, whether in the single large group or its constituents.

Comparing false alarms and misses.

As for distinguishing between false alarms and misses, this was best acomplished, as might be hoped, with the same variables that differed in comparisons of hits with misses. Compared with their values before false alarms, sleep stage was deeper and abdominal breathing was shallower before misses in both the large group and its initial component subgroup. In the second subgroup abdominal amplitude was smaller before misses and theta activity greater, but these changes were not significant. As shown by table 8, however, false alarms have a generally intermediate wakefulness pattern relative to the extreme differences in dependent variables when hits are compared with misses.

As reported, physiological characteristics of false alarms are in no way significantly different from those of hits. Such a conclusion, based as it is on a small sample population and a limited number of dependent measures, can hardly be conclusive. But it is consistent with the need for a certain minimum level of activation as a substrate for emitting a response, whether correct or incorrect.

Analysis VIII: Vigilance performance as a function of SO level

It will be recalled that a report of percent hits, by itself, is ambiguous. A low hit rate could be attributed to the observer's insensitivity or to the use of a conservative criterion about what constitutes a reportable signal. Accordingly, comparisons and discussions of this section are mainly in terms of p(A), a non-parametric estimate of sensitivity based on the percentage of hits and false alarms. (Because transforming the small number of false alarms to an index of criterion level is relatively hazardous, a similar non-parametric measure of criterion level was not included.) p(A) data for all subjects of experiment two were obtained from a table (McNicol, 1972) and appear in data table 10 at each sleep stage. Data for the second subgroup could not be reported owing to an insufficient number of stage 2 presentations.

Treating the group as a whole, when p(A) was compared across stages the anticipated results were obtained. From a mean of 94 in stage W, p(A) declined to 81 in stage 1. Results of a 2x2 ANOVA with repeated measures on stage (W vs 1) revealed significant differences for stage $\underline{F}(1,11) = 34$, $\underline{p} < 0.001$, for group $\underline{F}(1,11) = 13$, $\underline{p} < 0.005$, and also a significant interaction of stage and group $\underline{F}(1,1) = 11$, $\underline{p} < 0.008$.

TABLE 10

Values of p(A) for each sleep stage

Sub-group number	R&K sleep stages W	1	2
1	92	95	75
1	94	95	75
1	97	96	60
1	95	87	67
1	95	87	100
1	91	77	--
1	99	81	--
1	--	84	60
2	97	68	
2	90	73	
2	86	60	
2	90	73	
2	96	82	
2	91	--	

Five of the first 8 subjects provided 4 or more observations for determining pecentages of both hits and false alarms in all three sleep stages. Figure 3 shows their performance compared with the 5 subjects of the second subgroup, who didn't provide enough data for stage 2 determinations.

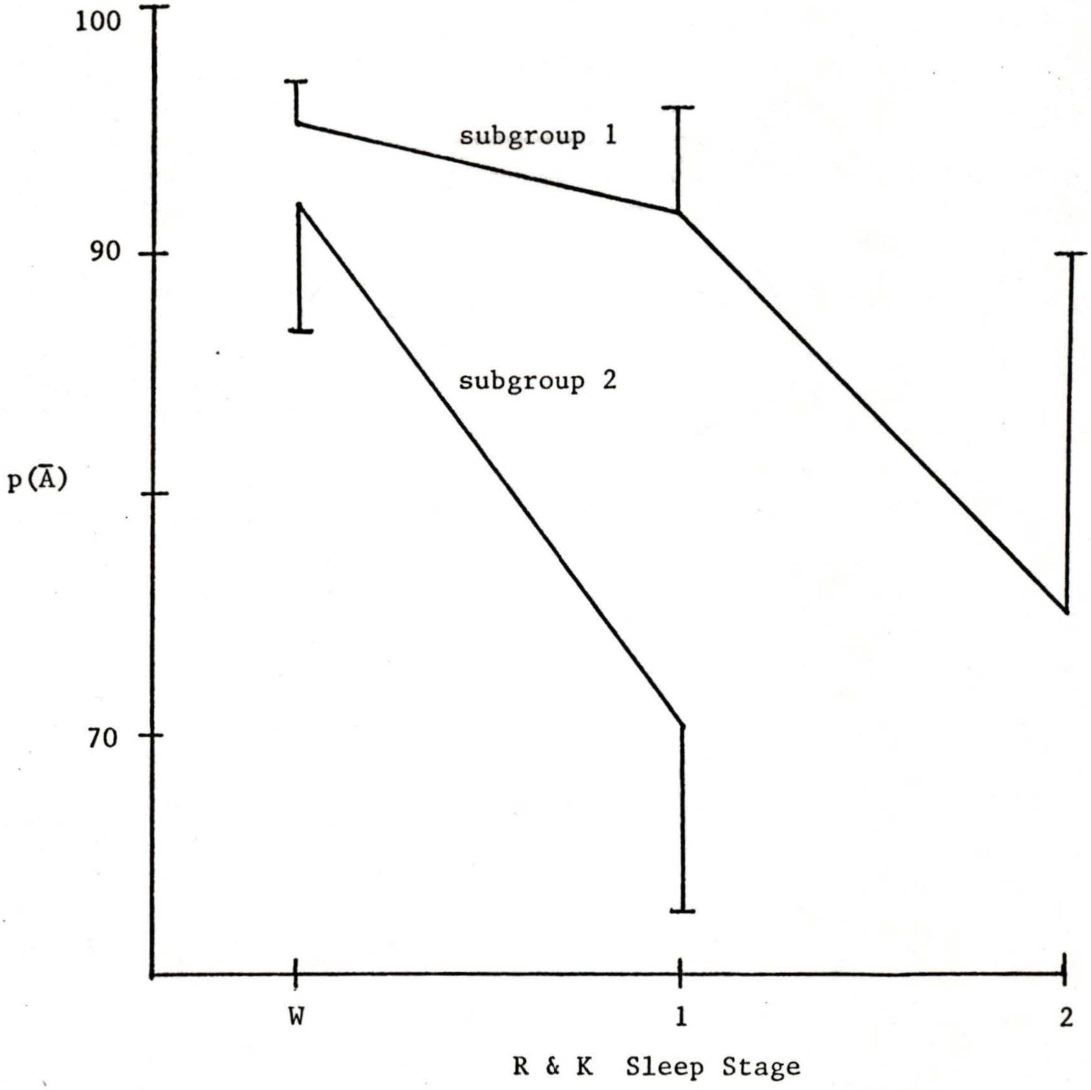

Figure 3: Sensitivity scores for each of the subgroups across sleep stages. Standard deviation units are indicated by bracketed vertical lines.

Group one showed a marked decrease in sensitivity from stage 1 to stage 2. Because of high variability in stage 2 this p(A) difference was not significant. Group two, for whom the task was intended to be more difficult, showed an earlier and comparable decline in sensitivity at stage 1 $\underline{F}(1,4) = 50$, $\underline{p} = 0.002$.

In a comparison of groups, mean p(A) values were indistinguishable in stage W. But sensitivity at stage 1 was significantly lower for group two $\underline{F}(1,9) = 25$, $\underline{p} = 0.001$. The groups differed considerably in stage 2 presentations as well. The first group, who found the discrimination task easy, logged considerable stage 2 sleep, where 21 percent of their trials were delivered. Stage 2 trials of the remaining subjects accounted for only 4 percent of their total $\underline{F}(1,13) = 8$, $\underline{p} = 0.015$.

It seems inescapable that sleep onset level influences performance. Whether sleep onset level - particularly a theta factor - is responsible for the frequently analyzed time effect remains an attractive but still untested hypothesis.

ADDITIONAL DISCUSSION: EXPERIMENT TWO

Rate of decrement as a function of stage

When the Wilkinson auditory task was employed at the usual discrimination parameters of 400 and 500 msec, it was judged easy and correct responses were often obtained from presentations that would have occurred long after behavioral lapse. But correctly discriminating between 420 and 500 msec tones failed much sooner. The discovery of this interaction of sleep onset level with task difficulty was a provocative addition to results of experiment two. If a discrimination task of greater difficulty is equivalent to one of diminished signal intensity, and if the time effect is reducible to a stage effect, then these results contribute to an unresolved controversy about the rate of performance decrement across time for signals of different intensity.

That signal strength affects detection performance in both visual and auditory tasks is well established (Parasuraman and Davies, 1982). But it remains inconclusive whether the rate of decrement is also affected.* Metzger et al (1974) found that visual signal intensity, operationalized as degree of displacement, didn't significantly affect the rate of performance decrement. Adams (1956) observed decre-

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* There is evidence that a related factor - signal duration - has such an effect (Baker et al, 1963).

mental performance on a visual detection task as functions of both stimulus intensity and duration, but didn't analyze differences in rate. Lisper et al(1972) studied rate of response to various intensities of a tone and reported a significantly greater increase in reaction time across the session - but only for the near-threshold tone. One reason why prior studies are inconclusive is because time is partly confounded with sleep stage. What is needed is an experimental design with a random series of tones differing only in intensity, and a dual analysis: a determination of performance decrement by sleep stage partialling out time, and a determination by time partialling out stage. It is hypothesized that only the former analysis will show significant decrements across stage with the same interaction discovered in the present study.

Performance limitations at higher thresholds

When the discrimination task is easy the performance-limiting factor may be a perceptual truncating of the tone. As one subject mentioned, "Sometimes I hear only the last segment of the tone and have to guess". Decreased sensitivity in stage 2 supports this possibility. As for a mechanism to account for truncating the tone, Elliot (1957) discovered periods of increased auditory threshold during detection tasks which he speculatively linked to episodes of drowsiness. It is plausible that the initial few msec of a tone is needed to evoke an EEG activation - as postulated for the stimuli of the Fischgold and Okuma experiments - which then alerts the subject to the presence of an event.

SUMMARY AND CONCLUSIONS

Much of the existing literature pertaining to sleep onset is largely unsystematic and inadequately reported. Studies to date convey an incomplete understanding of behavior- EEG relations and little information about correlations between behavioral changes and peripheral physiological measures. Even less is known about inter-relations of behavioral, physiological and subjective correlates of sleep onset in the same subject population. This thesis attempted a comprehensive review of studies relevant to sleep onset and undertook investigations intended to:

1. Replicate selected aspects of existing behavior-EEG studies.
2. Extend them by monitoring peripheral physiological and subjective variables.
3. Investigate the generality of sleep onset measures across sex and alpha abundance.
4. Attempt a signal detection analysis of decrements in vigilance performance at selected sleep onset levels.

Experiment One

Blake et al (1939) had used a passive behavioral lapse as an indicator of sleep onset. Their finding of a passive lapse associated with simultaneous or slightly prior loss of EEG alpha activity was replicated using subjects with abundant wakeful alpha.

Further established by the comparisons of the degree to which peripheral physiological measures changed with behavioral (key) lapse was the lack of an alpha abundance interaction with key level. Aside from confirming the usefulness of such a measure with subjects regardless of alpha abundance, these results suggest that any peripheral measure that correlates strongly with this behavioral measure can be used as a general indicator of sleep onset regardless of alpha abundance.

Also established, when males are compared with females tested during the luteal phase, was the lack of a significant interaction of sex with this passive behavioral lapse for any of the variables used in this research. This implies that these variables may be equivalently employed to monitor the progress of sleep onset in young adults regardless of sex.

A determination of the latency from alpha lapse to key closure generally replicates results of Blake et al (1939). Both studies found a majority of subjects who exhibited a passive lapse 1-4 sec after alpha loss and a minority of subjects with a considerably longer latency.

Because of faulty data sampling, conclusions about subjective indicators of sleep onset are tentative. Use of the Stanford Sleepiness Scale resulted in sleepier mean ratings for the key closed condition and for the second half of the session. An interaction was also found such that the differential SSS ratings between key up vs closed largely disappeared in the second half of the session. A similar test of objective measures failed to find a significant key/time interaction for any of the physiological variables used in experiment one. A replication attempt using more and better-scheduled SSS reports is strongly indicated to determine the degree of association of subjective report with condition and across time. Except for sleep deprivation studies the SSS has not been compared with objective sleep onset changes. If a replication attempt were to reveal the same discrepancy reported above regarding an interaction of session time and condition for SSS report but not for physiological variables, then the utility of the SSS as a simple report measure of sleepiness level would be questionable.

Experiment Two

Subjects who completed experiment one were identically monitored during a modified version of the Wilkinson auditory vigilance test administered in session two. The first eight subjects discriminated the shorter (400 msec) target tone of 500 cps from a 500 msec non-target tone of the same frequency. Because of both objective and subjective evidence that their task was too easy, the remaining subjects had the more difficult 420-500 msec discrimination.

Previous investigators found errors of omission associated with prior signs of depressed EEG activity, and studies of sleep deprivation showed depressed values of peripheral physiological measures as well. An attempt to correlate performance on the Wilkinson test with several physiological variables was partly successful here. For all subjects misses differed from hits by having a higher-numbered sleep stage in the four sec prior to tone delivery, and both a lower abdominal amplitude and higher TA ratio in the preceding breath cycle. The group with the easier discrimination also showed more theta activity in the 1 sec preceding misses. No other variables discriminated between these response categories. And none of the variables distinguished hits from false alarms, whether determined for subgroups or for all subjects combined.

A complete signal detection analysis was precluded by insufficient data. But a non-parametric estimate of sensitivity, p(A), was calculated for all subjects. This measure decreased from stage W to stage 1 in accord with prediction. When sensitivity data for the two subgroups were analyzed in a 2x2 ANOVA, significant main effects were found for both sleep stage and task difficulty. An interaction was also discovered. In stage 1 the group with the more difficult discrimination showed a significantly lower sensitivity compared with their similarly high values in stage W. Group one, who had the easy discrimination, underwent a similar sensitivity decrease from stage 1 to 2.

Previous investigators had found sensitivity decrements on the Wilkinson test when scores were calculated across blocks or correlated with the extent of sleep deprivation. Because the present experimental performance could not be analyzed across time, and because the stated modifications were made to the Wilkinson test, a direct comparison of sensitivity decreases across both time and condition could not be made. But the available evidence is consistent with an explanation of sensitivity decreases across time in terms of an erosion of discriminatory capacity with the progress of sleep onset.

In a wider perspective, reliance on EEG criteria to indicate functional level was essentially validated by both of these experiments. Spontaneous lapse of muscle tone and decreased auditory sensitivity were both related to easily visible changes in the EEG of high alpha subjects.

Also noteworthy was the good agreement of respiratory changes with changes in the more primary behavioral and EEG criteria. In view of their correlations with both passive lapse and EEG loss regardless of sex or alpha abundance, further exploration of their utility as substitute measures of sleep onset and vigilance performance is more than justified.

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