N74-10087

FINAL REPORT

SLEEP MECHANISMS: SLEEP DEPRIVATION

AND DETECTION OF CHANGING LEVELS OF CONSCIOUSNESS

NGR 05-020-576

A Research Project Supported by

The National Aeronautics and Space Administration

William C. Dement, M.D., Ph.D. Professor of Psychiatry Stanford University School of Medicine Jack D. Barchas, M.D. Associate Professor of Psychiatry Stanford University School of Medicine

CASE FILE COPY

William C. Dement, M.D., Ph.D.

h W Kanduz Jack D. Barchas, M.D.

For Stanford University

Introduction

The basis of the research program stemmed from the fact that there is currently no method of assessing the need to sleep and to make up for lost sleep. This research program attempted to develop information relevant to that issue using both physiological and behavioral measures. The work performed and initiated in this project could have application to that problem and could enable prediction of the need for further sleep.

The long-term goal was directed toward an understanding of the effect of sleep deprivation in a restricted environment. This type of study is of practical importance in that almost every situation where sleep loss must be sustained, the individuals are under some kinds of environmentallydetermined constraints. In nearly all laboratory studies, the interaction of a restricting environment sleep loss has not been investigated.

The studies were to have been divided into two phases. The first phase, conducted during the year which was funded, concentrated on examining basic parameters of sleep. The major effort was the development of the Stanford Sleepiness Scale and ways to derive further information relevant to determining sleepiness from the EEG. Part of this phase has involved establishing procedures to the occurrence of phasic REM events in wakefulness. In the second phase, for which funding was informally promised but not actually available, we hoped to assess applicability of the knowledge gained in Phase 1. Investigation of the Stanford Slow Tracing technique was to have been extended, as was work on PGO spikes in wakefulness. Central to that plan, Phase 2 of the study would have been the investigation of the effects of chronic limited sleep deprivation on man in terms of performance capability, which we believe would have had considerable relevance to NASA. The following is a brief description of the accomplishments of Phase 1. The research project terminated abruptly, preventing the planned accomplishments of Phase 2.

Brief Description of Research Accomplished During Phase 1

Cross-Validation of the Stanford Sleepiness Scale. In the past year, we have done detailed work in cross-validating the Stanford Sleepiness Scale (SSS). The SSS is a self-rating scale used to quantify the degree of sleepiness. We have attempted to determine the degree of association between scores on this scale and performance scorés on various mental tasks. Five college students served as subjects in a six-day experiment. All subjects completed SSS ratings each 15 minutes of their waking day. A brief test of memory and the Wilkinson Addition and Vigilance Test were administered daily. On the first three nights of the study, subjects were allowed eight hours in bed. On the fourth night, all subjects underwent sleep deprivation, and on the fifth night, subjects were allowed eight hours sleep. The SSS-Wilkinson Addition and Vigilance Test correlation was .68. When performance dropped off after sleep deprivation, correlations reached .80. The SSS correlated with scores on memory tests at a lower level (.47). In addition, we found that SSS ratings reflected the predictable fatigue during and after the sleep deprivation period. We now feel that the SSS represents a self-rating scale which sensitively and reliably indicates levels of sleepiness.

PGO Spikes and Phasic Events in Sleep and Wakefulness. Our progress in analyzing phasic activity in human sleep has progressed along two distinct lines. First, and most simply, we are now routinely recording phasic integrated potentials (PIPs) from the extraocular muscles in normal subjects and subjects with sleep pathologies such as narcolepsy, insomnia, and hypersomnia with periodic respiration. Preliminary data suggest that for the narcoleptic attack, PIPs occur during cataplexy and during the after-sleep onset REM periods in patterns not unlike those of normal REM sleep. Thus, in narcolepsy, it appears that some phasic generator is operating in a fashion similar. to that during REM sleep. The second direction utilizes pattern recognition computer programs. The strategy involves using our standard cat preparation and recording PGO spikes from the lateral geniculate nucleus along with EEG potentials from various sites on the surface of the brain. It may be possible to predict the geniculate spike record from an analysis of surface recordings only. To do this, a program has been devised to "learn" the average surface correlates of the geniculate waveform. It's then a simple matter to map the distribution of such surface correlates and thereby predict a posterior occurrence of a PGO spike. In the future, we believe it will be essential to try correlating analogous surface records in the human with the occurrence of the less-defined PIP and proceed in the same logical fashion to predict the occurrence of a phasic event from surface recordings alone. Ultimately, such a program might allow us to understand the interrelationship between

-2-

phasic events within the brain and alteration of performance.

Use of Stanford Slow Tracing Technique. The original Stanford Slow Tracing succeeded in opening the door to a new way of looking at all-night-sleep EEG data. Our initial technique, however, had several shortcomings. The ultraslow playback required unreasonable amounts of time and energy to output one final slowgram, and this final product resulted in no matrix of numbers on which quantification or comparison could be based. The original technique also dealt only with *peak* amplitudes which, from a theoretical point of view, is of questionable validity, and is certainly most subject to movement artifacts. Thus, the first step in the successful utilization of this new approach was to redesign the technique enabling the efficiency, accuracy, and versatility of computer hardware to be applied to the problem.

With the use of our small laboratory computer, a quick, reliable system for obtaining slowgram was developed, and, thus, Stanford Integrated Slowgram (SIS) was born. The raw EEG data is delivered to the machine from as many as four separate channels simultaneously, at off-line speeds of up to 30 times the real time (the data may be handled in real time if desired). The data is full-wave rectified and integrated for 10 second epochs. Thus, a data array is generated of double precision values, representing the total amplitude power for each 10-second period throughout the night. These values are stored on digital tape. To output a hard-copy slowgram, the operator inputs the epoch lengths of interest, the length of the complete record, and the desired axis and title labeling. Seconds later, a fully-labeled plot is completed by the Cal Comp plotter. The "epoch length of interest" is a new and important variable of the Stanford Integrated Slowgram. The input variable allows us to smooth our data and minimize the effects of artifact by allowing us to choose any multiple of 10 seconds for the length of the epoch which we want to be integrated and plotted. (We have routinely been using a 90second, i.e., each entry on the graph represents the total integrated value for a 90-second interval.) It is necessary to perform further work on techniques for subsequent analysis of a completed slowgram. Hemming, weighted smoothing, second order integration, curve fitting, and cursor techniques all need to be exploited.

We are currently analyzing the SIS's from four subjects who slept in the laboratory under baseline and sleep-deprived (36-hr deprivation) conditions.

-3-

While numerical analysis is not yet complete, it is clear that certain changes in the SIS from baseline to deprivation conditions are markedly consistent across subjects. The slope of the leading edge of the first slow-wave period is nearly double for all subjects on the post-deprivation night as compared with baseline supporting our original hypothesis that the rate of approach to peak amplitude will vary with pre-sleep tiredness. The decrement in peak amplitudes for successive cycles is much closer to a pure logarithmic function on post-deprivation nights as compared with baseline. All in all, the SIS pattern, under conditions of increased sleep pressure, appears to "regularize" the slowgram pattern, giving rise to a much smaller error when curve fitted against an artificially-generated, damped sinusoid.

It is certainly clear that our new technique is very sensitive to such manipulations as lengthening prior wakefulness. However, our notion of slowgram sensitivity to sleep-need has yet to be tested. Our new system enables us to readily test chronic long and short sleepers and output their slowgrams for comparison, and this is work that must be pursued in the future.