

THE EFFECT OF
PERCEPTUAL DEPRIVATION
ON THE HUMAN EEG

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ABSTRACT

The slowing of the alpha rhythm of the EEG during perceptual deprivation was investigated to test John Zubek's hypothesis that the time course of the slowing was influenced by the length of the deprivation period; to provide a more comprehensive description of the EEG changes produced by the deprivation procedure using computer power spectral analysis of the EEG; and to discover the effect of deprivation on the other rhythms of the EEG, chiefly the 12-14 Hz. sleep spindles since their mechanism of generation is reasonably well understood and it has been suggested that the alpha rhythm is generated in a similar manner.

The experimental subjects (eight adult humans) were divided into two groups of four, one group undergoing four days and the other seven days of perceptual deprivation. Two control groups of two subjects each experienced either four or seven days of confinement. All subjects slept in the laboratory for three nights before and three nights after the deprivation period. The EEG was recorded polygraphically throughout the deprivation period, while samples of EEG for computer analysis were recorded on magnetic tape at regular intervals throughout each day.

The power spectral analysis of the EEG enabled us to provide a good quantitative description of several characteristics of the normal human EEG which were previously unknown or inadequately described. We found that all subjects showed a regular circadian variation in alpha frequency with the lowest values occurring in the early morning and the high-

est values occurring in the afternoon or evening. Three subjects showed bimodal distributions of power in the alpha range, indicating two distinct frequencies of alpha. The slower alpha was more prominent in frontal regions while the faster alpha was more prominent in occipital regions. Two subjects possessed high amplitude alpha in the occipital region during Stage I REM sleep. This alpha was about 1 Hz. slower than the occipital alpha during the waking state. The slower frontal and REM alpha rhythms also showed a circadian variation in frequency. Three subjects showed a different frequency of alpha when the eyes were open than when the eyes were closed. Sleep spindles showed a bimodal power distribution, the slower frequency spindle occurring only in the frontal area while the higher frequency spindle was prominent in the frontal and parietal regions.

The alpha frequency of the deprivation subjects decreased by 1.5 to 2 Hz. while the control subjects showed little or no change in alpha frequency. The time course of the decrease in alpha frequency was similar in the four and seven day deprivation subjects, disconfirming Zubek's hypothesis that the seven day group would show a more gradual slowing. Subjects with high amplitude alpha showed a decrease in alpha amplitude while subjects with low amplitude alpha showed no change or an increase in alpha amplitude over the deprivation period. The low frequency frontal alpha was also slowed during deprivation but showed no reduction in power. The theta rhythm showed some increase in power but no change in frequency over deprivation in two of the four subjects who showed measur-

able amounts of this activity. The REM alpha was slowed to the same degree as the waking alpha remaining 1 Hz. slower throughout the deprivation period. In contrast, the frequency and amplitude of the sleep spindles were unaffected by the deprivation procedure and there appeared to be no regular circadian variation in spindle frequency. This suggests that current assumptions that alpha and spindles share a common mechanism may not be valid.

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Table of Contents

	Page
INTRODUCTION	1
BACKGROUND	4
PROCEDURE	57
RESULTS	66
DISCUSSION	83
SUMMARY	98
FIGURES	100
TABLES	192
BIBLIOGRAPHY	198

List of Figures

	Page
Figure 1. The deprivation chamber, showing the subject wearing the translucent mask and cardboard tubes during deprivation.	100
Figure 2. Frequency response curves of Grass Model IV S polygraph and the filter used prior to digitization of the EEG signal.	102
Figure 3. A sample of the EEG recorded from subject P. W. on the morning of the first pre-deprivation night.	103
Figure 4. A sample of the EEG recorded from subject P. W. on the morning of the seventh day of deprivation.	105
Figure 5. Power spectra produced from 1 minute of EEG from the occipital lead in each of the preceding examples (Figures 3 and 4).	107,
Figure 6. Modal alpha frequency of subject P. W. during the eyes closed condition.	109
Figure 7. The modal power of the occipital alpha rhythm of subject P. W. in the eyes closed condition.	111
Figure 8. The modal alpha frequency of the four-day and seven-day experimental groups, taken from the occipital lead in the eyes closed condition.	113
Figure 9. The modal power of the occipital alpha of the four-day and seven-day experimental groups during the eyes closed condition.	115
Figure 10. A sample of the EEG of subject R. G. recorded on the evening of the third pre-deprivation night. The subject's eyes are closed.	117

	Page.
Figure 11. A sample of the EEG of subject R. G. recorded on the afternoon of the fourth deprivation day. The subject's eyes are closed.	119
Figure 12. The modal frequency of the occipital alpha of the four-day and seven-day experimental subjects in the eyes open condition.	121
Figure 13. The modal power of the occipital alpha of the four-day and seven-day experimental subjects in the eyes open condition.	123
Figure 14. The weighted average frequency of the occipital alpha in the range ± 2 Hz. from the mode of the alpha peak in the power spectrum, in the eyes closed condition.	125
Figure 15. The total power in the spectrum in the range ± 2 Hz. from the mode of the alpha peak, in the eyes closed condition.	127
Figure 16. The weighted average frequency of the occipital alpha in the range ± 2 Hz. from the mode of the alpha peak in the power spectrum, in the eyes open condition.	129
Figure 17. The total power in the spectrum in the range ± 2 Hz. from the mode of the alpha peak, in the eyes open condition.	131
Figure 18. The variance of the distribution of power in the range ± 2 Hz. from the mode of the occipital alpha peak in the power spectrum, in the eyes closed condition.	133
Figure 19. The variance of the distribution of power in the range ± 2 Hz. from the occipital alpha peak in the power spectrum, in the eyes open condition.	135
Figure 20. Power spectra produced from 1 minute of the sample of EEG shown in Figure 3.	137

	Page
Figure 21. The modal frequency of the occipital and frontal alpha from subjects W. P. and P. W.	139
Figure 22. The power of the mode of the alpha peak in spectra of the frontal EEG in subjects W. P. and P. W.	141
Figure 23. Examples of the occipital EEG, eye movements and EMG during a REM period of subject W. P. on the first pre-deprivation night and on the fourth deprivation night.	143
Figure 24. Power spectra of 1 minute of the EEG samples shown in Figure 23.	145
Figure 25. The modal frequency of the occipital alpha during waking and during REM periods for experimental subject W. P. and control subject W. K.	147
Figure 26 a and b. Examples of power spectra of the frontal, central, temporal, parietal, and occipital EEG of subject W. P. containing peaks in the theta range.	149
Figure 27. The modal frequency of the theta peak in the frontal EEG of the four-day and seven-day experimental subjects.	152
Figure 28. The power of the mode of the theta peak in the frontal EEG of the four-day and seven-day experimental subjects.	154
Figure 29. A sample of EEG from experimental subject D. G. recorded during Stage 2 sleep on the third pre-deprivation night.	156
Figure 30. A sample of EEG from the experimental subject D. G. recorded during Stage 2 sleep on the seventh night of deprivation.	158
Figure 31. Power spectra computed from 1 minute of the frontal and parietal Stage 2 EEG shown in Figures 29 and 30.	160

	Page
Figure 32. The modal frequency of the low frequency frontal and high frequency parietal spindles occurring during Stage 2 sleep in the experimental subjects.	162
Figure 33. The power of the mode of the low frequency frontal spindle occurring during Stage 2 sleep in the experimental subjects.	164
Figure 34. The power of the mode of the high frequency parietal spindle occurring during Stage 2 in the experimental subjects.	166
Figure 35. The modal frequency of the occipital alpha of the four-day and seven-day control subjects. Each point represents the average of the modes from two spectra.	168
Figure 36. The power of the mode of the occipital alpha of the four-day and seven-day control subjects.	170
Figure 37. The weighted average frequency of the occipital alpha of the four-day and seven-day control subjects calculated over the range ± 2 Hz. from the mode of the alpha peak.	172
Figure 38. The total power in the occipital alpha peak of the four-day and seven-day control subjects calculated over the range ± 2 Hz. from the mode.	174
Figure 39. The variance of the peak in the power spectrum of the occipital alpha of the control subjects calculated over the range ± 2 Hz. from the mode.	176
Figure 40. Oral body temperature of the control subjects measured immediately before the alpha recordings.	178
Figure 41. EOG amplitude, total power in the spectrum of the occipital EEG over the range 8-13 Hz., and power of the mode of the alpha peak in the spectrum, plotted as a function of time, for three subjects.	180

	Page
Figure 42. A scatter plot showing the relationship between pre-deprivation modal alpha frequency and the magnitude of alpha shift (Δ Hz.) over deprivation.	182
Figure 43. A power spectrum of the occipital EEG of subject W. K. recorded during REM sleep.	184
Figure 44. A sample of the EEG of subject M. R. recorded during the eyes closed condition on the evening of the second pre-deprivation night. Continuous alpha activity and rapid eye movements or eye fixation indicate that the subject is alert.	186
Figure 45. A sample of the EEG of subject M. R. recorded seven minutes after the sample shown in Figure 44. The subject's eyes are closed.	188
Figure 46. Power spectra of the occipital EEG shown in Figures 44 and 45.	190

List of Tables

	Page
Table 1. The correlation between body temperature and alpha frequency measurements.	192
Table 2. The change in modal (Mode) and average (± 2 Hz.) alpha frequency in the four-day deprivation subjects.	194
Table 3. The change in modal and average alpha frequency over deprivation for the seven-day subjects.	196

INTRODUCTION

The continuous interaction of the brain with the environment in the form of sensory input and motor output which modifies sensory input was proposed by D. O. Hebb in 1949 as being the foundation of perceptual and cognitive processes. The disruption and distortion of these processes by drastically limiting the interaction with the environment for a number of days was confirmed by experiments conducted in 1956 by Bexton, Heron, and Scott. Their subjects reported hallucinations and difficulty in maintaining organized trains of thought during the deprivation period, and experienced perceptual distortion afterward. This restriction of sensory input also appeared to have neurophysiological effects, slowing the frequency of the alpha rhythm of the electroencephalogram (EEG). These changes in alpha frequency have remained puzzling, since the alpha frequency is generally quite stable and difficult to alter by other manipulations. Furthermore, the changes persist for a number of days following the deprivation period.

Since the very first recording of the electrical activity of the human brain, the alpha rhythm (a prominent sinusoidal waveform of about 10 Hz) is still an enigma. The mechanism of its generation remains

unknown, since, despite great advances in the understanding of the generation of electrical potentials in the brains of lower animals, a reliable analogue of alpha does not appear in such preparations. Its relationship to normal brain function remains obscure, since alpha in some subjects is a high amplitude (150 μ v.) rhythm of almost unbroken regularity, while in others it is difficult to pick out two or three regular waves in sequence which are distinguishable from the desynchronized background activity, yet behavioral differences between such subjects have been difficult to verify.

Alpha appears to be present at intermediate levels of arousal or stimulation, being blocked by patterned visual input or sudden stimulation in other modalities and disappearing under conditions of drowsiness. The frequency of the rhythm has a wide range across the normal population (8-13 Hz.), but an individual's alpha frequency is highly stable. Individual differences in frequency appear to be related in some way to basal metabolic rate. Two reliable methods of inducing a rise in alpha frequency are administration of thyroid hormone and raising body temperature.

As mentioned above, a third manipulation, sensory deprivation, has been shown to produce reliable decreases in alpha frequency which persist for as much as a week following the deprivation period. John Zubek has proposed that the time course of this decrease in alpha

frequency is dependent on the subject's expectation of the length of time to be spent in deprivation. He found that subjects who had contracted to do seven days of deprivation had a greater depression of alpha frequency at the end of the week than subjects who had contracted to do two weeks of deprivation, although the two-week subjects reached a lower final level than the one-week subjects. He has also shown that one, four, or seven days of deprivation all produce roughly equal shifts in alpha frequency.

The object of the present study was, firstly, to study carefully the time course of the change in alpha frequency throughout the deprivation period, as this has not previously been adequately described. Four and seven-day deprivation periods were used to test the hypothesis that the depression of alpha frequency would be more gradual in the seven-day group. Secondly, it was expected that power spectral analysis of the EEG would enable a more complete description of the changes being induced by deprivation. Thirdly, in view of the fact that the best documented model of the generation of alpha rhythms has been developed solely from the study of sleep spindles in cats, it was of interest to discover if deprivation produced any effect on sleep spindles in humans.

BACKGROUND

This investigation was conducted to study the effects of perceptual deprivation on the rhythmic activity in human EEG (the alpha rhythm and sleep spindles). Before examining the existing literature on this subject, it is useful to review the studies which have described the form and characteristics of these rhythms and the mechanisms which have been proposed to explain their generation.

Berger's Discovery

The study of the electrical activity of the human brain began with a remarkable series of experiments by Hans Berger. His initial experiments were done in 1924, yet he did not publish any of his work until 1929, when he had satisfied himself that the electrical activity he recorded was actually produced by the brain. He christened this brain activity the "Elektrenkephalogram" and distinguished two such patterns of activity: the alpha rhythm, a regular sinusoidal oscillation with a frequency of about 10 Hz., and the beta rhythm, with a frequency roughly twice that of alpha, although not an harmonic.

He found that the alpha was blocked by eye opening, or by sudden stimulation in any modality (Berger, 1930). However, he realized that alpha blocking could not be solely due to sensory stimulation, since non-visual alpha blocking tended to habituate, and alpha could be blocked by

intense mental activity. In fact, Berger stressed that the subjects had to be relaxed in order to obtain a successful recording of the alpha rhythm.

His proposed mechanism of alpha blocking was exactly the opposite of any suggested since. He thought that sensory stimulation should induce an increase in alpha activity in the local cortical receiving area and produce a widespread inhibition of the surrounding cortex. Berger believed that all the sensory receiving areas were buried in the sulci and, therefore, their electrical activity was not recordable at the surface. Consequently, the inhibitory action was all that was observable. Similarly, during active mentation, he thought the shifting patchwork of alpha activity remained confined to small areas at any one moment, the inhibitory surround thus dominating the recording.

Berger was always conscious of the possibility that artifacts (i.e., non-brain activity) might be contaminating his records, and many of his experiments were designed to show that the electrical activity from the scalp was not due to heart beat, breathing, brain pulsations, eye movements, or muscle tremor (Berger, 1929). He also claimed that alpha could be recorded from the cortical surface but not from needles 7 cm. apart and 4 cm. deep in the white matter, proving that the alpha rhythm originated in the cortex (Berger, 1931). However, simultaneous records from the two sites are not shown. In the course of his work with a large

number of normal and clinical subjects, young and old, he noted nearly every important aspect of the EEG, including its ontogeny, its reaction to various drugs, and its appearance in a wide variety of clinical states, including epilepsy. The one major oversight was an inadequate description of the EEG changes in sleep.

Adrian and Matthews (1934a) were the first to confirm Berger's discovery. They satisfied themselves that the alpha rhythm was not due to eye movements, as they could record no potentials in the occipital region that were related to active or passive eye rotations; and they noted that potentials due to eye movements should be maximal close to the eye, whereas alpha is maximal in the occipital region. However, they believed the best proof of the brain origin of alpha was the demonstration that alpha amplitude was maximal over a trephine hole in the skull. The muscles of the head and neck were eliminated as a source of alpha potentials, since alpha appeared to depend only on opening and closing the eyes and not on the position of the head or contraction of the head and neck muscles. These considerations, along with Berger's careful controls, appeared to leave the brain as the only remaining source of these potentials.

The Distribution of Alpha

One of the first controversies in the alpha literature concerned the

distribution of alpha over the head. Berger believed the potential to be widespread, whereas Adrian and Matthews(1934a) stated that maximal amplitude of the alpha rhythm could only be obtained by placing one electrode in the occipital region. Adrian and Yamagiwa (1935) verified the importance of the occipital region in the generation of alpha with the observation of a phase reversal in the records obtained from a chain of bipolar electrodes spanning the occiput. Using this phase reversal technique, Lindsley(1936a) managed to find four foci of alpha in each hemisphere: two in the occipital region, one in the central region and one in the temporal region. Since their recording apparatus had only two channels, they were unable to show that these four foci existed simultaneously.

Jasper(1936b) and Jasper and Andrews (1936, 1938) claimed that the EEG of the occipital and central regions were characteristically different, the occipital region being dominated by the alpha activity and having little rhythmic beta, while the central region had less alpha and more beta. It was also stated that the alpha could occur independently in the two areas and that the central alpha frequency could be different from the occipital one. Unfortunately, like many observations of that period, these claims were unsubstantiated with any quantitative data.

Rubin(1938b) examined the distribution over the head of the percent of time alpha was present in the EEG record (percent time alpha). Since

an amplitude criterion is necessarily used in judging the presence of alpha, there will be some relation between the amplitude of the rhythm and percent time alpha. Rubin demonstrated that percent time alpha is distributed over the head in much the same way as amplitude of alpha when bipolar recordings are used (i.e., a large maximum occurs in the occipital region, and there is a sharp monotonic decline as one approaches the front of the head). However, monopolar recordings show a more uniform distribution of percent time alpha and, in fact, a frontal as well as an occipital maximum may appear. It should be noted that such a maximum is not incompatible with a lower amplitude of frontal alpha; it merely demonstrates that when frontal alpha exceeds the minimum amplitude criterion, it can appear as often as occipital alpha. There thus appears to be general agreement that alpha can be recorded from anywhere on the scalp, but the highest amplitude potentials occur in the occipital regions.

Bilateral Synchrony

One simple question about alpha which has not been answered unequivocally, is whether the alpha rhythm is synchronous in homologous points of each hemisphere. Adrian and Matthews (1934) found that left and right parietal electrodes referred to the occiput (presumably midline) yielded records which were almost identical. They concluded that the hemispheres beat in phase, overlooking the fact that large amplitude

activity under the common midline electrode could obscure differences between the parietal regions.

Jasper and Carmichael (1935) stated that the EEG from the two sides of the head was typically in phase and of the same frequency, and Jasper claims "Normal persons present practically the same electroencephalogram from bilaterally homologous regions" (Jasper, 1936, p. 1132). Neither report mentions the recording configuration. In a more detailed treatment of the question, Jasper and Andrews (1936) reported that practically identical in-phase recordings could be obtained if one referred two homologously placed occipital electrodes to a common electrode on the forehead. However, they could also record alpha by referring one occipital electrode to the other, implying that these placements were not equipotential. The explanation for this discrepancy was thought to be that the forehead-occiput derivation recorded diffuse potentials from the whole head, eliminating any possibility of differentiating between the potentials in the two occipital regions. When they placed a midline electrode on theinion and recorded from points 2 cm. on either side, they reported that they could see no constant phase relationship, the two potentials being 180° out of phase as often as in phase, a result seemingly contradictory to that found by Adrian and Matthews (1934). In any case, they have ignored the possibility that the rhythmic potential oscillations on the two sides of the head may be loosely coupled;

i.e., no perfect or constant phase relationship may exist between the two rhythms, yet on the average they tend to beat in synchrony. The rhythms may even briefly become uncoupled. Careful quantitative analysis of long segments of record is necessary to test this hypothesis. The recording of alpha between two homologous points may merely be the reflection of the differential between slightly out of phase but synchronized oscillations in each hemisphere.

Cohn (1948), referring a midline occipital electrode to two bilaterally symmetrical placements at the back of the head, was unable to show any constant phase relationship between the hemispheres, although no quantitative measurements were made. Rubin (1938a) favored the idea that the hemispheres were independent, since he reported that bursts of alpha could occur independently in the two hemispheres in the occipital region. He found, however, that the alpha bursts had a much greater tendency to be symmetrical in the frontal region. He does not mention the electrode derivations which display this phenomenon.

Walsh (1958), using an automatic correlator which integrated the in-phase components and subtracted the out-of-phase components over a period of three to four seconds, analyzed the recordings from a transverse (coronal) chain of bipolar electrodes, and although he found phase reversals on either side of the head, indicating bilateral foci of activity, no consistent phase relationship could be seen between the two sides.

However, only a few examples are given and no attempt was made to quantify the data, so it is difficult to evaluate.

Garoutte and Aird (1958) appear to be the first to do any quantitative measurement of the phase relationship between the hemispheres. They measured the time differences between the peaks of alpha waves from the two hemispheres and found a "... high proportion of bilateral synchrony (about 50 percent within ± 1 msec., 75 percent within ± 10 msec.) and the apparent tendency for the pairs of waves when asynchronous to return toward synchrony. . . ." (Garoutte and Aird, 1958, p. 263). They also found that four subjects exhibited periods of several seconds when the waves were 180° out of phase. Unfortunately, they neglect to mention the electrode positions or recording configuration.

Significant bitemporal out-of-phase activity at a frequency of about 8 Hz. occurring during mental arithmetic (the "kappa" rhythm) was measured by Brazier and Casby (1952) using a cross-correlation of five minutes of EEG record from each temporal lobe referred to the chin.

Walter, et al. (1966) present a complex mathematical analysis of the bipolar EEG from C3 - P3, P3 - O1, P4 - O2, C4 - P4, and O1 - O2. The analysis shows a high degree of coherence (a mathematical term expressing the predictability of one recording from another given linear transformations of phase and gain) between homologous regions; i.e., symmetrical bipolar pairs. However, the alpha recorded bi-occipitally

(O1 - O2) is not coherent with any other channel. They interpret this result to mean that there must be at least three generation processes, one on either side of the head which exhibit mutual coupling, and a third unrelated process (not a linear combination of the first two) perpendicularly oriented. Let us suppose, however, that the third signal is in fact a linear combination of the signals from each hemisphere, in fact, the simple difference. If two sine waves are separated by a phase lag of 45° (12.5 msec. in the case of the alpha rhythm) the difference between them is also a sine wave of the same frequency and nearly the same amplitude. If the phase relationship between the first two signals was constant, the third signal would be perfectly predictable from the first two and, therefore, would be perfectly coherent with them. However, if the phase relationship between the first two signals was not constant (e.g., the frequency of the two signals was not perfectly stationary and the oscillators were loosely coupled), one signal now leading, now lagging the other in an unpredictable fashion, then the third signal would not be predictable from the other two over a long interval of time. The interval of analysis used in calculating the coherence in this study was 12 seconds. This would appear to be ample time for a number of phase shifts to occur, reducing the coherence of the b1-occipital recording to a low value.

They found that coherence between symmetrical leads was high

during high amplitude alpha and low during low amplitude alpha, which they say shows that the alpha process is superimposed on incoherent background activity. It may also indicate that the signals are more weakly coupled at low amplitude. They remark that the coherence was often high over the whole alpha band (as much as 2 Hz. from the peak spectral intensity) although the power varied by a factor of 10 over this range. This implies that a single alpha generation process covers the whole alpha bandwidth. However, the constancy of the coherence over the alpha band also implies that the incoherent part of the signal has the same power spectral density distribution as the alpha, since the proportion of incoherent activity is constant. This makes sense if one attributes the incoherence to loose coupling rather than to background noise. Some caution must be exercised in the interpretation of coherence measures since low values of coherence may be produced by non-linear relationships between the signals, by the signals being dependent on multiple processes, or by a lack of stationarity in the signals (Glassman, 1972).

Remond, et al. (1969) have developed a technique of averaging alpha activity by triggering data collection when a wave of the proper amplitude, polarity, and duration occurs, which identifies it as an alpha wave. If one second of data is collected following the trigger, a train of 10 alpha waves will usually be recorded. Summation of a number of such trains generates the alpha average. If recordings are made

simultaneously from a number of points over the head, a spatio-temporal map of the alpha average may be derived. Unfortunately, Remond uses a chain of bipolar electrodes arranged in either a sagittal (midline) or coronal (transverse) plane, the "active" electrode of one channel becoming the "reference" of the next. Thus, in-phase potentials recorded in adjacent channels indicates that a continuous gradient of potential exists along the chain, each electrode being equally more positive (or negative) than its neighbor. In fact, Remond's spatio-temporal topographic maps plot contours of equal gradient of potential, which makes it difficult to visualize how the potential itself is distributed. It appears from Remond's diagrams that in the transverse montage, the polarity of the amplifiers is not reversed on either side of the midline. Thus, if the lateral electrodes become more negative than the medial ones (as one would expect if the EEG on the two sides of the head was synchronous), it would be recorded as an upward deflection in one hemisphere and a downward one in the other. Remond's spatio-temporal maps of the alpha average consistently show a phase reversal across the midline. This should be interpreted to mean that the average alpha rhythm is synchronous and in phase in the two hemispheres.

The bilateral synchrony of the alpha has also been confirmed by Hoovey, et al. (1972). They recorded alpha from O1 and O2 using the nose as a reference and found that the difference between alpha wave

peaks in the right and left hemisphere averaged 2.5 msec. They also reported that a narrow variation in this measure (i.e., a close phase relationship) was associated with a high correlation between the amplitudes of the waves from the two hemispheres. Thus, it appears that a close coupling of phase is associated with a close coupling of gain in the two rhythms.

In summary, it appears that the alpha rhythms in the two hemispheres are loosely coupled. That is, on the average over long periods the waves are in phase, although one rhythm may lead or lag the other as much as 10 msec. Brief periods exist when the rhythms become uncoupled and phase reversals may be seen.

Topographic Analysis

Ideally, a topographic study of the EEG should show a series of equipotential contour maps of the scalp at successive instants in time. This requires the use of some type of reference to which the potential at each electrode may be compared. The obvious deficiency of such a system is that no "inactive" reference exists and any potential variation at the reference electrode will appear on all channels, or will cancel identical potential changes in other placements. However, for the purposes of topography, this appears to be a better system than bipolar recordings from which it is difficult or impossible to infer the distribution of potential at any instant.

The use of the ~~Walter~~ toposcope by Cooper and Mundy-Castle (1960) emphasizes this difficulty, quite different results being obtained from the two recording techniques. One of their clearer results using monopolar recordings showed the alpha to be an anterior-posterior wave: when the frontal electrodes were positive, the posterior ones were negative and vice versa. A similar pattern was shown by Remond, et al. (1969) using an anterior-posterior chain of bipolar electrodes. Although the most anterior electrode was only as far forward as the vertex, a reversal of phase appears in the parietal region. Unfortunately, with bipolar electrodes this could be due either to the spanning of an actual phase reversal (i.e., a node of a standing wave), or to the spanning of a region of maximum potential.

However, the concept of the alpha rhythm as an anterior-posterior standing wave with a node in the parietal area has been substantiated by Lehmann (1971). The potential over the head was measured simultaneously from an array of 38 to 45 electrodes referred to the average of all the electrodes. Equipotential contour maps of the scalp were produced at 8 msec. intervals. It could be seen that at the peak of the alpha cycle (the point at which there is greatest "relief" in the map, or the greatest potential difference between regions), the head is maximally polarized in an anterior-posterior fashion. The line of zero potential appears to run from ear to ear through the mid-parietal region. The

potential difference between the front and back begins to decline until one-quarter of the way through the alpha cycle when the whole head becomes almost equipotential. At this point, the polarity of the front and back of the head reverses, sometimes through an apparent rapid rotation of the field during which the head may be slightly polarized in a left-right fashion. The anterior-posterior polarization then builds up to a maximum, declines, and reverses once again, reestablishing the initial polarity, and when maximum polarization is reached, the alpha cycle is completed.

There are three regions where maximum polarization is reached: the left and right occipital areas, and a strip along the midline from the central (vertex) area to the most anterior placements (F_z). In five subjects, the frontal maximum appeared most often at F_z but occasionally bilateral frontal maxima were seen. In general, the occipital regions do not reach maximum polarization at exactly the same time, the phase lags in the examples shown vary from +20 msec. to -10 msec. If the potentials on the head were being produced by two anterior-posterior generators, one in each hemisphere, one would predict that a slight phase lag between the generators would produce a diagonally oriented "checkerboard" potential distribution around the time of the polarity reversal since, if one side reversed potential slightly in advance of the other, the left front and right rear regions would be similarly

polarized for a short period of time. One can see from Lehmann's diagram (Figure 3, p. 441, Lehmann, 1971) that this does in fact happen.

In summary, it appears from topographical studies that the two loosely coupled alpha generators produce an anterior-posterior polarization of the head which oscillates as a quasi-standing wave. One must say "quasi-standing wave" since the position of the node is probably not stationary.

Frequency Analysis

Investigators measuring the frequency of the alpha were at first merely concerned with establishing the normal limits of variability, both within and between subjects. Frequency was most often measured by the simple, although rather crude, expedient of counting the number of alpha waves occurring in a given time period (usually one second).

The first such report (Jasper and Carmichael, 1935) stated that the normal adult alpha frequency varied between 8 and 12 Hz. Presumably, they were referring to the range across individuals, since Jasper (1936b) stated that the outside limit of variation of individual alpha frequency was 1 to 2 Hz. Lemere (1936) repeated frequency measurements (presumably wave counts) on 26 subjects three to five times, finding that only five subjects showed a different frequency on different trials, this difference never being greater than 1 Hz. Individual alpha

frequencies varied from 9 to 12 Hz., and there appeared to be no correlation between alpha amplitude and frequency. Jasper and Andrews (1936) and Lemere (1937) both report the alpha frequency to be equal on the two sides of the head in normals. Rubin (1938b) reported that individual alpha frequencies exhibited a daily variation of 5 to 10%.

Rubin (1938a) found that in some individuals the frontal alpha could be 1 to 2 Hz. slower than the occipital alpha. This slow frontal alpha was also blocked by eye opening. Jasper and Andrews (1938) also noted a difference in alpha frequency between the precentral and occipital regions in some subjects. They found the average variation in alpha frequency over days was 6.8% in 14 subjects with 6 subjects having a variation of only 1 or 2%. They also demonstrated a 10 to 20% rise in alpha frequency in three subjects whose body temperature was raised 5 or 6 degrees F. by inducing fever with typhoid vaccine. This corroborates an earlier observation by Hoagland (1936) that raising the temperature of general paresis patients by diathermy also raises their alpha frequency.

Frequency analyzer power spectra of monopolar EEG from frontal, parietal, and occipital regions (Johnson and Ulett, 1959) shows that the alpha in frontal and parietal leads appears to be slower than the occipital alpha by 1 to 2 Hz. Since the only data presented is an average of 35 subjects, it is impossible to say whether this pattern was characteristic

certain individuals or whether it was a general phenomenon.

A study of the EEG characteristics of 500 normal adults was published in 1944 (Brazier and Finesinger, 1944). No details were given of the recording montage or whether the subjects' eyes were open or not. The number of waves of each frequency in a two-minute sample of EEG were added up and a histogram was compiled for each subject of the percentage of time each frequency was present. The mode of this distribution was taken to be the dominant frequency. The mean dominant frequency of the group was 10.5 Hz. with an S.D. of 0.9 Hz. In 94% of the subjects, the dominant frequency was between 8 and 13 Hz. The rest showed dominant frequencies scattered over the higher frequencies. Forty-five subjects had four or five repeated measures made on the same day. The mean variation of dominant frequency was less than 1%.

Percent time alpha was defined as the percent of time occupied by waves between 8 and 13 Hz. The mean percent time alpha was 61%, with 48% of the subjects having a percent time alpha between 50 and 75%, and only 5% of the subjects having less than 25%. They found a relationship between alpha frequency, percent time alpha, and maximum alpha amplitude as shown below:

	<u>Maximum Amplitude (A)</u>	<u>Percent Time Alpha</u>	<u>Mean Dominant Frequency</u>
N = 10	A < 25 μ v.	40.3%	11.0 Hz.
N = 95	25 μ v. < A < 50 μ v.	48.4%	11.0 Hz.
N = 274	50 μ v. < A < 100 μ v.	65.4%	10.4 Hz.
N = 95	A > 100 μ v.	73.6%	10.0 Hz.

(Brazier and Finesinger, 1944, p. 309).

Thus, one can say that people with high amplitude alpha tend to have high percent time alpha and low alpha frequency.

There appears to be only one report of a circadian variation in alpha frequency. Bjerner (1949), in the course of studying reaction times while sleep depriving subjects over night, noticed that alpha frequency fell steadily during the night, reaching a minimum around 6 to 8 a.m., after which it began to rise again. Measurements were discontinued at 10 a.m. Since at the time it was not realized that this overnight fall in alpha frequency could also occur while the subject slept, Bjerner's data could have been interpreted as merely reflecting the subject's drowsiness.

Another frequency characteristic of the alpha is the so-called "squeak" phenomenon. It has been reported by several investigators (Jasper, 1936a; Lindsley, 1938a; Cooper and Mundy-Castle, 1960; and

Kawabata, 1973) that the frequency of the first second of alpha following a visual alpha block is higher (as much as 15%) than the baseline alpha frequency taken some time later.

It appears that individual differences in alpha frequency may in some way be related to basal metabolic rate. Lindsley and Rubenstein (1937) reported a correlation of 0.90 between total calories per hour and alpha frequency in 13 subjects. No significant relationship was found between alpha frequency and body temperature, although one must accept this with reservation since the temperature was measured before getting out of bed and the alpha frequency was not measured until a few hours later. Ross and Schwab (1939) attempted to replicate this finding in hypo- and hyperthyroid patients during treatment. They found that the group as a whole showed a correlation of 0.668 between alpha frequency and total calories per hour, but a better correlation ($r = 0.883$) was obtained in subjects in non-basal (non-fasting) condition than in the basal condition ($r = 0.392$ and 0.421). The reason for this discrepancy is not clear. The best relationship was found for the myxedemic group ($r = 0.9$), the hyperthyroids having a correlation of 0.51.

Herman and Quarton (1964) found a significant correlation between alpha frequency and initial measurement of basal metabolic rate ($r = 0.73$) and protein bound iodine ($r = 0.60$) and between change in alpha frequency and change in basal metabolic rate ($r = 0.85$) and protein bound iodine

($r = 0.83$) in treatment of hypo- and hyperthyroid patients. Further evidence that alpha rhythm may be related to metabolic rate comes from the finding of Brazier, et al. (1944) that lowering blood sugar levels with insulin injections caused a 0.56 Hz. decrease in alpha frequency in 16 subjects.

It is not known what rôle body temperature plays in the determination of alpha frequency. Certainly body temperature and metabolic rate are closely related, both showing similar overnight drops (Kreider, et al., 1958), and myxedema may also be associated with low body temperature. The rate of change of alpha rhythm with body temperature shown by Jasper and Andrews (1938) would predict that a circadian variation of body temperature of 2°F . would produce a concomitant variation of alpha frequency of 0.75 Hz. However, it is clear that individual differences in body temperature cannot account for individual differences in alpha frequency. The normal range of daytime body temperature is only 2°F . and the distribution has a standard deviation of 0.4°F . (Ivy, 1944), while the alpha frequency has a range of 5 Hz. and a standard deviation of 0.9 Hz.

Ontogeny of the Alpha Rhythm

The development of the alpha rhythm was first studied by Berger, who found the rhythm to be generally slower in children. Lindsley (1936)

published a preliminary report of a cross-sectional study showing that alpha is first seen in infants at about four months of age, the average frequency being about 4 Hz. The frequency gradually increases with age, reaching 8 Hz. by age three or four and 10 Hz. (the adult average) by age eight. Increasing the number of subjects in each age group (Lindsley, 1938a) only served to substantiate this picture. In addition, alpha blocking time was shown to decrease from 0.54 seconds at age six months to 0.30 seconds at age 12 years or older. Taking the frequency change into account, this indicates that roughly equal numbers of alpha waves are produced before an alpha block (2 1/2 at age six months; 3 at age 12). Although it was claimed that there was no consistent change in percent time alpha, a general decreasing trend was apparent. Alpha amplitude was found to increase to a maximum at one to two years of age, and thereafter shows an exponential decline (Lindsley, 1939).

Cross-sectional study of 200 children by Bernhard and Skoglund (1939), 60 of whom were followed up 10 years later by Larsson, et al. (1949), substantiate Lindsley's description of the alpha frequency development.

Smith's serial and cross-sectional observations on the development of the EEG confirm Lindsley's data (Smith, 1938b). However, he has shown a markedly different ontogenetic development of occipital and pre-central alpha (Smith, 1939; 1941). Whereas the occipital alpha begins

at 4 Hz. at age four months and increases rapidly in frequency over the first year to about 6 Hz., the precentral alpha occurs shortly after birth at 7 Hz. and shows no frequency change over the first year. Thereafter its frequency begins to increase in parallel with the occipital alpha, a 1-Hz. difference being maintained until about age four, when the frequencies begin to converge. By age eight there is little difference in frequency of the two rhythms, and following this age the occipital rhythm was sometimes seen to be faster. It is possible that this precentral rhythm may be related to the so-called mu or "wicket" rhythm which Chatrian et al. (1959) claims is closely related to skeletal motor output.

Although Brazier and Finesinger (1944) were unable to find any relation between alpha frequency and age in adults up to age 45, Obrist (1954) and Frielander (1958) have shown that the mean alpha frequency of the population progressively declines beyond this age.

Ontogeny of Sleep Spindles

In contrast to this well-defined developmental increase in alpha frequency, the sleep spindles are found to have their onset at birth with the normal adult frequency of 12 to 14 Hz. (Smith, 1938a; b). Schulz, et al. (1968) report that by eight to ten weeks of age spindles are seen in 37% of 20 second epochs of quiet sleep, and at 12 to 13 weeks in 78%. This development has been well described by Metcalf (1969) in normal

and premature infants. He reports that "pre-spindle" activity (more variable in frequency and very low amplitude) appears at about four to seven weeks of age. One to two weeks later normal spindles are fully developed. In premature infants, this development is accelerated by about four weeks.

It is interesting to note that the ontogeny of the EEG in the monkey *Macaca Mulatta* exactly parallels that of the human, with a considerably shortened time scale (Caveness, 1962). The alpha may be seen in the first week of life and is initially at a frequency of about 4 Hz. A rapid exponential rise in frequency occurs in the first year, the adult frequency being reached at about nine or ten months of age. As in the human, a rapid increase in amplitude occurs over the first two months, followed by a progressive decline until two years of age. In contrast, the spindles are present in the neonate with the usual adult frequency.

EEG Mechanism

Having reviewed some of the normative and descriptive characteristics of the human EEG we will now consider some of the neurophysiological mechanisms which have been proposed to account for these gross potential variations which can be recorded from the scalp. Two fundamental problems are posed by the EEG: (1) the origin of the electrical potential, and (2) the mechanism of its rhythmicity.

Adrian and Matthews had already addressed themselves to the first problem in 1934 in an analysis of injury discharges in the cortex. They observed that if a short (1 mm.) shallow (1/2 mm.) cut is made in the cortex near one of two electrodes on the cortical surface, a burst of regular high frequency waves (35 to 90 Hz.) is induced which may last for as long as half a minute. These waves begin as small monophasic spikes of about 10 msec. duration which broaden and increase in amplitude until a sinusoidal rhythm is produced. Frequency and amplitude then gradually decline until the response disappears. They interpreted this sequence of events as a spreading of the response to larger numbers of neurons discharging in near synchrony. They believed that the initial monophasic spikes of the discharge were the responses of individual neurons and that these spikes normally summated to produce slow potential changes in the cortex. Thus, they argued that these slow waves are composites of all-or-none spikes of constant amplitude and duration, although they conceded that these spikes had a much longer time course than those of peripheral neurons.

In 1936 the Cold Spring Harbour Symposium was devoted to neurophysiology. These papers and discussions provide an excellent picture of the range of theories and hypotheses which were being proposed at that time to account for EEG phenomena. Davis (1936) outlined three possible mechanisms for the production of slow potential

waves in the cortex. First was the idea that slow waves were composed of summated spikes (either conventional ones (1-2 msec.) or slow ones (10-20 msec.)). He thought this possibility unlikely in view of the smooth contours of such EEG waves as the alpha rhythm. Second was the theory that slow waves were analogous to the long after-potentials of peripheral nerves. This was rejected on the grounds that such potentials were too small. Lastly, he hypothesized that cortical potentials may be the result of "partial, perhaps subliminal, depolarization of cell bodies under the influence of chemical mediators liberated at the termination of impinging axons". He judged this to be the most likely alternative, since slow waves in the spinal cord had been shown to be associated with facilitation and inhibition.

Gerard (1936) was also reluctant to accept the notion of summated action potentials being responsible for EEG slow waves for the same reason as Davis. Evidence from isolated groups of neurons in fish and invertebrates convinced Gerard of the "inherent capacity of neurons to exhibit a rhythmic electrical fluctuation in the absence of impinging nerve impulses". He believed that slow waves were due to synchrony of these spontaneously rhythmic neurons and that the slow waves represented similar potential changes in each neuron. That is, each neuron was thought to produce potential changes identical with those recorded on the surface. Evidence that EEG waves were occasionally, but not

invariably correlated with cell discharge led Gerard to state that "The potential wave may mark an excitation state which rises and falls rhythmically, sometimes reaching the threshold for discharge on the rise or at the peak, but more often not." He thought these changes in excitability were spontaneous and not dependent on synaptic activation and that synchrony was the result of electrical interaction between neurons.

Attempts were made to settle the question of whether slow waves were produced by slow variable depolarization in neurons or by all-or-none spikes of constant amplitude and duration by comparing monopolar recordings with closely spaced bipolar ones. It was argued that bipolar recordings should show lower amplitude slow waves if they were composed of summated spikes, since summation would occur from a smaller population. Jasper (1936c) reported slow waves to be equally prominent in bipolar and monopolar montages even in recordings between two microelectrodes 40 microns apart and stated he could find no evidence for the integration of higher frequency bursts into slow potentials.

A dissenting and more conservative position was held by Bishop (1936). He claimed that any wave form could be reconstructed from nerve axon potentials in the proper orientation and temporal dispersion. While not denying the other possibilities, he was reluctant to invoke new processes unless it was absolutely necessary.

Thus, it appears that by 1936 the majority of researchers had already recognized the possibility that something more than all-or-none activity may be present in single neurons and that this slower, graded activity may be the source of EEG potentials, although there was insufficient evidence to enable a conclusive decision between the two positions.

The classic paper of Renshaw, et al. (1940) resolved the question in favor of slow potential changes in individual neurons. They were able to demonstrate with microelectrode recordings that spikes could only be recorded from very localized depths in the cortex, whereas slow waves had a wide distribution. Further, spikes and slow waves appeared independently, one being obtainable in any phase relation to the other. Spikes appeared to ride on top of the slow potential changes when they appeared simultaneously and no intermediate forms of waves were ever seen. Slow waves were often recorded in the absence of spikes. The inversion of slow waves at certain depths in the cortex suggested to them that end to end polarization of pyramidal neurons due to synaptically generated sinks were responsible for the origin of the EEG potentials.

These conclusions were fully confirmed by Brookhart, et al. (1951) in their recordings from the cerebellum. Additionally, they were able to abolish spike activity by ischemia without affecting the slow waves. Li and Jasper (1953) were able to demonstrate the relative independence

of slow waves and neural firing. Thus, by the late 1950's it seemed reasonable to assume that the cortical EEG was due in large part to differential depolarization of the soma and apical dendrites of the pyramidal cells (Purpura, 1959). It remained only to demonstrate this by intracellular recording.

An attempt was made in this direction by Klee, et al. (1965), who demonstrated a significant cross-correlation between EEG potentials and the membrane potential of neurons in the sensorimotor cortex during spontaneous activity, augmenting and recruiting responses, and after application of strychnine.

A more detailed analysis of intracellular potentials and the EEG under those same conditions was carried out by Creutzfeldt, et al. (1966). They found that the familiar augmenting response to stimulation of specific thalamic nuclei (a sharp surface positive wave, peak latency 4-6 msec.) followed by a slower negative wave (peak latency 7-15 msec.), which is in turn followed by a very slow positive deflection (peak latency 15-25 msec.), is accompanied by the following alterations in membrane potential. There appear to be three sequential EPSP's with latencies of 1 to 4, 15 and 30 to 50 msec. which are superimposed on a slow IPSP which begins about 5 to 10 msec. after the stimulus, and has a peak latency of 15 to 25 msec. The surface response is thus explained in the following manner: the initial sharp positive wave is thought to be

partially due to the discharge of the afferent fibers which terminate on or close to the soma producing the primary deep EPSP. This generates a deep sink which adds to the surface positivity produced by the discharge of the afferent fibers, as does the efferent discharge. Electrotonic spread of the primary EPSP plus the secondary EPSP (which may be due to recurrent collaterals terminating further from the soma) add to the primary negative wave thought to be generated by the deep IPSP. As the IPSP spreads up the dendrites, the surface potential will turn positive. A second negative wave which is sometimes present may be explained by a slow tertiary EPSP in combination with the central decay of the IPSP.

The recruiting response produced by stimulating nonspecific nuclei is a negative wave which begins 10-20 msec. after the stimulus, with a peak latency of 30-50 msec. The membrane response is a slow EPSP which parallels the time course of the surface wave. Thus the recruiting response is interpreted as being generated by the production of a superficial sink by dendritic EPSP's. Similar analyses of the direct cortical response, after discharges, and spontaneous spindles have been attempted. Certain caution must be exercised in the use of the terms EPSP and IPSP, since in this paper they are merely synonyms for depolarization and hyperpolarization. However, this makes no difference to their source-sink analysis.

Investigations of the mechanisms involved in the control of synchrony may be divided into two categories: those concerned with arousal mechanisms and those concerned with the pacemaker of cortical spindles. Only the second category will be considered here.

Since the study of recruiting and augmenting responses has been closely linked with investigations of thalamic organization and spindling mechanisms, it is necessary to examine this work in some detail.

These responses are cortical potentials evoked by stimulation of non-specific and specific thalamic nuclei. They are usually small or non-existent when single shocks are applied to the thalamus, yet grow rapidly in amplitude over the first four or five stimuli if repetitive stimulation in a limited frequency range is used (5-15 Hz.). If this repetitive stimulus is continued, the response waxes and wanes in an irregular fashion reminiscent of spontaneous spindling.

The recruiting response was discovered by Morison and Dempsey (1942). They described a long latency (20-35 msec.) monophasic surface negative response to repetitive stimulation of midline thalamic nuclei. The cortical distribution of the response coincided almost exactly with that of the spontaneous spindling activity which occurs during barbiturate anaesthesia. This contrasted with the "primary" response to stimulation of the specific nuclei, which was of shorter latency (3-4 msec.), initially positive, was confined to the cortical projection area

of the nucleus, and followed higher frequencies of stimulation (30 Hz.). They concluded that the midline thalamic nuclei project diffusely to the areas where recruiting is found.

Further investigation of the phenomenon (Dempsey and Morison, 1942a; b) revealed that the recruiting of the cortical response was most rapid when the thalamus was stimulated at 5 to 10 Hz. At higher frequencies (10-15 Hz.) the response exhibited alternation, i.e., every second response was attenuated. High frequency stimulation did not produce cortical responses but blocked spontaneous spindling. In lightly anaesthetized preparations, single shocks often elicited a full-sized cortical response or triggered a spindle.

The removal of posterior cortex did not affect frontal spindling or recruitment, and vice versa, eliminating the possibility that the thalamic projection responsible for recruiting was directed to a particular cortical area and then relayed to the rest of the cortex. They found that the cortical evoked potentials to stimulation of the sciatic or radial nerves did not interact with the spontaneous spindle waves or with recruiting responses, although the sciatic evoked potential was blocked by prior stimulation of the specific relay nucleus. They inferred that spindling and recruiting waves did not utilize the specific thalamo-cortical projections. Recruiting and spindle waves did demonstrate occlusion. This, coupled with the similarity in cortical distribution and frequency

characteristics indicated that spindling and recruiting may share a common mechanism.

As previously mentioned, stimulating the specific projection nuclei produces a short latency, initially surface positive cortical response limited to the projection area of the nucleus (the primary response). If this stimulus is repeated at 5 to 15 Hz., this primary response is followed by a longer latency negative wave (the augmenting response) which grows in a similar fashion to the recruiting response. (Dempsey and Morison, 1943). It was found that the augmenting portion of the response to stimulation of the specific nuclei was more widespread than the primary response yet more limited than the recruiting response.

Single shocks to the specific relay nuclei or peripheral nerves were often found to elicit spindle bursts which were localized to the projection area of the nucleus. This was in contrast to the wide-spread distribution of spindles evoked by stimulation of non-specific thalamic nuclei. They found that although the primary response to peripheral nerve stimulation did not interact with the spindle waves it evoked, the augmenting response was occluded by such spindles. Spontaneous spindles or recruiting responses did not appear to affect the spindle evoked by radial nerve stimulation, implying that there are two distinct sources of spindles, one from the specific and one from the non-specific nuclei. This was further supported by their demonstration that high frequency

stimulation of the non-specific nuclei which blocked spontaneous spindles failed to block the spindle evoked by radial nerve stimulation.

Morison and Dempsey (1943) made several attempts to discover the mechanism underlying the augmenting response. They found that cutting the internal capsule did not prevent augmenting and evoked spindles in the thalamus, but did abolish these responses in the cortex when the internal capsule distal to the cut was stimulated. Thus the rhythmic generator appeared to lie in the thalamus. However, application of acetylcholine to the cortex enhanced the magnitude of both cortical and thalamic evoked spindles, indicating that cortico-thalamic fibers enhanced the response. Shocks to the internal capsule or specific nuclei were found to retune the potential waves of an evoked spindle so that the next wave always occurred about 100 msec. following the last stimulus. This suggested that a long-lasting refractory period was involved in the rhythmic mechanism. Morison and Dempsey thus proposed that the rhythmic mechanism resided in the thalamus and consisted of recurrent collateral excitatory input to thalamic interneurons and efferents, accounting for the build up of the response to repetitive stimulation and a refractoriness or "subnormality" of the thalamic interneurons following their firing to account for the rhythmicity. This hypothesis is not far removed from the thalamic recurrent collateral inhibition proposed by Andersen and Andersson (1968) in explaining the thalamic generation of

cortical spindles.

Hanbery and Jasper (1953) were able to show that the recruiting responses were independent of the specific nuclei by lesioning them one at a time. Furthermore, they noted that stimulation of the inferior medial portion of the ventralis anterior (VA) nucleus produced short latency (5-10 msec.) responses which were diffusely projected over much of the cortex. These responses did not exhibit much change in amplitude with repetitive stimulation but the latency and length of the response increased. Widespread spindles could also be elicited by single shocks to the VA while lesioning this portion of the VA eliminated recruiting from stimulation of the centre median (CM). They concluded that the VA nucleus may be the final common path of the diffuse projection system of the non-specific nuclei.

Hanbery et. al. (1954), however, point out that the non-specific system does not operate as a unit. Stimulation of the medial portion of the anterior reticularis produces responses in the anterior cortex, while more lateral stimulation induces responses in the lateral and posterior cortex. Anterior cortical responses to stimulation of CM were eliminated by lesion of the ventral VA, while more posterior cortical responses were eliminated by dorsolateral VA lesions. Unilateral VA lesions, however, did not eliminate contralateral cortical responses to stimulation of the homolateral CM. Lesions of the CM, on the other hand, did

not disrupt the cortical response to stimulation of the VA. It is unfortunate that they did not test for the presence of augmenting responses in VA lesioned animals as this might have helped to resolve the question of whether augmenting and recruiting responses utilize common pathways.

Brookhart *et. al.* (1957) showed that augmenting and recruiting responses are mutually facilitatory in that the cortical negative wave was absent when either specific or non-specific nuclei were stimulated at 5 Hz. but appeared when the two stimuli were interposed, bombarding the cortex at 10 Hz. However, the negative wave did not look like that produced by 10 Hz. stimulation of either the specific or non-specific nuclei. It is possible that the interaction took place at the thalamic level, for it has been shown with intracellular recordings that the specific and non-specific nuclei have strong mutual connections (Purpura, 1969).

Enamoto (1959) has shown that the contralateral recruiting response is primarily mediated by the massa intermedia. He found that primarily unilateral responses were obtained from stimulating the more lateral non-specific nuclei, while midline stimulation naturally evoked bilaterally symmetrical responses. However, stimulation one or two millimeters from the midline evoked largely bilateral responses in which the ipsilateral response led the homotopic contralateral response by up to 20 msec. Section of the corpus callosum attenuated this contralateral response by about 25%, but subsequent cutting of the massa intermedia

completely abolished it. If the massa intermedia alone was cut, low amplitude responses persisted in the contralateral hemisphere, indicating that the corpus callosum may play some role in interhemispheric synchrony. This is supported by the observation of Villablanca and Schlag (1968) that thalamic spindles were asynchronous in completely decorticate preparations. Furthermore, bilateral recruiting responses have been obtained in humans although they lack the massa intermedia (Housepian and Purpura, 1963).

It has been reported that single shocks to the head of the caudate can elicit cortical spindles in the cat (Buchwald, et. al., 1961), the monkey (Kitsikis, et. al., 1968), and the rat (Gusselnikov, et. al., 1973). In the cat these spindles are most reliably found in the ipsilateral anterior sigmoid gyrus, but under favorable circumstances it is also found contralaterally as well as in more widespread regions of the cortex. Although the threshold was higher and the duration shorter, spindles could still be evoked in the alert, behaving animal. These spindles are probably mediated by the thalamus, since they are blocked ipsilaterally (but not contralaterally) by lesions of the ipsilateral VA, and totally abolished by bilateral VA lesions. Centre median lesions did not affect the evoked spindle. Separation of the caudate from the thalamus with a knife cut eliminated evoked spindles, although bilateral spindles could still be elicited by stimulation of the other caudate (Heuser, et. al., 1961).

It appears from thalamic multi-unit recordings that the caudate may exert a strong inhibitory effect on the thalamus. The caudate stimulus may thus synchronize the thalamic units by simultaneously suppressing their activity (Gusselnikov, et. al., 1973).

It has been suggested by Velasco and Lindsley (1965) and Velasco et. al. (1968) that the orbital frontal cortex plays a crucial role in the production of spindles. They reported that orbital cortex lesions (but not other cortical lesions) prevented recruiting and spindle bursts in the thalamus and cortex of unanaesthetized paralyzed cats. However, Villablanca and Schlag (1968) have reported that complete decortication does not eliminate spindling in paralyzed cats. They found that interruption of the connection between the thalamus and orbital cortex (the inferior thalamic peduncle) did eliminate thalamic spindles, but they returned following complete decortication. Robertson and Lynch (1971) found that orbital cortex lesions eliminated spindling only in unanaesthetized animals, while Dahl, et. al. (1972) reported a disappearance of spindles in orbital cortex lesioned animals only if the preparation was in poor condition (damage to blood supply or edema). In short, it appears that the orbital cortex is not essential for the generation of spindles, although it may facilitate their appearance in certain conditions.

Spencer and Brookhart (1961) carried out a careful study of spontaneous spindle waves in the cortex. In examining the individual

waves, it became clear to them that there were two different types of negative wave, one preceded by a sharp positive deflection and one followed by a slow afterpositivity. Some additional waves appeared to be a mixture of the two types. Laminar analysis of the potentials in the cortex showed that the Type 1 spindles (initial positivity) were very similar to augmenting responses, while the Type 2 spindles could be identified with recruiting responses. The relationship of these spindle waves to unitary discharges also supported this theory. They found that most spindles contain both types of wave, the spindle usually being initiated by Type 1 waves and soon changing into Type 2 and mixed waves. This indicates that spindles are normally initiated in the specific nuclei and spread to the non-specific areas of the thalamus.

The most comprehensive theory of the mechanism of generating rhythmic activity in the EEG has been proposed by Andersen and Andersson (1968, 1974). Briefly, the theory states that the rhythmic oscillators reside in the specific thalamic nuclei and consist of thalamo-cortical efferents which possess recurrent collateral inhibition. Thus if one of these efferent cells fires, it and neighboring cells are immediately subjected to a long-lasting (100 msec.) period of inhibition. When this small group of cells is released from inhibition, their activity will thus tend to be synchronized; they will fire, inhibit themselves and a larger group of surrounding cells, thus recruiting an increasingly

larger number of cells into synchronous activity with a period of about 100 msec. This activity will not build up indefinitely, however, since the inhibitory periods of different cells will not be identical, and they will consequently get out of phase after a few cycles. In this manner the rhythmic activity will tend to wax and wane in a spindle-like fashion. Bursts of impulses from the thalamo-cortical efferents will depolarize cortical dendrites, causing rhythmic potentials in the cortical EEG.

The evidence for this theory is quite strong, and since it has been reviewed in detail (Andersen and Andersson, 1968, 1974), only a brief summary of the work will be given here. The rhythmic activity studied has almost exclusively been the "barbiturate spindle" in the cat. This spindle, which is very similar to the sleep spindle, has a frequency of about 10 Hz. and occurs primarily in the frontal and central regions of the cortex of the barbiturized preparation. That it is driven by the thalamus is shown by the fact that it is absent in the cortex of the thallectomized animal, yet present in the thalamus of the decorticated preparation. That it is dependent on the specific thalamic nuclei is shown by the fact that it persists if the midline "non-specific" nuclei are lesioned, yet is absent when the specific nuclei are subsequently removed. (This finding must be accepted with reservation, for in the example shown (Andersen, et. al., 1967, Figure 9, p. 271) the medial lesion spared the VA and reticularis nuclei, while the lateral lesion

was so large that it may have caused edema to the surviving non-specific nuclei.) A point-to-point projection of the rhythmic activity of the specific thalamic nuclei can be shown, as adjacent thalamic and cortical points often show independent rhythmic activity. Long-lasting IPSP's follow activation of thalamo-cortical relay cells both when orthodromically stimulated or antidromically stimulated in the chronic decorticate preparation in which cortico-thalamic fibers have degenerated, indicating the inhibition is due to recurrent collaterals. Thalamic spindles are found to consist of bursts of activity followed by 100 msec. periods of inhibition.

Thus, the evidence is strong that sleep spindles may be generated by thalamic recurrent inhibition. However, there are a number of reasons to doubt that alpha and sleep spindles share a common mechanism. Firstly, they have quite different frequencies and distribution over the head in humans. The alpha, as we have seen, has a frequency of about 10 Hz. and occurs with highest amplitude in the occipital region, whereas the spindles have two distinct frequencies--12 and 14 Hz., the faster spindle occurring maximally in the frontal and parietal regions, while the slower one occurs primarily in the frontal region (Gibbs and Gibbs, 1950; 1952). The alpha appears to be bilaterally synchronous, yet spindles show no such close relationship, often occurring independently in the two hemispheres (Caveness, 1962). The alpha frequency is greatly affected

by thyroxin injections, yet only spindle amplitude appears to be affected (Lenard and Bell, 1973). As noted earlier, the ontogeny of the alpha rhythm and sleep spindles is completely different. Last but not least, it is obvious that the two rhythms occur in entirely different states of arousal, the alpha disappearing in drowsiness, the spindle appearing only in sleep or anaesthesia.

These facts indicate that it is unlikely that spindle and alpha mechanisms are identical and that one should use caution in applying principles based on the study of spindles to the human alpha rhythm. This conclusion has been supported by Lopes da Silva et al. (1973a;b), who found a much higher coherence between thalamus and cortex for barbiturate spindles than for alpha in dogs, although one must bear in mind the caveats concerning the use of coherence.

The possibility has been raised in a recently published book by Lippold (1973) that the alpha rhythm may merely be due to the oscillating corneo-retinal potential, which is being modulated by eye tremor. It will be remembered that the rotation of the eye was eliminated as a possible source of alpha potentials, since it was noted that eye movement artifacts in the EEG due to large rotations were largest in the front of the head and virtually undetectable in the occipital region. However, the possibility remains that translational movements of the eye in the optic axis (i.e., in-out movements) might produce the largest potential

change directly behind the eye in the occipital region. Lippold has measured such a translational tremor and found it to be correlated with the alpha. He has accumulated further evidence that these eye movements produce the alpha and not vice versa. The magnitude of the corneo-retinal potential changes slowly over time and can be induced to vary by a factor of two or three by changing the illumination. He reports that alpha amplitude is highly correlated with the variation of corneo-retinal potential. Even increasing the corneo-retinal potential in one eye while decreasing it in the other can be correlated with concomitant alpha changes in ipsilateral hemispheres. Much other supportive evidence is provided, including altering alpha frequency by warming or cooling the orbits and producing potential changes at the back of the head by prodding the eye. However, much of this additional evidence is open to other possible interpretations.

Most of Lippold's results can be incorporated into a theory which assumes that the alpha potentials have a neural origin. For instance, a cross-correlation between alpha and the eye tremor may be due to cortical input to the oculo-motor control system. If the cortex was simply driving the oculo-motor nuclei, which in turn emitted bursts of impulses simultaneously to opposing extra-ocular muscles, one would expect a cross-correlation between the tremor and the cortical EEG, but with a phase lag which merely represented the conduction

time and twitch time of the muscle. However, oculo-motor feedback to the cortex from extra-ocular muscle spindles via the oculo-motor nucleus could produce a rhythm in which the muscle twitch was 180° out of phase (or in phase, depending on the recording polarity) with the cortical potential. This feedback model could account for the slowing of the rhythm on cooling the orbit. Even the non-feedback model is not incompatible with the results of this experiment, since it is entirely possible that the blood supply to the brain is being cooled by this procedure, slowing the neural rhythm. Cortical potentials produced by prodding the eye may merely be neural evoked potentials to such stimulation.

The experimental results which are most difficult to explain with a neural model are those which show a relationship between alpha amplitude and the magnitude of the corneo-retinal potential. This is especially true of Lippold's experiments in which the corneo-retinal potentials of the two eyes were manipulated independently and showed that the alpha amplitude is related to the ipsilateral corneo-retinal potential. Since the visual input from each eye is bilaterally symmetrical, this is most difficult to explain.

Since the interpretation of the results of our deprivation experiments is obviously different if one rejects the traditional neural theory of alpha potentials, we have attempted to replicate Lippold's finding of a correlation between alpha amplitude and magnitude of the corneo-retinal

potential. We did not try to manipulate the corneo-retinal potential independently in the two eyes, since negative results in that experiment might be due to the inability to separate the potentials at the back of the head from each eye if the exact electrode placements necessary for this were not achieved (Lippold, 1970). We could establish no relationship between the corneo-retinal potential and the amplitude of the alpha rhythm, although in replicating Lippold's experiment we were successful in altering the magnitude of the corneo-retinal potential by a factor of two.

Naturally, Lippold's theory has not escaped criticism from other researchers. Shaw, et. al. (1970) and Chapman et. al. (1971) have both produced alpha recordings from eyeless subjects. In the former case, however, it was found that the entire contents of the orbit had not been removed, and a DC potential, modified by movements of the extraocular muscles, could be demonstrated in this subject (Lippold and Shaw, 1971). Chapman's examples are more difficult to dismiss. Although the subject with bilateral enucleation did not have the entire contents of the orbit removed, two other one-eyed subjects did meet this criterion. Using Lippold's recording montage (Lippold, 1970) to record the alpha from each side of the head, they were unable to demonstrate any amplitude difference between the left and right sides of the head.

Butler and Glass (1970) have attempted to refute Lippold's

hypothesis by recording eye movements photoelectrically while simultaneously monitoring the EEG. Unfortunately, their technique was sensitive only to rotational eye movements, and, therefore, does not provide a true test of Lippold's hypothesis. It is interesting that they did note some frequency components in the alpha range in their eye movement recordings. In these cases, Lippold might argue that the translational tremor may have been modulating the rotational movement. It should be noted that a stretch reflex has yet to be demonstrated in the extra-ocular muscles, and even if such a reflex existed, the fact that the extra-ocular muscles have a twitch time which is three or four times faster than any other skeletal muscle would predict that eye tremor would be much higher frequency than that of other muscle systems which Lippold claims oscillate at about 10 Hz. This argues against Lippold's proposed mechanism of the tremor, but not, of course, against the findings themselves.

Alpha and Deprivation

It is puzzling and at the same time rather intriguing that the alpha frequency which is normally highly stable and difficult to alter except by gross changes in body temperature or metabolic rate or the administration of potent drugs such as phenothiazines, can be easily lowered 1 to 3 Hz. by the rather innocuous procedure of exposing the subject to an unpatterned environment for a few days. Moreover, recovery may

take a week or more when the subject returns to his normal environment.

Experiments involving restriction of sensory input have been divided into two broad categories in order to achieve a standard nomenclature. Those experiments whose primary object is not to reduce sensory input to a low level, but to eliminate any patterning of input (e.g., conditions where vision is restricted to white light and sound to white noise), have been termed perceptual deprivation (Kubzansky, 1961).

The slowing of the alpha rhythm was first reported by Heron, et. al. (1956). They required subjects to lie in bed for four days wearing cardboard tubes on their forearms to restrict somesthetic sensation, and translucent goggles to restrict vision to white unpatterned light. The alpha rhythm was found to be progressively slower on the the second and fourth day of deprivation. A more detailed description of this effect (Heron, 1957) shows that the alpha slowed from 1 to 2 Hz. in three subjects.

This finding was first replicated by Zubek, et. al. (1961). They exposed eight subjects to a sensory deprivation (darkness and silence) condition for seven days. Six of the subjects were reported to show a slowing of the alpha frequency of 1 to 2 Hz. measured before and after deprivation. Three subjects also showed unusual amounts of theta activity.

Zubek and Welch (1963) compared the effects of seven days of

sensory deprivation with seven days of perceptual deprivation, finding a significantly greater alpha frequency shift in the perceptual deprivation group (-1.21 Hz. vs. -0.85 Hz. for the sensory deprivation group). They compared these results with a recumbent and an ambulatory control group. The ambulatory control group merely had two EEG recordings taken one week apart, maintaining a normal living pattern in the interim. The recumbent controls lay on air mattresses in groups of three or four to a room, and they were only allowed to sit up for meals or stand up to use the washroom. During the seven-day period they were allowed to "talk, read, listen to the radio and watch television; all lights were put out at night". The ambulatory controls showed a frequency shift of $+0.01$ Hz., while the recumbent controls had a shift of -0.04 Hz., which were not significantly different from each other, but both were different from the experimental groups. Each group had an N of 10.

Zubek (1963) attempted to show that the stimulation due to exercise could reduce the effects of perceptual deprivation. In this study, the subjects wore translucent goggles and were subjected to white noise, but were free to move about the deprivation chamber. In addition, six five-minute exercise periods were required during the day. An average alpha frequency shift of -0.48 Hz. was found, which was significantly different from the -1.21 Hz. change shown previously for perceptual

deprivation without exercise (Zubek and Welch, 1963). However, Zubek (1963) mentions that in this experiment (the exercise group), the mask was removed for one hour each day for behavioral testing. At first glance, this might appear to be an important difference from the "no-exercise" perceptual deprivation group (Zubek and Welch, 1963), since in this report no mention is made of any interruption of the deprivation. However, it appears likely that the data reported by Zubek and Welch (1963) is from the same experiment reported previously (Zubek, et. al., 1962) in which behavioral changes were measured with paper and pencil tests (which presumably necessitates removal of the mask). Zubek (1963) stresses that the "exercise" and "no-exercise" perceptual deprivation groups experienced identical conditions (with the exception of the exercise, of course). This casts new light on the interpretation of the difference between the effects of sensory and perceptual deprivation on the EEG. It may be that interrupting the sensory deprivation by turning on the light and administering tests for an hour a day has a greater ameliorating effect than in the perceptual deprivation condition, where no gross illumination change occurred. This hypothesis is supported by a recent report (Zubek, et. al., 1971) in which no difference in magnitude of alpha shift could be seen between four days of perceptual deprivation (-1.14 Hz.) and four days of sensory deprivation (-1.07 Hz.). It is likely that the deprivation was uninterrupted in this study, since

all testing was done before and after the deprivation period.

Zubek and Wilgosh (1963) further investigated the role of exercise by immobilizing a subject by strapping him in a coffin-like box for 13 hours a day for seven days, while attempting to otherwise maintain a "normal" level of patterned input. An alpha shift of -0.56 Hz. was found for a group of ten subjects.

This effect is again reported with two additional subjects showing an alpha shift of -0.59 Hz. ($N = 12$) (Zubek and MacNeill, 1966). A control group required to be in the immobilization box (but otherwise unrestricted) showed an alpha shift of -0.11 Hz., which was not significantly different from ambulatory controls ($+0.04$ Hz.), but was different from the immobilized subjects.

The effects of immobilization were further studied by combining immobilization with perceptual deprivation for a seven-day period (Zubek, et. al., 1969a). They found an alpha shift of -1.42 Hz., which is presumably not significantly different from perceptual deprivation alone (-1.21 Hz.) (Zubek and Welch, 1963). The two groups are comparable, since in both the deprivation was interrupted for one hour a day for behavioral testing.

Zubek, Welch and Saunders (1963) reported that three subjects undergoing 14 days of perceptual deprivation exhibited an alpha frequency shift of -3.03 Hz. For the first time in Zubek's studies, the alpha was

measured during the deprivation period on days 7, 10, and 12, during recovery three hours after deprivation, and one, two, and seven days later. The average alpha frequencies measured were as follows: Day 0, 11.56; Day 7, 10.90; Day 10, 10.04; Day 12, 9.09; Day 14, 8.53; Hour 3 (post-deprivation), 9.10; Day 1, 9.71; Day 2, 9.98; Day 7, 11.02. Thus, the alpha can be seen to get progressively slower throughout the second week of the deprivation period, and recovery is a gradual process, not complete after seven days.

Seven more subjects were later run for 14 days of perceptual deprivation to increase the group N to 10 (Zubek, 1964). The general picture remained the same, but the magnitude of the alpha shift observed (-1.04 Hz.) is significantly different ($p < .005$) than that seen in the first three subjects (-3.03 Hz.). This difference, which is not noted by Zubek, is puzzling.

Saunders and Zubek (1967) and Zubek (1969) claim that a difference in the magnitude of the alpha shift at the midpoint of the 14 days of deprivation (-0.66 Hz. for the first three subjects, -0.40 Hz. for the next seven) and the alpha shift produced by seven days of perceptual deprivation (-1.21 Hz.) indicates that the rate of change of alpha frequency is determined by the length of time the subject expects to remain in deprivation (i.e., the longer the subject expects to stay, the more gradual the EEG change will be). However, the two groups are not strictly

comparable; the seven-day subjects were required to lie in bed, while no restrictions were placed on the movement of the 14-day subjects. Thus, a more comparable group would be the "exercise" group of Zubek (1963), which showed an alpha shift of -0.48 Hz. at the end of seven days of perceptual deprivation. Zubek (1964) states that the 14-day subjects were not allowed to exercise per se, but it is impossible to say how much of the "exercise" effect (Zubek, 1963) was due to the 30 minutes of required exercise, and how much was due to the lack of restriction on mobility for the other $23\frac{1}{2}$ hours of the day. A further difference between the 14-day group and previous experiments was that behavioral testing was not done during the deprivation period. Presumably, (although not specifically stated) this means that the deprivation of the 14-day group was not interrupted by removal of the mask.

Further support for the theory that the alpha shift occurs more rapidly if the subject expects to experience a shorter period of deprivation is provided by a comparison of the effects of one, four, or seven days of sensory deprivation (Zubek, et. al., 1970). He finds an alpha shift of -0.92 Hz. for one day of deprivation, -1.08 Hz. for four days of deprivation, and -0.85 Hz. for seven days of deprivation. This last figure is from the Zubek and Welch (1963) study in which the deprivation was interrupted for an hour a day. It is possible, however, that in each of these groups the 1 Hz. shift occurred over the first day of deprivation

and little change occurred after that. The only contradictory evidence to this hypothesis is the data from the original perceptual deprivation study at McGill showing a progressive change in alpha frequency over four days of deprivation, the alpha being slower on Day 4 than on Day 2. The data from Zubek's 14-day subjects show a progressive slowing over the second week of deprivation, but only a small shift occurred over the first week. (As mentioned earlier, this might be due to the fact that these subjects were not confined to bed.)

The Present Experiment

There were three main reasons for doing the present experiment. Firstly, we wanted to test the prediction that the alpha rhythm slowed down more rapidly over shorter deprivation periods. Secondly, we wanted to provide a more complete description of the effect of deprivation on the EEG. This was made possible by computer power spectral analysis of the EEG, which would provide information about the whole frequency band of the EEG. Previous studies merely reported frequency changes of the dominant occipital rhythm, while we wished to be able to quantify statements about the amplitude, frequency, and regularity of all spectral components of the EEG from frontal, central temporal and parietal, and occipital regions, particularly in view of reports that frontal alpha might have different characteristics from the occipital

rhythm.

Thirdly, the relationship between alpha and spindle mechanisms could be tested; since perceptual deprivation produced a powerful and reliable effect on alpha, it would be expected to affect sleep spindles in a similar manner if they shared a common mechanism. If all brain rhythms were affected in a similar manner, it might imply that deprivation produces some general change in brain metabolism.

In view of a report by Zubek, et. al. (1969b) that the alpha rhythm could be slowed by social isolation (-0.62 Hz.) or confinement (-0.50 Hz.), a control group was run exposing the subjects to the same degree of confinement and social isolation as the experimental group. Since the sleeping patterns of the subjects were also being studied, all subjects were required to sleep three nights in the laboratory before and after the deprivation period.

PROCEDURE

Subjects

Twelve adult humans (11 males and one female) between the ages of 21 and 30 were paid to participate in the experiment. The selection of the subjects was done on the basis of an EEG recording, a psychiatric interview, and an MMPI profile. No subjects were rejected due to the psychiatric interview or MMPI, but a few applicants had insufficient alpha amplitude to produce a reliable power spectral peak and, therefore, were not used in the experiment.

Experimental Design

The experimental subjects were divided into two groups of four, one group undergoing four days and the other seven days of deprivation. Two control groups of two subjects each experienced either four or seven days of confinement. All subjects slept in the laboratory for three nights immediately prior to, and three nights immediately following the deprivation or confinement period so as to provide baseline data and a record of the recovery from the deprivation or confinement effects.

On the pre- and post-deprivation nights the subjects retired at midnight, and were awakened at about 8 a.m. The deprivation and control confinement period began at midnight following the third pre-deprivation night of sleep in the laboratory and ended at 8 a.m. on the

fifth or eighth deprivation day. Deprivation thus actually lasted $4 \frac{1}{3}$ or $7 \frac{1}{3}$ days.

The Deprivation Procedure

A perceptual deprivation procedure (constant unpatterned light and white noise) was used. The conditions were much the same as those used in the initial studies at McGill. During deprivation, subjects were confined to bed in a small airconditioned audiometric room (Figure 1) (Ekel Industries; 4'0" by 7'4" by 6'8" high). Movement was restricted by the 3-foot long EEG leads which were attached to the head of the bed and did not permit the subject to sit up. Although the room was not completely soundproof, during deprivation it was flooded with 80 db. white noise (Grason-Stadler model 901B noise generator) from an overhead speaker which effectively masked all outside sounds. A two-way intercom provided communication with the subject, who could be seen through an observation window at the head of the bed.

The subject wore a soft plastic translucent mask which was trimmed around the edge with foam rubber and individually fitted to ensure that no patterned vision was possible. Illumination was provided by two 40 watt bulbs over the subject's head. The intensity of the light transmitted by the mask was 1.2 log foot lamberts. During the pre- and post-deprivation nights sleep, one of the 40 watt bulbs was extinguished so that the intensity of light at the subjects' eyes approximated that of the

deprivation condition.

In order to limit somesthetic stimulation and restrict manipulation of the environment, the subject wore cotton gloves and cardboard tubes which covered his hands and forearms.

The deprivation was interrupted at 8 a.m., 1 p.m., and 7 p.m. for meals, at which time the subject left the deprivation chamber and sat at a table. The white noise was turned off and the cardboard tubes and gloves removed, though the translucent mask was kept in place. The subject was fed a normal diet. The electrodes were filled with conducting paste, the resistances checked, and the electrodes replaced if necessary. Conversation was not restricted during meal times, the subject being questioned about his visual imagery and dreams. Subjects averaged 3.5 hours a day out of the deprivation chamber. This included occasional visits to the chemical toilet located in the same room as the deprivation chamber, on request.

The Control Condition

The control condition was designed to determine whether the deprivation effects were actually due to the reduction of patterned visual, auditory and to some extent, somesthetic sensory input, and not to the routine of the experiment, the confinement to bed, and the social isolation for relatively long periods of time.

The control subjects were thus subjected to the same routine as the experimental subjects. They slept in the laboratory for three nights before and after the confinement period, which lasted for four or seven days. During confinement they were required to lie in bed in a room by themselves and were monitored with a TV camera. They were fed according to the same schedule used in deprivation, and interaction with the experimenters was restricted to mealtimes.

In contrast to the deprivation situation they wore no translucent mask or cardboard tubes and gloves, and white noise was not presented. They were permitted to watch TV and listen to the radio. (The TV was turned off at midnight so as not to disrupt sleeping patterns with late, late shows.) Five-foot EEG leads allowed them to sit up in bed if they wished.

EEG Data: Alpha Rhythm

The samples of EEG for computer analysis were recorded before the subject retired and on arising on each of the pre- and post-deprivation (or confinement) nights, and before and after each meal (8 a.m., 1 p.m., and 7 p.m.) during the deprivation (or confinement) period. It was decided to record alpha before and after meals, since it was thought to be desirable to have some control over the subjects' arousal level during the recording, and yet we did not want to disturb the subject in the intervals

between meals, as sleeping patterns were being studied as well. Additional alpha recordings were taken at midnight at the onset of deprivation and one and two hours following the termination of deprivation. Alpha recordings were also made at noon on the fifth and seventh post-deprivation day.

Each alpha recording session lasted 15 minutes, during which four three-minute samples of EEG were taken, the subject having his eyes open or closed in alternate three-minute periods. A one-minute period of artifact-free record was selected from each of these three-minute samples. The data for computer analysis thus consisted of two minutes of record with the subject's eyes closed and two minutes with them open.

It was noticed that the pre-deprivation EEG records of the first subject (D. M.) showed an appreciable drop in alpha frequency from evening to morning. An attempt was thereafter made to control one simple variable, blood sugar, by having the subjects drink a glass of orange juice sweetened with two level teaspoons of sucrose 15 minutes before each alpha recording that did not immediately follow a meal. This procedure appeared to have no effect on the circadian fluctuation of alpha frequency.

In order to ensure the subjects' arousal level was the same at each alpha recording session, they were roused by the experimenter before each session and required to perform three or four mental arithmetic

calculations of reasonable difficulty (e.g., 12×17). In fact, arousal level may be roughly monitored, since alpha disappears with the onset of drowsiness. On occasion it was necessary to give the subject a continuous task, such as subtracting serial 7's from a large number, in order to obtain sufficient alpha during the recording for computer analysis.

The Recording

Chlorided silver electrodes were filled with electrode cream (EKG Sol) and fixed to the head with gauze squares and collodion. The EEG was recorded monopolarly from the right frontal (F4), central (C4), temporal (T4), parietal (P4), and occipital (O2) placements using the right ear (A2) as a reference and the vertex (Cz) as ground (International Federation 10-20 system, Jasper, 1958). The electro-oculogram (EOG) was recorded bipolarly from a pair of electrodes placed on the outer canthus of each eye, and the electromyogram (EMG) was recorded from a pair of electrodes about 1 cm. apart on the submental muscles under the chin. These last two recordings were to enable the scoring of Stage I REM in the sleep records. Electrode resistance was kept below 10 K ohm.

A continuous recording of the EEG was made on a Grass Model IVS polygraph during the pre- and post-deprivation nights of sleep and throughout the entire deprivation or confinement period. The five channels of EEG were recorded on an Ampex SP-300 FM tape recorder for computer

analysis. The frequency response of the polygraph is shown in Figure 2. The tape recorder had a flat frequency response from 0 to 300 Hz., a wide band (hash) noise level of 50 μ v. peak-to-peak, and a 2 Hz. wow 150 μ v. peak-to-peak.

Data Analysis

The EEG on magnetic tape was further filtered to reduce the problem of "aliasing" in the calculation of the power spectrum. The frequency response of the filter is shown in Figure 2.

Power spectral analysis was then carried out with a PDP/8 digital computer. One-minute samples of EEG were digitized at a rate of 50 Hz. and stored in memory. The autocovariance and power spectrum were then calculated using 100 lags. (See Dixon (1964) for calculation procedure.) The program produced a histogram, the power spectrum, showing the average power in the sample at each frequency over the range 0-25 Hz. The first bar in the histogram represents the average amount of power present between 0 and 0.25 Hz.; the second, the power between 0.25 and 0.50 Hz.; ... the last, the power between 24.75 and 25.00 Hz. This provides a resolution of 0.5 Hz., which means one can distinguish two simultaneous sine waves in the signal if their frequencies are separated by 0.5 Hz. The power spectra were plotted on graph paper with a Moseley X-Y recorder (Model 3D3) and stored on punched paper tape.

The distribution of power attributable to the alpha rhythm in the power spectrum has an appreciable width (usually about 2 Hz.) which is due partly to the fact that the alpha frequency is not perfectly stationary (i.e., it varies slightly from second to second), and partly to the fact that the power spectral histogram is smoothed (hamming), spreading power in high peaks to adjacent bands. For this reason, the frequency of the alpha in each power spectrum was calculated in three separate ways. First, the mode was found, representing the frequency with the greatest average power in the one-minute sample of EEG. Second, the weighted average frequency was calculated over a range of ± 2 Hz. on either side of the mode (the "center of gravity" of the histogram). This measure took into account all the power the subject was producing in the alpha band. If the alpha power was symmetrically distributed around the mode, this average would, of course, be identical to the mode. Third, the weighted average frequency was calculated over a fixed range of the spectrum (6-13 Hz.). This range was chosen to contain the mean alpha frequency of all subjects throughout the experiment with 1 Hz. added at each end of the range.

Other measures calculated were the power of the mode, the total power within the range of analysis (± 2 Hz. from the mode or 6-13 Hz.), the variance of the distribution in the range of analysis, and the "spread" of the peak. The spread of the alpha peak was defined as the frequency

range over which the power was greater than one-quarter of the modal power and is analogous to the calculation of "Q" in resonant circuits. Since there were occasional power spectra of very low amplitude alpha (usually from "eyes open" samples), whose distributions were broad and close to the "noise level" of the background desynchronized EEG, it was necessary to define the presence or absence of an alpha peak. An alpha peak was said to exist if the "spread" was less than 6 Hz. This, in effect, means that the alpha amplitude had to be at least twice the noise level.

RESULTS

The results will first be briefly summarized and then considered in detail.

1. The frequency of the alpha rhythm decreased during the deprivation period by 1.5 to 2 Hz. The control subjects showed little or no reduction in alpha frequency.
2. The time course of the decrease in alpha frequency was similar in the four-day and seven-day deprivation subjects.
3. Subjects with high amplitude alpha showed a reduction in power of the alpha, while subjects with low amplitude alpha showed no change or an increase in alpha power over the deprivation period.
4. Three subjects showed bimodal distributions of power in the alpha range. The slower alpha was more prominent in the frontal regions, while the faster alpha was more prominent in the occipital regions.
5. The frequency of the slower frontal alpha was reduced during deprivation, although there was no reduction in power, in contrast to the large reduction in power of the occipital alpha in these subjects.
6. The frontal and occipital alpha showed a regular circadian variation in frequency, with a low point in the early morning and a high point in the afternoon or evening.
7. Some subjects showed a different frequency of occipital alpha when the eyes were open than when the eyes were closed.

8. The alpha which occurs during Stage I REM sleep is about 1 Hz. slower than the waking alpha frequency. During deprivation it is slowed in the same fashion as the waking alpha.
9. Sleep spindles showed a bimodal power distribution, the slower frequency spindle occurring only in the frontal area, while the higher frequency spindle was prominent in both frontal and parietal regions.
10. The sleep spindles showed no reduction in frequency or power over the deprivation period and showed no evidence of a circadian variation in frequency.
11. There appeared to be some increase in the power of the theta rhythm over the deprivation period in two of the four subjects who showed measurable amounts of this activity. There was no evidence of a change in theta frequency.

Effects of Deprivation on the Alpha Rhythm

It is clear from our results that the "alpha rhythm" is not unitary; that is, in some subjects there is a frontal rhythm which differs in frequency from the occipital rhythm. Different frequencies may occur depending on whether the subject's eyes are open or closed, and there is a rhythm which occurs during REM sleep which is slower than that occurring during the waking state. For this reason it is appropriate to discuss the effects of the deprivation procedure on each of these.

various rhythms in turn.

Occipital Alpha

The data from one of the seven-day deprivation subjects (P. W.) will be discussed in detail, as this subject exhibited most of the phenomena of interest. A sample of his EEG record taken on the morning after the first pre-deprivation night is shown in Figure 3. The subject's eyes are closed, and some rhythmic activity at about 10 Hz. can be seen in all channels, although it is of maximal amplitude in the occipital lead. Figure 4 shows a sample of the subject's EEG after seven-days of deprivation. It can be seen from simple visual inspection that the frequency and amplitude of the alpha have been reduced. This is confirmed by power spectra produced from one minute of EEG from the occipital lead in each of these examples, shown in Figure 5. There is a shift in the mode of the dominant peak in the spectrum from an initial 9.75 Hz. before deprivation to 7.0 Hz. after seven days of deprivation. There is also a marked reduction in the power of the alpha. (The peak in the spectrum at 2 Hz. is due partly to tape "wow" and partly to slow activity in the EEG, and no change is seen in this activity.)

Figure 6 shows the modal alpha frequency for subject P. W. throughout the pre-deprivation, deprivation, and post-deprivation period. One can see that the alpha frequency is progressively slowed over the first four days of deprivation, after which there is little further decline.

Recovery is also a progressive process, the alpha frequency gradually returning to normal levels over five to seven days. Figure 6 also shows that there is a striking circadian variation in alpha frequency with a low point in the morning and a high in the evening.

The power of the mode (the height of the alpha peak in the power spectrum) is graphed in Figure 7. The power of the alpha declines to its lowest level on the third deprivation day and remains there for the duration of the deprivation period. Unlike the frequency, however, there is an almost immediate rebound to normal levels on the evening of the first post-deprivation day. The power of the alpha rhythm also appears to have a circadian rhythm with a high in the evening and a low in the morning. In contrast to the circadian variation in alpha frequency which persists throughout the deprivation period, the daily rise in power is not evident after the first deprivation day.

The effect of deprivation on the modal alpha frequency of the two groups of experimental subjects in the eyes closed condition is shown in Figure 8. Deprivation appears to affect the four-day and the seven-day subjects in a similar way: a progressive decline over the first four days of deprivation to a low point, after which only a small further decrease occurs over the following three days in the seven-day group. All of the experimental subjects except P. C. showed evidence of a circadian rise in alpha frequency over the day. Subject P. C. is

anomalous in that the alpha frequency actually rose slightly above pre-deprivation levels in the first two deprivation days before beginning to decline to a low point on the morning of the fourth deprivation day, whereupon it rose sharply to pre-deprivation levels over the fourth day of deprivation. During this period (the fourth day), the subject experienced increasingly intense visual hallucinations, as well as some stress in his personal relationships. Unfortunately, since none of the other experimental subjects experienced this type of hallucinatory activity or stress, it is impossible to say whether its relationship to the rise in alpha frequency is coincidental or not.

Figure 9 shows the effect of deprivation on the power of the alpha of the two experimental groups during the eyes closed condition. All subjects except P. C. and I. T., who had rather low amplitude alpha, showed a reduction in power during the deprivation period. A pilot subject (R. G.) who underwent four days of deprivation in the initial stages of designing the experiment had virtually no alpha in the pre-deprivation baseline recordings (Figure 10), and gradually developed a prominent rhythm of moderate amplitude over the deprivation period (Figure 11). This subsequently disappeared in the post-deprivation period.

So far only the alpha which occurs in the eyes closed condition has been discussed. The alpha which occurs in the eyes open condition

has been analyzed separately for two reasons. Firstly, three subjects had a consistently different frequency of alpha in the eyes open and eyes closed condition, experimental subject W. P. and control subject L. G. having a slower alpha with eyes open, and experimental subject T. S. having faster alpha with eyes open. Secondly, the power of the alpha was much reduced by opening the eyes. These frequency and power differences disappeared when the translucent mask was worn by the experimental subjects. Figures 12 and 13 plot the modal frequency and power for the experimental subjects during the eyes open condition. It can be seen that during the deprivation period the alpha during the eyes open condition reflects essentially the same pattern seen in the eyes closed condition.

The analysis has thus far been in terms of the frequency and power of the mode of the alpha peak in the power spectrum. However, considerable power is present in the neighborhood of the mode, and since the distribution of power in the spectrum in the alpha range is often skewed or distributed so that the power of the mode and the power of the adjacent frequencies are nearly equal, it was thought that the "center of gravity" of the distribution and its area would provide a fairer estimate of the frequency and power of the alpha signal in the EEG. Consequently, the weighted average frequency and the total power present in a range of ± 2 Hz. from the mode were calculated.

These data are presented in Figures 14 and 15 for the eyes closed condition, and Figures 16 and 17 for the eyes open condition. Although there are often small differences between these measures and the results of the previous analysis in terms of the frequency and power of the mode, the earlier picture presented is fully confirmed.

In some subjects there appeared to be a broadening of the spectral distribution of the alpha rhythm during the deprivation period, possibly indicating a decrease in the stability of the alpha frequency. Calculations of the variance of the distribution of power within the range ± 2 Hz. from the mode of the alpha peak showed that the variance did increase in five of the eight subjects (Figures 18 and 19). Subjects P. C., I. T., and T. S. showed no apparent change in the variance during deprivation.

Frontal Alpha

Two of the experimental subjects (W. P. and P. W.) had two distinct frequencies of alpha rhythm, one more prominent in occipital regions, and a slower rhythm which was more prominent in the frontal regions. Figure 20 shows power spectra produced from one minute of the EEG from the four anterior leads shown in Figure 3. The vertical scale has been expanded by a factor of two compared to the occipital spectra shown in Figure 5 in order to show the distribution of peaks more clearly. It can be seen that the dominant occipital rhythm becomes progressively

smaller towards the front of the head, while the rhythm which is dominant at the front of the head appears in all leads, but is of much lower amplitude than the occipital rhythm at the back of the head. We have labeled this the "frontal" alpha, although this is partly a misnomer since activity at this frequency is seen in all the other leads. However, it is usually dominant and appears most consistently in the frontal leads, and, therefore, it is convenient to call it frontal alpha.

Figure 21 shows the modal frequency of the occipital and frontal alpha throughout the experiment. It is apparent that the frontal alpha is also slowed by deprivation, but to a lesser extent. Indeed, in subject P. W. the frequencies merge on the third deprivation day. There is also a circadian variation in frontal alpha frequency similar to that of the occipital alpha. In contrast to the occipital rhythm, however, the frontal rhythm shows no clear change in power over the deprivation period (Figure 22). The great variability in the power of the frontal alpha may be partly due to the fact that the rhythm is not easily distinguished from the occipital rhythm in the polygraph record, and thus it is difficult to select samples with maximal amplitude of frontal alpha. Thus, as mentioned in the procedure section, all records were selected for maximal amplitude of occipital alpha. The fact that many more samples were analyzed over deprivation than in the pre-deprivation period may account for the wider range of power observed in the frontal

rhythm during this period. Obviously, occipital and frontal alpha amplitudes are not highly correlated.

REM Alpha

In some subjects alpha can be seen during sleep rapid eye movement (REM) periods. Unfortunately, these bursts of alpha are usually short and infrequent, occurring only in occasional intervals between eye movements. Consequently, they are difficult to capture on magnetic tape for computer analysis without recording the complete REM period. However, enough samples were recorded from experimental subject W. P. and control subject W. K. to permit a description of the REM alpha and its reaction to deprivation. Examples of the REM alpha of subject W. P. before deprivation and after four days of deprivation are shown in Figure 23. Figure 24 shows power spectral analysis of the occipital alpha in these examples. It is evident that there is a reduction in power and frequency of the REM alpha over the deprivation period equivalent to that which occurred in the waking state. Figure 25 shows the modal frequency of the REM alpha and the waking alpha for subjects W. P. and W. K. In both subjects the REM alpha is consistently about 1 Hz. slower than the waking alpha. Although some exceptions occur, the REM alpha appears to follow the same circadian pattern of a lowering of frequency through the night and a rise during the day, a piece of information made possible by the fact that during the deprivation and control confinement

periods the subjects often slept during the day.

Theta

Zubek et al. (1961) suggested that deprivation produced an increase in theta activity in the EEG. Only half of the experimental subjects showed peaks in the power spectrum which one could label as theta activity (between 5 and 7 Hz.). These peaks appeared most prominently in the spectra of the frontal EEG, although they could occasionally be seen in the other leads, especially central. Figures 26a and b show power spectra of one minute of the EEG of subject W. P. containing peaks in the theta range. In general, these theta peaks are very small, even in comparison with the low amplitude frontal alpha rhythm. Clear cut theta activity is difficult to distinguish in the polygraphic record. The power and frequency of the mode of the theta peak for the four subjects who exhibited such activity is shown in Figures 27 and 28. There is some support for the notion that there is an increase in power of the theta activity, especially in subjects P. W. and W. P., although the scarcity of pre- and post-deprivation points makes the conclusion tentative. There is no evidence of a change in theta frequency with deprivation.

Sleep Spindles

Only five deprivation subjects (W. P., M. R., P. C., D. G., and I. T.) had sleep spindles (bursts of rhythmic activity in the EEG with a

frequency of 12 to 14 Hz.) of sufficient amplitude to produce reliable peaks in the power spectrum. Figure 29 shows a sample of the EEG from subject D. G. during Stage 2 sleep on the third pre-deprivation night. The first three spindles are easily discernable in all EEG channels, while the fourth appears to be confined primarily to the frontal and central leads and is of lower frequency. Power spectral analysis of one minute of this EEG from the frontal and parietal leads reveals two distinct spindle frequencies, one at about 13 Hz. which is widely distributed, and a second at about 11.5 Hz. which is restricted to the anterior leads (Figure 31). Figure 30 shows a sample of EEG from the same subject on the seventh day of deprivation. The power spectral analysis (Figure 31) shows no change in frequency, although by this time the subject's alpha frequency had decreased by approximately 2 Hz. or 20%.

The average spindle frequency for each of the five subjects is plotted in Figure 32. Samples of Stage 2 sleep were recorded on tape for computer analysis at least twice during each pre- and post-deprivation night and during both daytime and night deprivation sleep. The times at which the samples were taken necessarily varied since the spindle frequencies could be determined best in Stage 2 sleep containing little delta activity. However, enough points are available to state that there is no consistent evidence of a circadian variation in spindle frequency. During

deprivation there is no slowing of the spindle frequency. In fact, there seems to be a slight tendency towards higher frequencies, since in every subject this is where the fastest spindle frequencies occur. Finally, there was no apparent effect of deprivation on the power of the spindles, as indicated in Figures 33 and 34, where the modal power of the frontal and parietal spindles is shown.

There appears to be no correlation between spindle and alpha amplitude. Subjects I. T. and P. C. had the lowest amplitude alpha of any of the subjects, yet had excellent spindles. Subjects W. P., M. R., D. G., D. M., P. W., and T. S. all had reasonably high alpha amplitude, yet the first three had high amplitude spindles and the last three had insufficient spindle amplitude to produce reliable power spectra.

The Control Subjects

Zubek et al. (1969) reported that confinement and social isolation produces a small slowing of the alpha rhythm. Figure 35 shows the modal occipital alpha frequency for each of the four control subjects. Subjects W. K. and M. L. appear to show a small (less than 0.5 Hz.) decrease in alpha frequency over the control confinement period. The other two subjects show no appreciable change. The daily circadian rise in alpha frequency can be seen in all subjects.

The modal power of the occipital alpha of the control subjects is

shown in Figure 36. Three of the subjects showed no consistent change in the power of the alpha, while subject M. L. showed somewhat higher alpha power over the confinement period. The weighted average alpha frequency and the total power in the alpha peak calculated over the range ± 2 Hz. from the mode (Figures 37 and 38) confirms these observations. The variance of the alpha peak calculated over the range ± 2 Hz. from the mode was not affected by the control confinement (Figure 39).

In view of the observations of Jasper and Andrews (1938) on the relationship between body temperature and alpha frequency, and the well-known fact that there is a circadian rhythm of body temperature which appears to coincide with that of the alpha frequency (an overnight drop and a daily rise), it was of interest to discover if there was a correlation between body temperature and alpha frequency. Consequently, oral body temperature was measured immediately before the alpha recordings of the control subjects (Figure 40). The weighted average alpha frequency was correlated with body temperature (Table 1). Naturally, such correlations do not imply causality, for circadian rhythms in any two biological variables may be in phase without being causally linked. However, it is interesting that in the post-deprivation recordings of subject L. M. the circadian rhythm in alpha frequency reversed with the evening frequency being low and the morning frequency high. The temperature rhythm also appeared reversed, although, unfortunately,

one data point is missing. This disruption of one rhythm accompanied by an immediate concomitant change in the other is suggestive of some interaction between the two variables.

As noted in the introduction, Lippold has evidence which suggests that the alpha rhythm is generated by variations in the electro-ocular potential induced by eye tremor. Since this theory is obviously of great importance in the interpretation of our results, we decided to replicate one of the experiments which critically distinguishes between the neural and ocular origin of the alpha rhythm. This experiment was an attempt to replicate Lippold's finding that the alpha amplitude is related to the magnitude of the corneo-retinal potential. Only the relative magnitude of the corneo-retinal potential can be easily measured. The technique involves measuring the DC electro-oculogram (EOG) while the subject performs large horizontal eye movements of fixed magnitude. Any change in the absolute magnitude of the corneo-retinal potential should be reflected in a change in the magnitude of the DC EOG. It has been found that the EOG can be made to vary over a two-fold range by reintroducing light following 10 or 15 minutes of dark adaptation (Arden and Kelsey, 1962).

Method: The subject was seated in a comfortable easy chair, and his head was stabilized with a bite bar. Three fixation points (small, dim

yellow lights) were positioned at eye level four feet from the subjects' eyes, one directly in front, the other two 20° to the left and 20° to the right respectively. The subject was asked to move his eyes left, right and center, repeating this sequence at least 10 times, fixating on each point for about one second. The DC EOG was recorded by placing Ag-AgCl electrodes on the inner and outer canthi of each eye. The center fixation point was used to ensure that the left and right fixation points produced symmetrical EOG deflections on either side of zero volts. The EEG and EOG were recorded on a Grass model 7 polygraph. The EOG was recorded with a Grass Model 7PIA DC pre-amplifier and a Model 7DAC driver amplifier. The EEG was also recorded on an FM tape recorder.

Two channels of EEG were recorded bipolarly, one from each side of the head using the parieto-occipital placements described by Lippold (1970). In addition, two monopolar channels were recorded from the occipital electrodes using the right ear as a reference. The left ear was grounded.

The subject was asked to open his eyes, perform the sequence of fixations, and then close his eyes. This was repeated about every two minutes. Thirty-second artifact-free periods of EEG were chosen for analysis immediately before and after each eye movement sequence, when possible. The 30-second periods of EEG were subjected to a

digital power spectral analysis carried out on a PDP 8/E computer. A sampling rate of 50 Hz. and 100 lags used in the computation of the autocovariance produced a spectrum in the range of 0 to 25 Hz. in steps of 0.25 Hz.

A baseline period of 15 to 20 minutes was taken under conditions of normal room illumination. Then the room lights were extinguished for 15 to 20 minutes, the only illumination coming from the dim fixation points. The room lights were then turned on, causing the corneo-retinal potential to rise to a maximum in about 10 minutes. When the corneo-retinal potential returned to baseline levels, the dark-light sequence was repeated once more. This produced two maxima and minima of the corneo-retinal potential.

Results: Measurements of the corneo-retinal potential were very stable and fully replicated the changes in magnitude observed by Arden and Kelsey (1962) and Lippold (1973, p. 113). The values of EOG deflection spanned a two-fold range (Figure 41).

The measurements of power in the alpha frequency range, however, showed a larger variability. Two measures of power were used: height of the mode and the area of the spectrum between 8 and 13 Hz. There appears to be no consistent relationship between the power of the alpha rhythm and the magnitude of the corneo-retinal potential. If

such a relationship were present, one would expect it to be magnified by the power spectral analysis, since the power is related to the square of the signal amplitude, and only linearly related to its abundance. This analysis confirmed visual inspection of the record, which revealed no apparent difference in the abundance or amplitude of the alpha during periods of high and low corneo-retinal potential.

DISCUSSION

The data we have collected are of relevance to three major areas of research. Firstly, our results represent a significant advance in the study of the effects of deprivation on the EEG. We have shown that the slowing of the alpha rhythm is substantially complete by the fourth day of deprivation, and the time course of the slowing is independent of the length of the deprivation period. This slowing involves both the occipital and frontal alpha rhythms and the alpha which occurs during REM periods. In contrast, we found that the other rhythms of the EEG (theta and sleep spindles) show no reduction in frequency. This places some constraints on the theories which may be proposed to explain the effects of deprivation on the EEG.

Secondly, our findings are of relevance to the investigation of the neurophysiological basis of the alpha rhythm. The fact that all manifestations of the alpha rhythm are similarly slowed in frequency by deprivation, while the sleep spindles are not, indicates that these two types of rhythm may not share similar neurophysiological mechanisms as has been commonly supposed (Andersen and Andersson, 1968, 1974). This suggests that the search for a neurophysiological substrate for the alpha rhythm must be undertaken in animals which demonstrate EEG rhythms which are homologous to the human alpha rhythm (e.g., monkeys).

Finally, we have provided a quantitative documentation of some phenomena which are of importance to investigators currently studying the alpha rhythm. Although some of these phenomena have been previously described (e.g., the existence of a frontal alpha rhythm with a different frequency from the occipital rhythm), they have been difficult to quantify by the older methods of analysis, and consequently have received little attention in the current literature. Other phenomena (the circadian variation in alpha frequency, the change in alpha frequency on eye closure and the presence of a distinctly slower rhythm during REM sleep) have not been well documented. Any comprehensive theory of alpha mechanism must be able to account for these phenomena.

Effect of Deprivation on the Alpha Rhythm

Zubek's "expectancy" theory that the rate of slowing of alpha frequency during deprivation depends on the length of time that the subject expects to remain in deprivation was not confirmed in our experiment. The subjects we exposed to four or seven days of deprivation experienced similar degrees of alpha slowing at the end of four days of deprivation (Table 2). The seven-day group showed only a small further decline in alpha frequency in the last three days of deprivation (Table 3). Thus, the rate of change of the alpha frequency did not appear to be affected by the length of the deprivation period.

How can we account for the discrepancy between our results and those of Zubek? He bases his hypothesis principally on two results. First, he found that the alpha frequency at the midpoint of 14 days of deprivation was not slowed as much as the alpha frequency at the end of seven days of deprivation (Saunders and Zubek, 1967). However, as noted earlier, the two groups were not strictly comparable, the seven-day group being confined to bed, while the 14-day group was not. It is possible that this accounts for the difference between the two groups, since Zubek has already shown that exercise has an ameliorating effect on the deprivation-induced alpha frequency shift.

The second study which indicates that the alpha shift may be influenced by the expectancy of the subject is a comparison of the magnitude of the alpha shift due to one, four, or seven days of sensory deprivation (Zubek, Shephard and Milstein, 1970). They showed that each condition produces an alpha shift of about 1 Hz. It is possible that in their sensory deprivation condition the greatest alpha frequency shift occurred over the first day of deprivation, after which there was little further change. Seven of our subjects (excluding P. C.) showed an average shift of 0.77 Hz. in the modal frequency of the alpha by the morning of the second day of deprivation, although in our experiment further alpha slowing occurred on subsequent days. We must conclude that there is little convincing evidence that expectancy influences the

rate of slowing of the alpha rhythm during deprivation.

The deprivation procedure also affected the power of the alpha rhythm in a complex fashion. Subjects with high amplitude alpha showed a large reduction in the power of the alpha rhythm, while subjects with low amplitude alpha showed no change or an increase in alpha power. In fact, the pilot subject (R. G.) who had little alpha in pre-deprivation recordings (Figure 10) developed a substantial amount of alpha of moderate amplitude over the deprivation period (Figure 11). Thus, the reduction in alpha frequency can be dissociated from changes in the power of the rhythm.

The variance of the alpha peak in the power spectrum was calculated in order to determine if an increase in the spread of the peak occurred over the deprivation period, indicating a decrease in the stability of the alpha rhythm. Unfortunately, an increase in variance without a change in stability of the signal might be expected to occur in the presence of large changes in power as the signal becomes closer to the noise on which it is superimposed. Since the subjects who did show an increase in variance also showed a large drop in the power of the alpha, one cannot draw any firm conclusions about the effect of deprivation on the stability of the alpha.

The slowing of the frontal alpha during deprivation was similar to that of the occipital alpha, although the absolute magnitude of the

frequency change was not as great, so that during deprivation the frontal alpha frequency approached that of the occipital alpha. It is interesting that the two deprivation subjects with a frontal alpha which is discernably different in frequency from the occipital alpha had large reductions in the power of the occipital rhythm, while there was no apparent change in the power of the frontal rhythm. The response of the frontal alpha (a low amplitude rhythm) is thus similar to that of low amplitude occipital alpha. Therefore, one may tentatively conclude that the effect of deprivation on the power of the alpha is truly related to its normal baseline amplitude, since both effects (reduction of occipital alpha power and non-reduction of frontal alpha power) are seen in the same subject.

Our finding that alpha which occurs during Stage 1 REM sleep is also slowed by the deprivation procedure indicates that the deprivation effects are not confined to the waking state. This REM alpha is consistently about 1 Hz. slower than the waking occipital alpha; and, unlike the frontal rhythm, this frequency differential is maintained during deprivation, both rhythms showing similar absolute magnitudes of frequency reduction.

There appears to be some relationship between the baseline alpha frequency and the amount of slowing during deprivation, in that subjects with high frequency alpha generally show large reductions in frequency

over deprivation, while subjects with low frequency alpha show smaller reductions in frequency (Figure 42). This generalization also holds within subjects, since the lower frequency frontal rhythm is slowed less than the occipital rhythm. The REM alpha, on the other hand, shows the same reduction in frequency as the waking occipital alpha over the deprivation period; even though it is about 1 Hz. slower.

What mechanisms might be postulated to account for the observed changes in frequency of the alpha rhythm during deprivation? The simple hypothesis that the brain undergoes some general metabolic change is discredited by our finding that the effect of deprivation is specific to the alpha rhythms (frontal, occipital and REM alpha), the theta and sleep spindles demonstrating no reduction in frequency. One may also discount a general change in arousal level as being responsible for the frequency change of the alpha. Firstly, we have found that arousal level does not appear to influence alpha frequency: the alpha frequency is usually similar under aroused and drowsy conditions. Figure 44 shows a sample of EEG from a subject in an alert condition, while Figure 45 shows the EEG of the subject some minutes later in a drowsy condition. Power spectral analysis of the occipital EEG from these two samples reveals no difference in alpha frequency (Figure 46). Secondly, the subjects experienced the greatest lowering of arousal in the first day of deprivation, during which time they doubled their normal

sleeping times. However, sleeping time returned to normal levels by the third or fourth day, at which time the slowing of the alpha was nearly maximal. Furthermore, on termination of deprivation the subjects could in no way be termed hypo-aroused, yet the slowing of the alpha persisted for a number of days.

Since it is known that raising body temperature results in an increase in alpha frequency, and since we have noted a strong correlation between circadian variation in body temperature and alpha frequency, one might propose that deprivation induced a condition of hypo-thermia, which in turn was responsible for the slowing of the alpha rhythm. Unfortunately, we neglected to measure the body temperature of our deprivation subjects. However, we can estimate the magnitude of the temperature change necessary to account for the observed changes in alpha frequency. Raising the body temperature above normal up to a maximum of 105° F. has been reported to produce an acceleration of the alpha frequency at a rate of between 0.25 to 0.60 Hz. per degree. This is in the same range as the frequency-temperature relationship we found within the normal range of body temperature (0.17 to 0.51 Hz. per degree).

Simple extrapolation would predict that the temperature would have to fall about 4° F. to account for the average frequency change seen over deprivation. In other words, the subjects' body temperature would have to fall to about 92° F. (assuming the early morning body temperature

to be about 96° F.). Even a large non-linearity in the frequency-temperature relationship doubling the rate of change to 1 Hz. per degree would require body temperature to be lowered by 2° F. to 94° F. It has been reported that neither body temperature nor basal metabolic rate is altered over four days of deprivation (Heron, 1961). It is unlikely that such a large change in body temperature could have been overlooked.

If one assumed the validity of the neurophysiological model of the alpha rhythm proposed by Andersen and Andersson (1968), one can postulate certain synaptic alterations which might produce the observed effects. Two such changes which might account for our observed results are an increase in thalamic recurrent inhibition and a decrease in tonic thalamic excitation.

An increase in the strength of the thalamic inhibitory recurrent collaterals would produce a longer-lasting inhibition, increasing the intervals between bursts of thalamic activity, resulting in a lower frequency cortical rhythm. However, stronger inhibition would be expected to produce a more widespread recruitment and greater synchrony, resulting in an increase in the amplitude of the cortical potential. This is because stronger inhibition would presumably produce a more complete cessation of firing of the neurons within the field of the inhibitory interneuron, resulting in a greater synchrony of cortical bombardment.

A more complete inhibition of cells at the fringe of the field of the inter-neuron which previously had been only weakly inhibited might produce a more widespread recruitment. However, subjects with initially high amplitude alpha showed a reduction in power of the alpha rhythm.

A slowing of the alpha rhythm could also be produced in this model by a reduction in the tonic excitation of the thalamo-cortical efferents, since recovery from inhibition would thus take longer. This might also account for a reduction in the amplitude of the cortical potentials, since the cortical afferents would fire less vigorously. What kind of mechanism might produce a slowly developing and slowly recovering decrease in tonic thalamic excitation? If one speculates that the source of the tonic excitation (e.g., the reticular formation), like many other neural systems, may be regulated by an inhibitory negative feedback, then a tonic reduction in sensory input would result in a low level of firing of the inhibitory feedback. This might give rise to a gradually increasing supersensitivity to the inhibition, lowering the tonic level of output of the system. Such a supersensitivity would account for the persistence of the effects after deprivation, since the output of the reticular formation would be regulated at a lower level in spite of the increase in sensory bombardment. Unfortunately, such a model would predict that the alpha frequency would vary strongly as a function of arousal, while, in fact, the alpha rhythm merely disappears under conditions of

drowsiness, with little or no change in frequency. It may be, however, that the condition of drowsiness, being related to the onset of sleep, is qualitatively different from a tonically lowered output of the reticular formation during the normal waking state.

The complex interaction of both of these mechanisms (the increase in thalamic recurrent collateral inhibition and the reduction in tonic excitation of the thalamus) might plausibly account for the paradoxical effects of deprivation on the power of the alpha, reducing it in subjects with high amplitude alpha and increasing or not altering it in subjects with low amplitude alpha.

The major objection to this whole hypothesis is that our data show that deprivation produces a profound depression of alpha frequency, with little (or the opposite) effect on sleep spindles. The alpha rhythm exhibits a circadian variation which takes the form of an overnight fall in frequency, while the sleep spindles share no such tendency. Neither could we find any relationship between alpha and spindle amplitude. For example, of the subjects with high amplitude spindles, two (P. C. and I. T.) had low amplitude alpha, and three (W. P., M. R., and D. G.) had high amplitude alpha. Of the subjects with low amplitude spindles, two (D. M. and T. S.) had moderate, and one (P. W.) had high amplitude alpha. Thus, the model of recurrent collateral thalamic inhibition, although convincing as a model for sleep spindles, may not be applicable

to the alpha rhythm.

It can be argued that the frontal alpha rhythm involves neural circuits distinct from those which generate the occipital rhythm. Firstly, it has been shown to have a different ontogeny from the occipital rhythm (Smith, 1939, 1941). Secondly, its distribution over the head is different from the occipital rhythm, which has a large occipital maximum. Thirdly, it appears to be less affected by deprivation, showing a smaller frequency shift and no reduction in power, although this may be due to the fact that it is a low amplitude, low frequency rhythm, and is thus in keeping with the fact that lower amplitude, lower frequency occipital alpha shows less change over deprivation than high amplitude, high frequency alpha.

Is the REM alpha, which is consistently about 1 Hz. slower than the waking occipital alpha, related in any simple way to the rhythms seen in the waking state, or is it a distinct entity? For example, could it merely be the equivalent of the alpha that occurs with eyes open? This is plausible for subject W. P., as it is only slightly slower than the eyes open alpha frequency for that subject. However, the eyes open alpha frequency for subject W. K. is often slightly faster than the eyes closed frequency, yet his REM alpha is consistently about 1 Hz. slower than his waking alpha. Could the REM alpha be related to the slow frontal rhythm? This is unlikely for a number of reasons. Firstly, the REM alpha is almost non-existent in the frontal leads, yet very

prominent in the occipital region. Secondly, it is initially of substantially higher frequency than the frontal alpha in subject W. P., yet it falls to a lower frequency at the end of deprivation. The frontal alpha frequency in this subject is minimally affected by deprivation. Thirdly, the frontal alpha of subject W. K. is the same frequency as the occipital alpha, which is, as noted above, about 1 Hz. faster than the REM alpha. Is the REM alpha then simply a slowed-down version of the waking occipital rhythm? There is tentative evidence from subject W. K. that this is not the case either. One could occasionally see a lower amplitude, higher frequency rhythm during the REM period that was also largely confined to the occipital leads. The frequency of this rhythm coincided with that of the waking occipital alpha, and so one can tentatively identify it as the version of the waking alpha which occurs during REM. These two rhythms occasionally appeared to co-occur during the REM period (see Figure 43) allowing one to suggest that the slow REM alpha is a distinct rhythm peculiar to the REM period. The fact that it has the same spatial distribution as the waking occipital rhythm and is affected by deprivation to a similar extent indicates that they may share common circuit elements.

Characteristics of the Human EEG

The excellent capacity of the computer-generated power spectrum to resolve the frequency components of the EEG, and its relative ease

of application has enabled us to produce good quantitative documentation of some phenomena which have previously been unrecognized, or described only with difficulty due to the inadequacies of the traditional methods of analysis (e.g., wave counting by hand, or relatively broad band electronic filtering). For example, it is immediately apparent from our results that some subjects exhibit two distinct frequencies of alpha rhythm concurrently. These two rhythms appear to have different spatial distributions over the head, the occipital alpha having a strong occipital maximum, while the slower "frontal" alpha has approximately equal amplitude in occipital and frontal regions. It is more prominent than the occipital alpha in the frontal region due to the great reduction of occipital alpha amplitude in this area. It has previously been noted that the alpha frequency in frontal and central regions may differ from that in the occipital area (Jasper and Andrews, 1938; Rubin, 1938b); indeed, Smith (1939, 1941) has shown that it has a distinctly different ontogenetic development. However, with their methods of analysis it was difficult to identify the two frequency components in the occipital area where the occipital rhythm dominates, and so the full extent of this rhythm has remained unrecognized. We have observed this distinct frontal rhythm in three subjects out of 12. This raises the question of whether or not the other eight subjects exhibit such a rhythm, whose frequency is nearly equal to that of the occipital alpha. Identification of

such a rhythm would require a careful analysis of the phase relationships between the alpha in frontal and occipital regions, and would only be clear in cases where the occipital rhythm was of negligible amplitude in frontal regions. In such cases one would expect the phase relationships between frontal and occipital regions to be essentially random over long samples if two independent rhythms exist. As we have seen, Remond (1969) and Lehmann (1971) have shown that the occipital rhythm normally consists of an anterior-posterior quasi-standing wave with the result that the frontal aspect of the occipital rhythm is usually 180° out of phase with the occipital regions.

We have noted distinct and reliable frequency changes in the occipital alpha on opening the eyes. It is difficult, however, to postulate a generalized mechanism, since two subjects showed a lower frequency with eyes open and one subject showed a higher frequency. These frequency changes (as well as the classical "alpha blocking" reduction in amplitude) seem to be related to patterned visual input, since the alpha of the subjects wearing the translucent mask is identical with eyes open or closed.

All manifestations of the alpha rhythm demonstrate a circadian variation in frequency with the lowest frequencies occurring in the early morning and the highest occurring in the afternoon or evening. Bjerner (1949) noted this phenomenon while doing all-night EEG recordings on

sleep-deprived subjects. However, the fact that his subjects were sleep deprived makes interpretation difficult. The present study shows the circadian rhythm in alpha frequency, like the rhythm in body temperature, is independent of the subjects' sleep. It is possible that the circadian variation in alpha frequency is related to the body temperature rhythm. We have noted in the control subjects that the two variables are well correlated, and that the rate of change of alpha frequency with respect to body temperature within the normal daily range (0.17 to 0.51 Hz. / degree) agrees well with the rate of change of alpha frequency when the temperature is artificially elevated outside the normal range (0.25 to 0.60 Hz. / degree) (Jasper and Andrews, 1938; Hoagland, 1936). Furthermore, one subject (L. M.) demonstrated a simultaneous inversion of both body temperature and alpha frequency rhythms in the post-control recordings.

Frank, et.al. (1966) reported a circadian periodicity in EEG "abundance", a measure produced by summing the outputs at all frequencies of an electronic EEG frequency analyzer. However, they averaged the data from 16 subjects recorded simultaneously over a period of 30 hours, recordings being taken every three hours. This procedure may be criticized on the grounds that changes in EEG may be a function of time in the experiment rather than time of day. Circadian rhythms are more satisfactorily demonstrated by recording at regular

intervals over a number of consecutive cycles.

They were unable to show a return to initial levels of EEG abundance at the end of the experiment, as both highest and lowest levels were found at about 9 a.m. Our subjects that showed evidence of a periodicity in alpha power had the highest amplitude alpha in the evening and the lowest in the morning.

SUMMARY

We have been unable to confirm Zubek's hypothesis that longer deprivation periods produce a more gradual slowing of the alpha rhythm, the four-day and seven-day deprivation subjects showing a similar time course in alpha slowing over the first four days of deprivation. We have shown that the alpha rhythm is not unitary, and that the other alpha rhythms (the slower frontal alpha and REM alpha) are also slowed by deprivation and show a circadian variation in frequency similar to that of the occipital alpha. In contrast, the sleep spindles were found to be unaffected by the deprivation procedure.

Serious hypotheses about the mechanisms underlying the phenomena we have recorded must await a detailed analysis of the mechanism of the alpha rhythm in monkeys analogous to the studies which have been done concerning sleep spindle mechanisms in cats. The EEG of monkeys

is ontogenetically and morphologically similar to human EEG and the of such experiments could thus be applied to humans with confidence. Although the difficulties of carrying out this research would be formidable as they would entail chronic single cell recordings in awake animals, such work is essential to understanding the basis of the differences between the alpha rhythm and sleep spindles, e. g. their occurrence in different states of arousal, the different cortical distribution of fast and slow alpha and fast and slow spindles, the different ontogeny of spindles and alpha, the difference in circadian variation of alpha and spindles and finally their different response to deprivation. In fact, deprivation should prove to be a particularly useful tool in distinguishing between the circuits involving the two rhythms as it alters one rhythm without affecting the other.

Figure 1

The deprivation chamber, showing the subject wearing the translucent mask and cardboard tubes during deprivation.

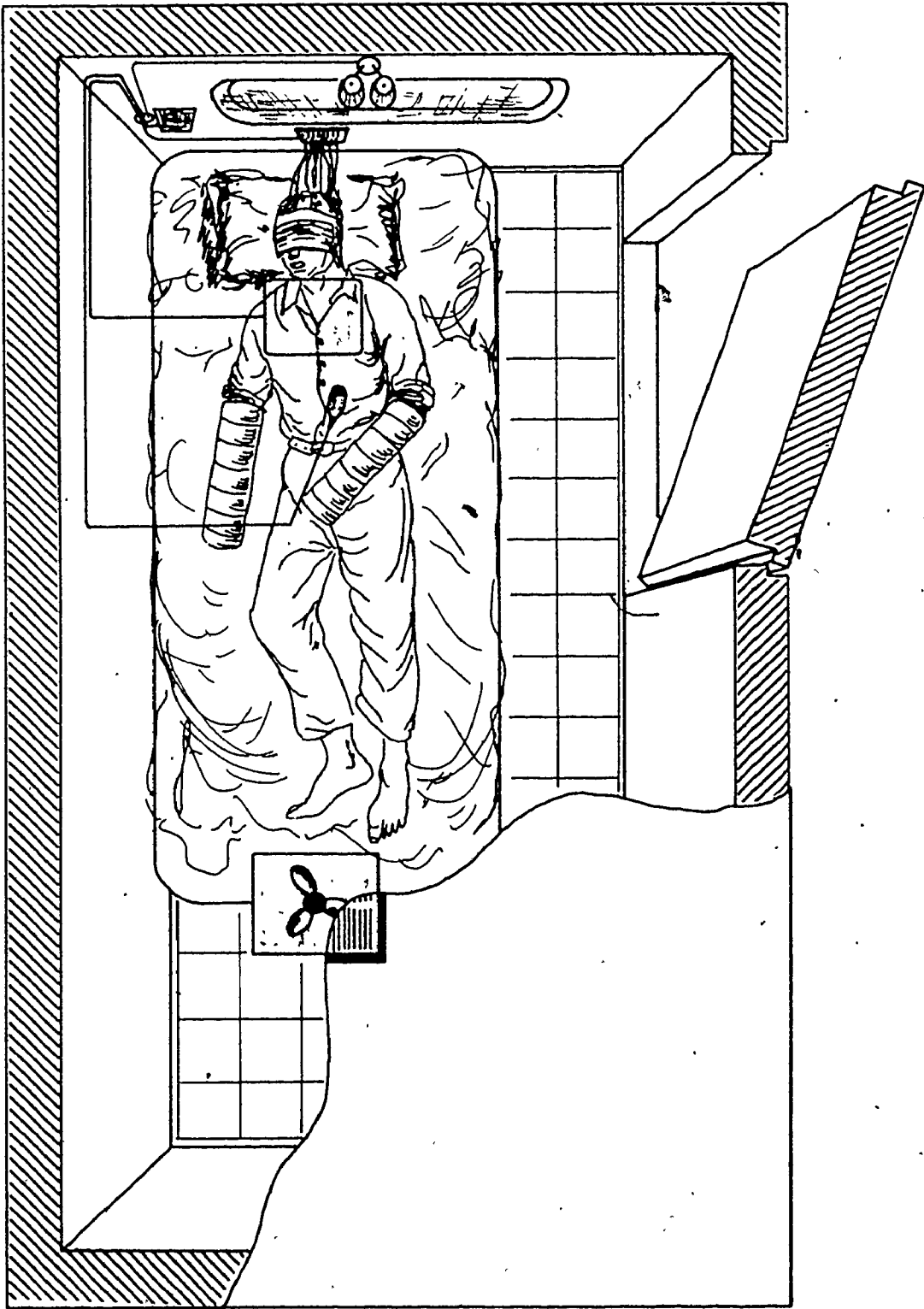
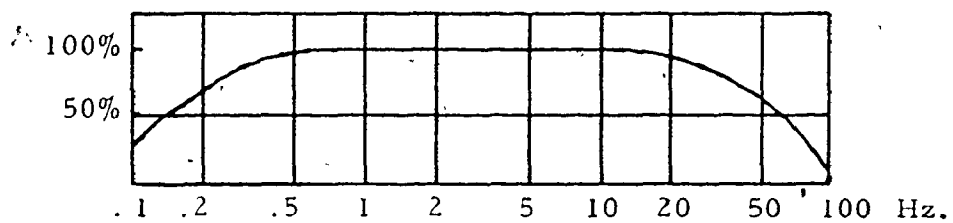
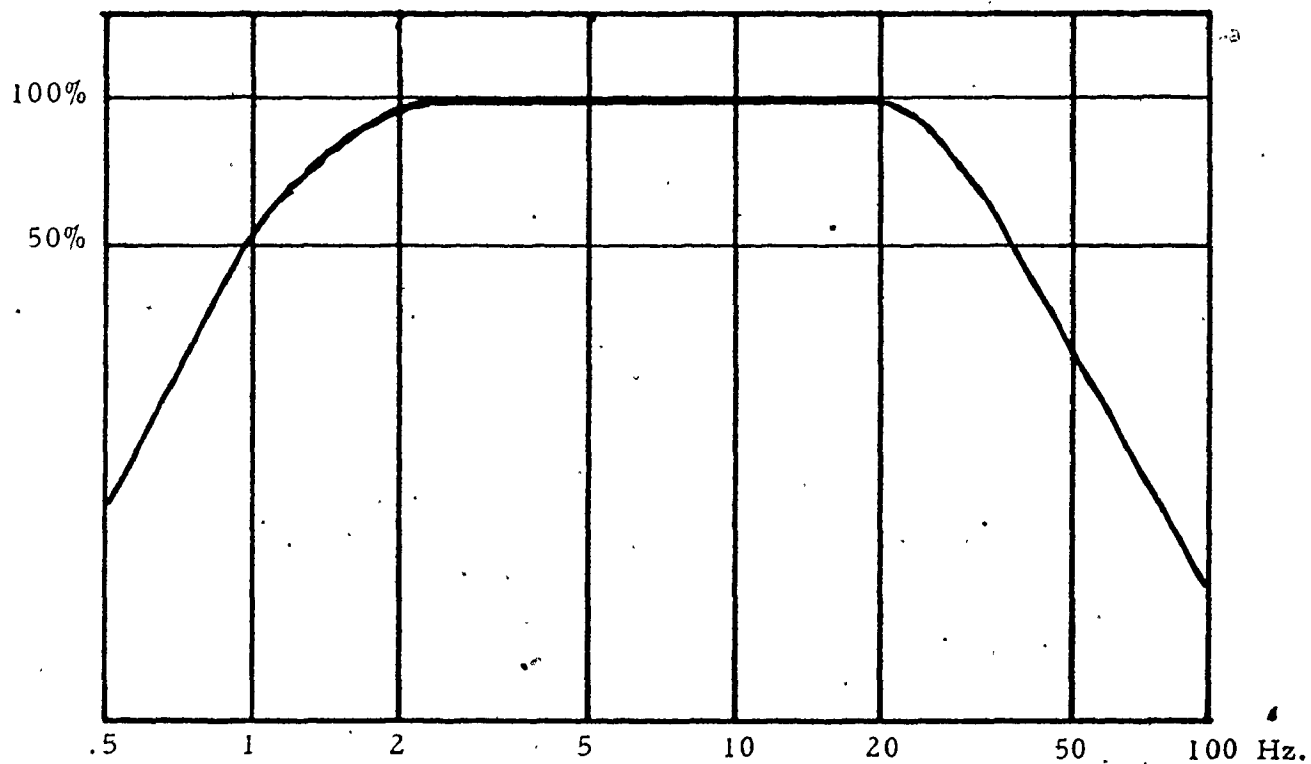


Figure 2



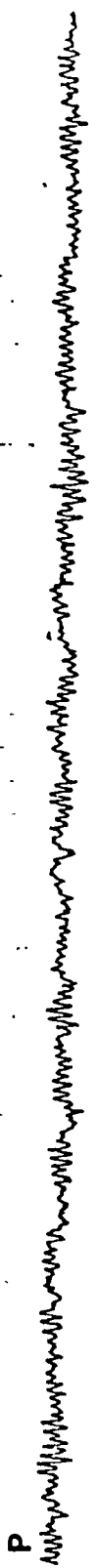
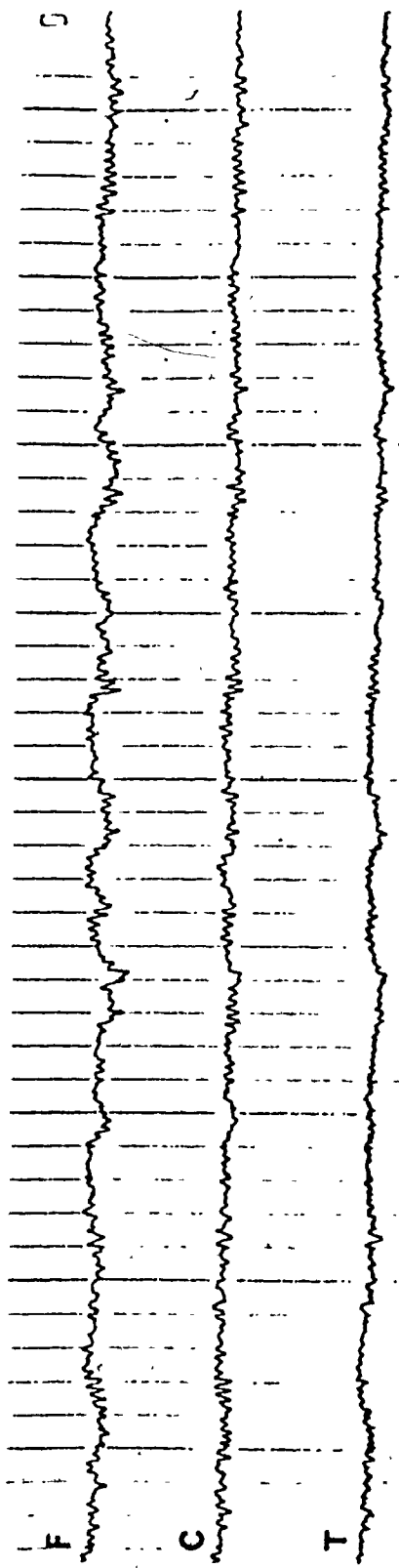
Frequency response curve of Grass Model IV S polygraph with "LO" filter at 0.3 Hz. and the "HI" filter at 35 Hz. (Redrawn from Grass instruction manual.)



Frequency response of the filter used prior to digitization of the EEG signal.

Figure 3

A sample of the EEG recorded from subject P. W. on the morning of the first pre-deprivation night. The subject's eyes are closed.



EMG

Subj.: P. W.

100 μ v
1 sec.

Pre Dep. 1 Morning Eyes Closed

Figure 4

A sample of the EEG recorded from subject P. W. on the morning of the seventh day of deprivation. The subject's eyes are closed.

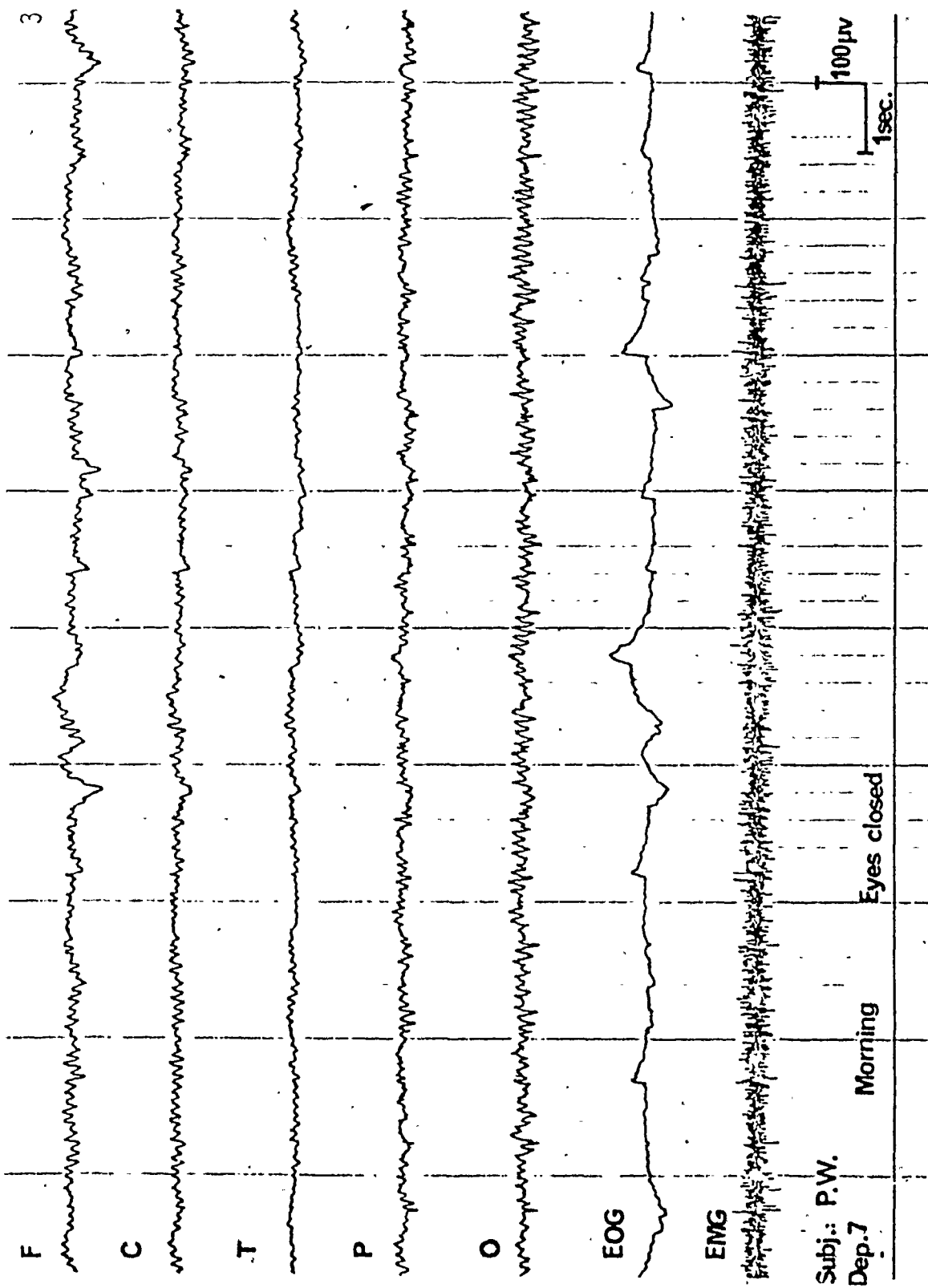


Figure 5

Power spectra produced from 1 minute of EEG from the occipital lead in each of the preceding examples (Figures 3 and 4). The ordinate values represent the power present at each frequency in 0.25 Hz. increments along the abscissa. The ordinate values are related to the power (μV^2) by a constant which depends on the amplification at each stage of the recording (polygraph and tape recorder) and playback (filter, oscilloscope and attenuator). Conversion of the computer generated ordinate values to μV^2 is difficult and inaccurate and is not practised in the published literature. Comparisons of power within the experiment can be made since the gains have been kept constant except in special circumstances which will be noted

SUBJ. P.W. OCCIPITAL ALPHA

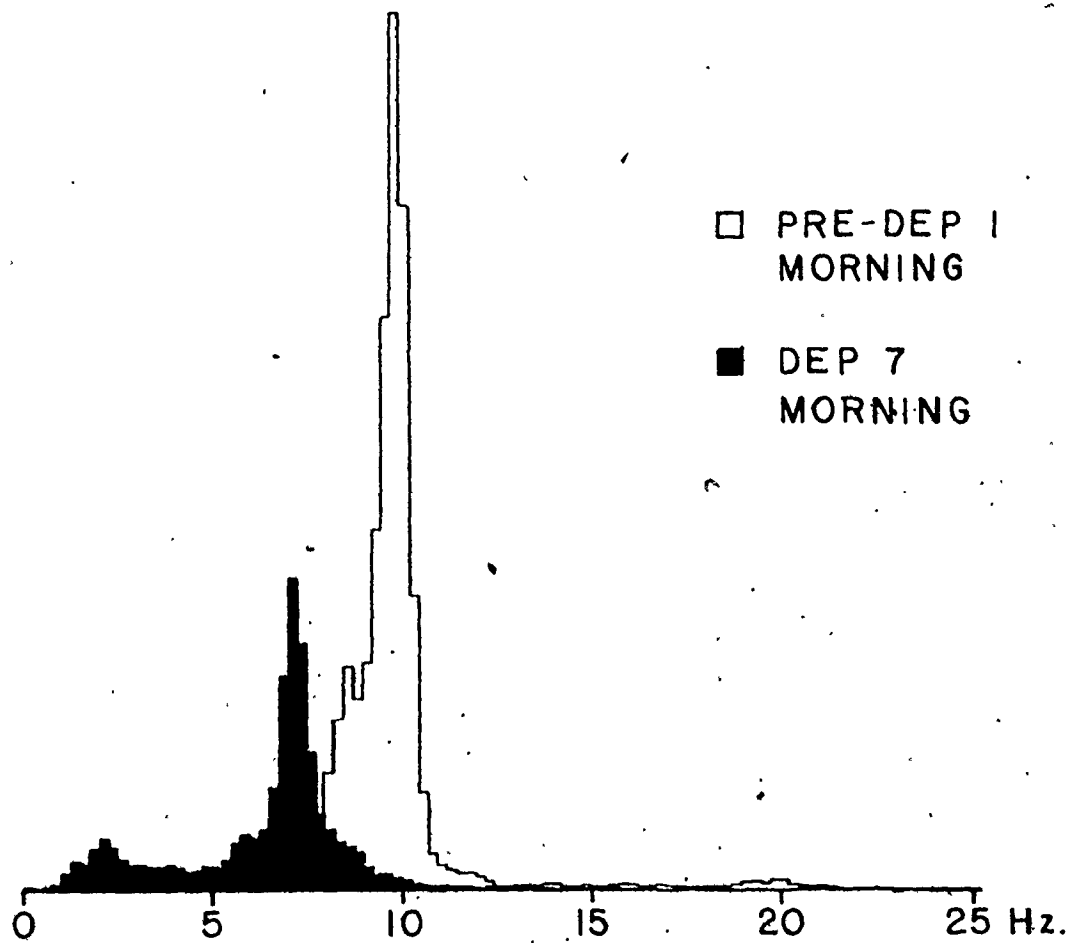


Figure 6

Modal alpha frequency of subject P. W. during the eyes closed condition. Each point in the graph represents the average modal frequency of the power spectra produced from two 1 minute samples of occipital EEG taken with the subject's eyes closed. The pre-deprivation points were recorded at midnight and 8:00 a. m., while the deprivation points were recorded before and after each meal (8:00 a. m., 1:00 p. m. and 7:00 p. m.). The post-deprivation points were recorded one and two hours after the termination of deprivation, at midnight and 8:00 a. m. on each of the three recovery nights and at noon on post-deprivation days 5 and 7. (Data points missing due to technical or experimenter error are indicated by gaps in the graph: e.g., after supper on Day 6.)

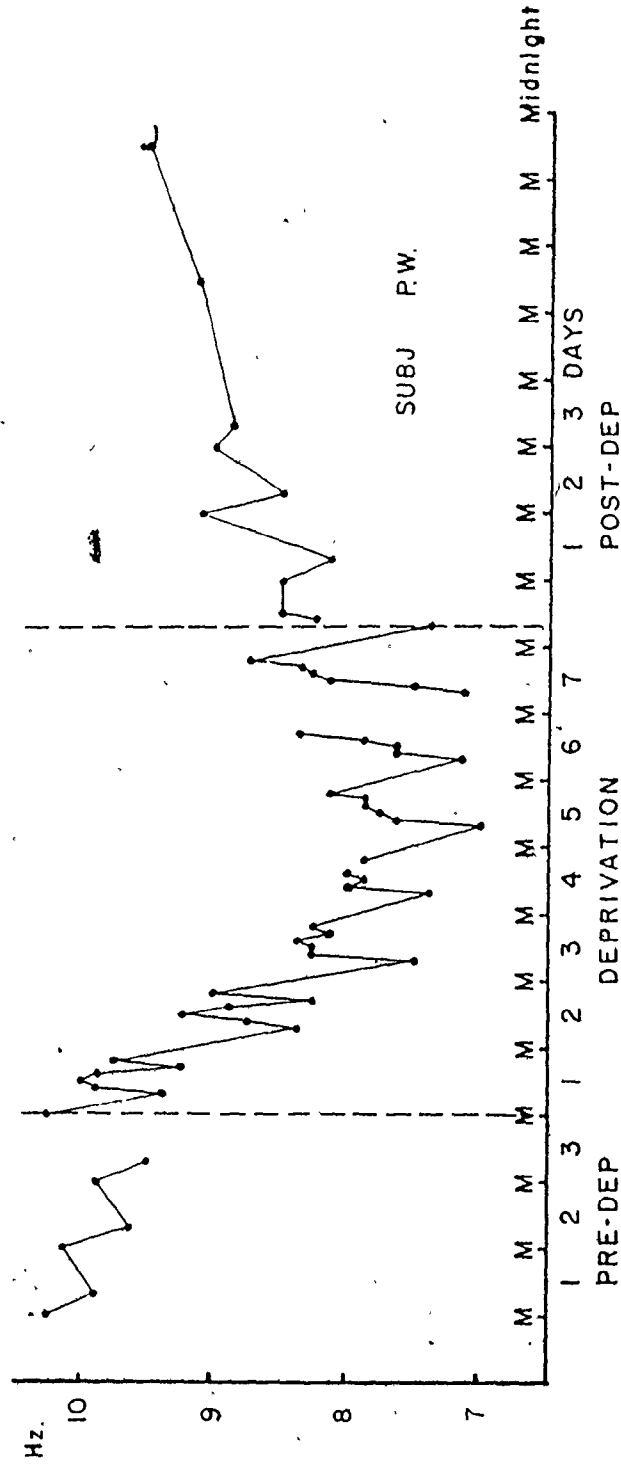


Figure 7

The modal power of the occipital alpha rhythm of subject P. W. in the eyes closed condition. Each point is the average from two spectra.

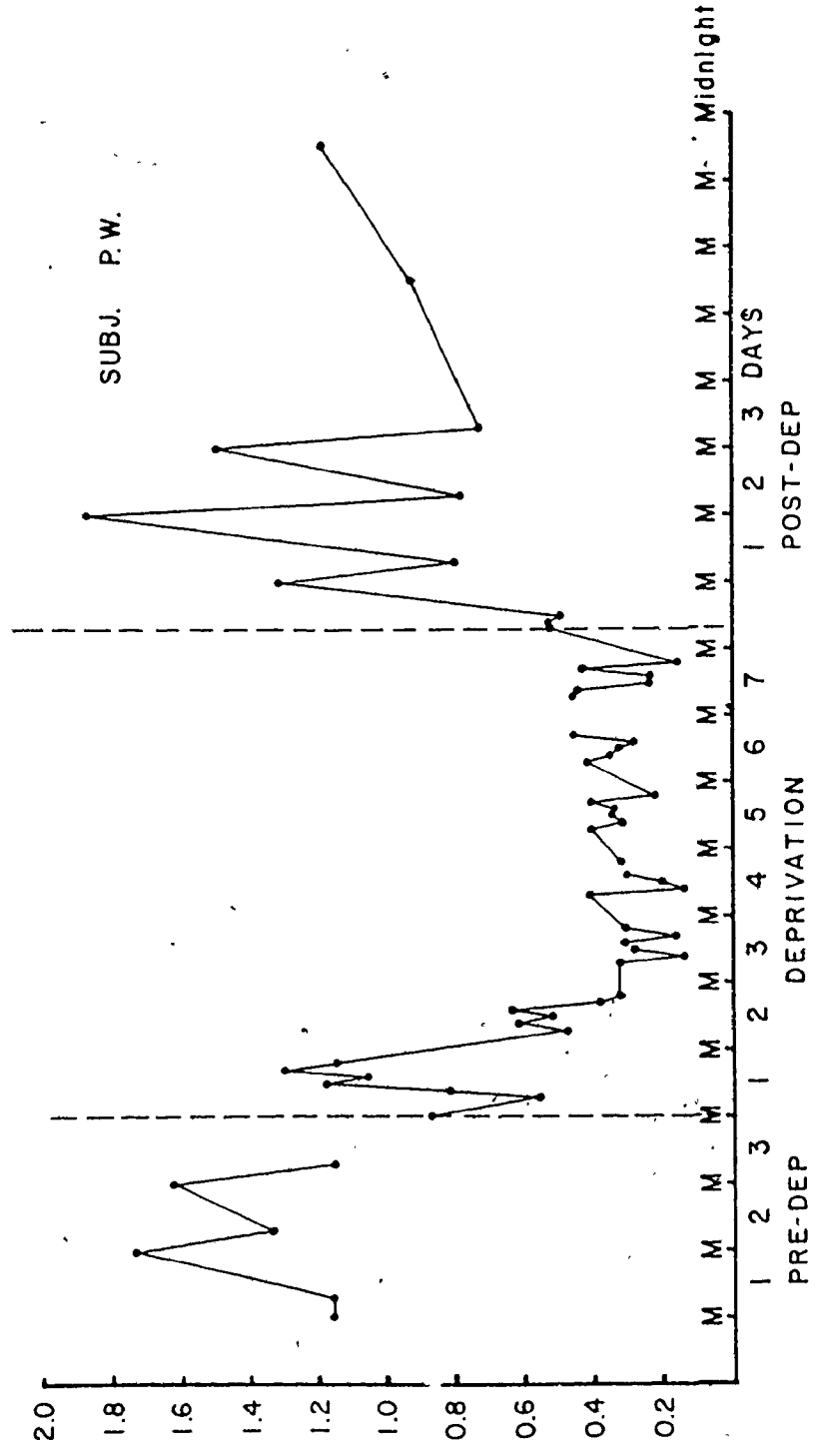


Figure 8

The modal alpha frequency of the four-day and seven-day experimental groups. Each point is the average of the modes of two spectra taken from the occipital lead in the eyes closed condition. (The deprivation period of subject D. M. had to be cut short at noon on the fourth deprivation day due to an unforeseen social commitment, yet his data appear to conform with that of the other subjects and so was not rejected.)

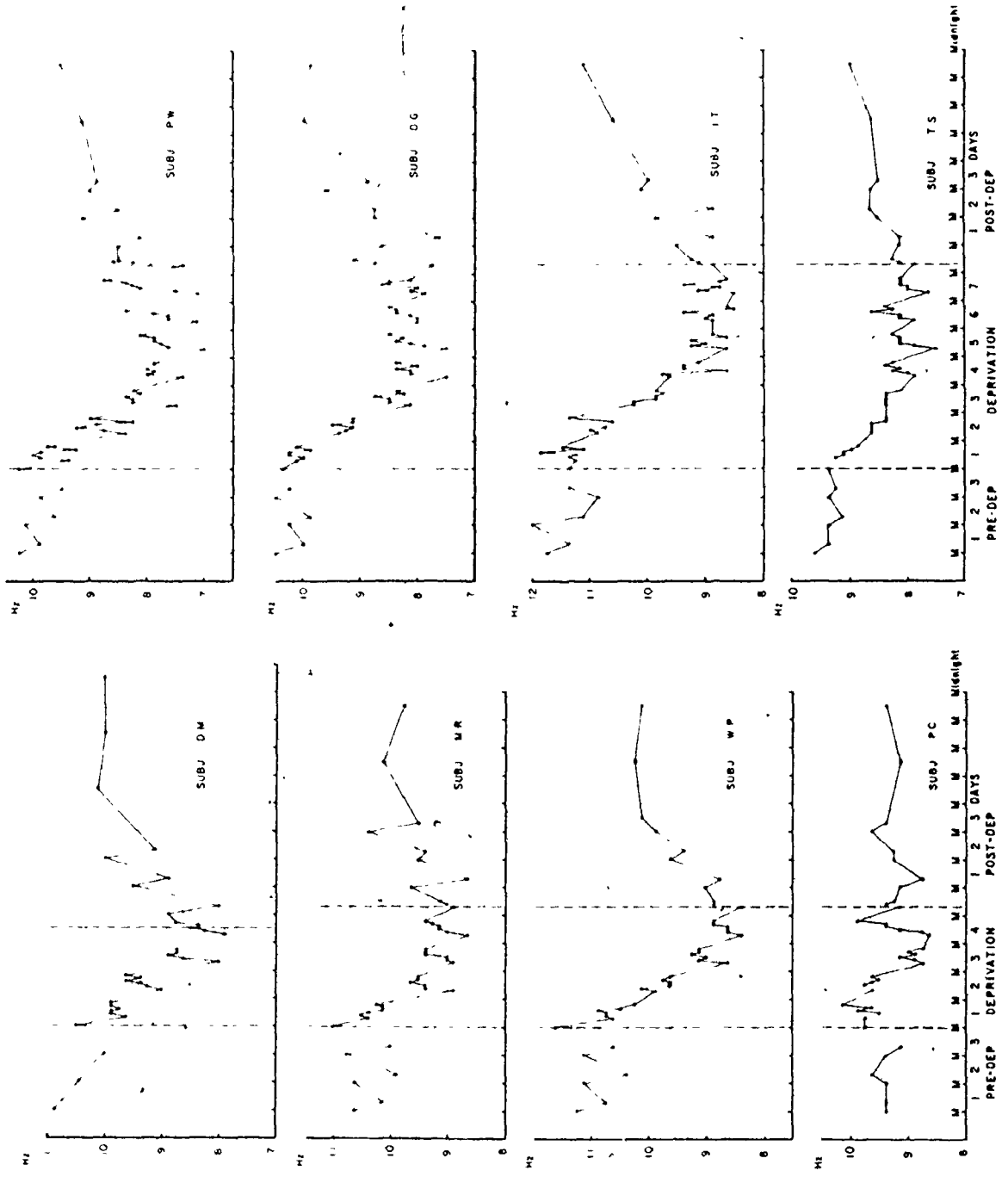


Figure 9

The modal power of the occipital alpha of the four-day and seven-day experimental groups during the eyes closed condition. (Note: The amplification of the EEG from subject I. T. was doubled during playback for computer analysis. This has the effect of quadrupling the power of the signal. Thus the scale of the graph of power has been reduced by a factor of four to make it comparable to the other graphs in the diagram.)

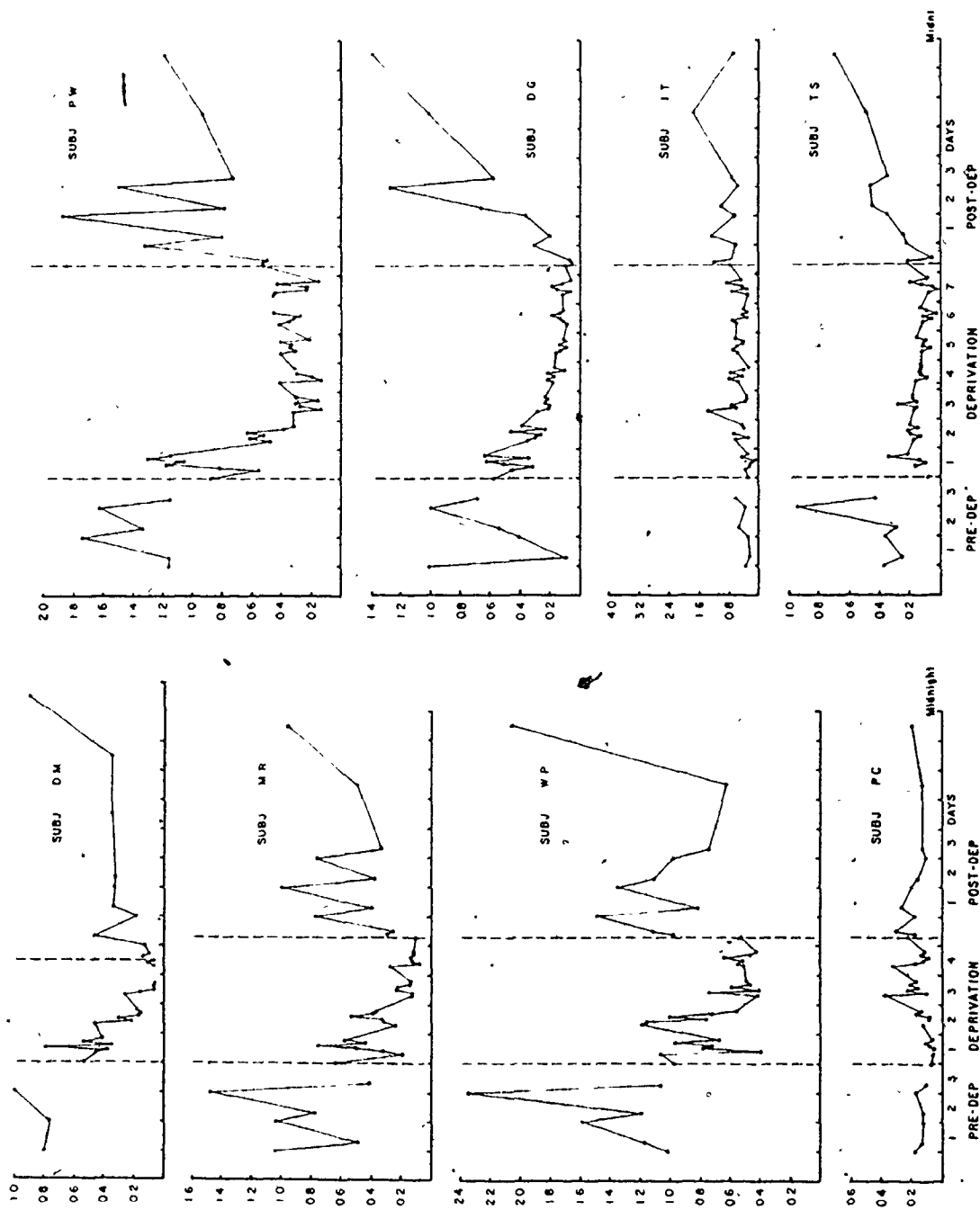


Figure 10

A sample of the EEG of subject R. G. recorded on the evening of the third pre-deprivation night. The subject's eyes are closed.



2

F

C

T

P

Q

EOG

EMG



Subj.: R.G.

100 μ v.

1 sec.

Pre-Dep.

0

Figure 11

A sample of the EEG of subject R. G. recorded on the afternoon of the fourth deprivation day. The subject's eyes are closed.



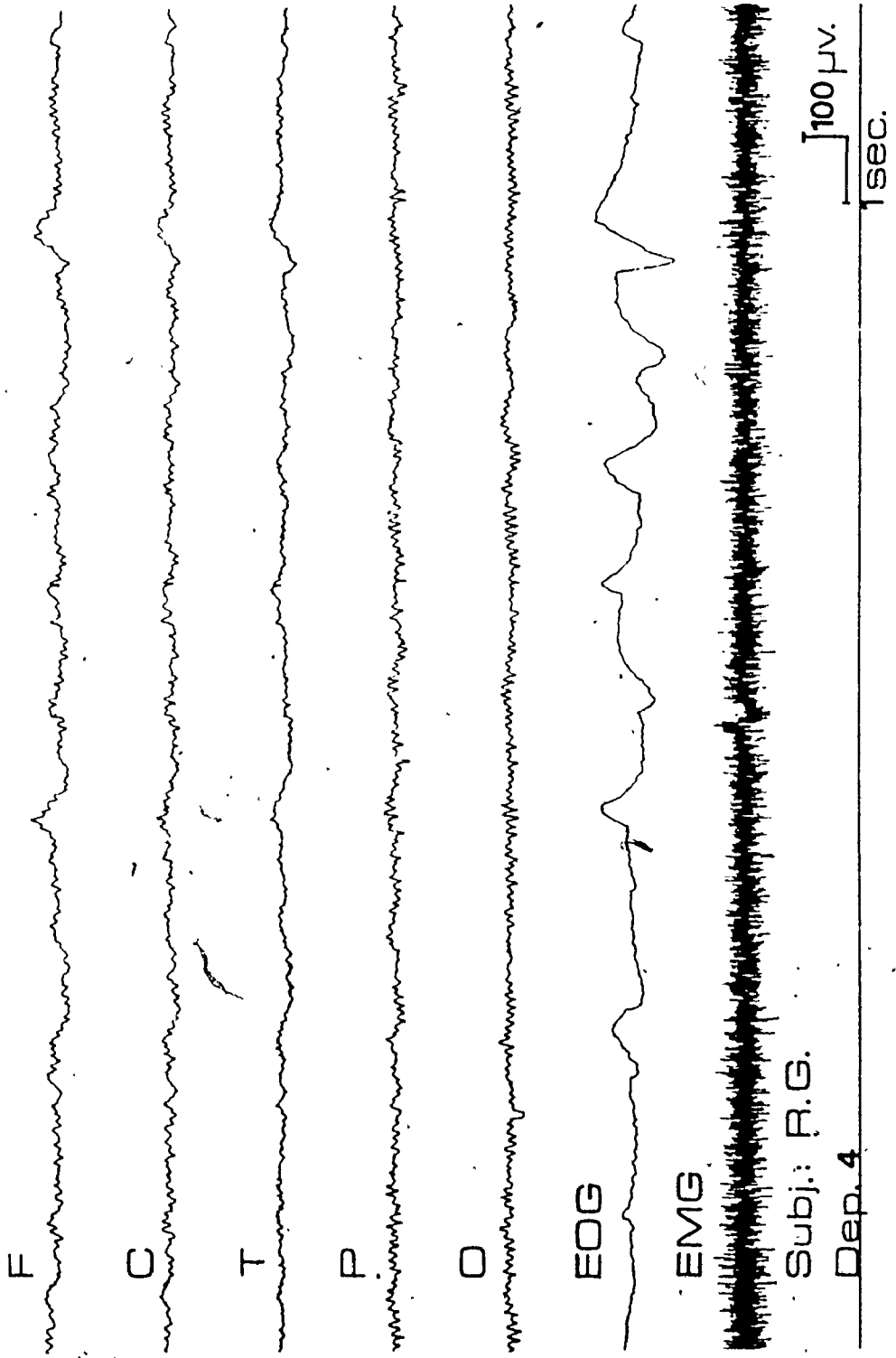


Figure 12.

The modal frequency of the occipital alpha of the four-day and seven-day experimental subjects in the eyes open condition. Missing data points in the pre- and post-deprivation periods are due to insufficient alpha to produce a reliable peak in the spectrum.

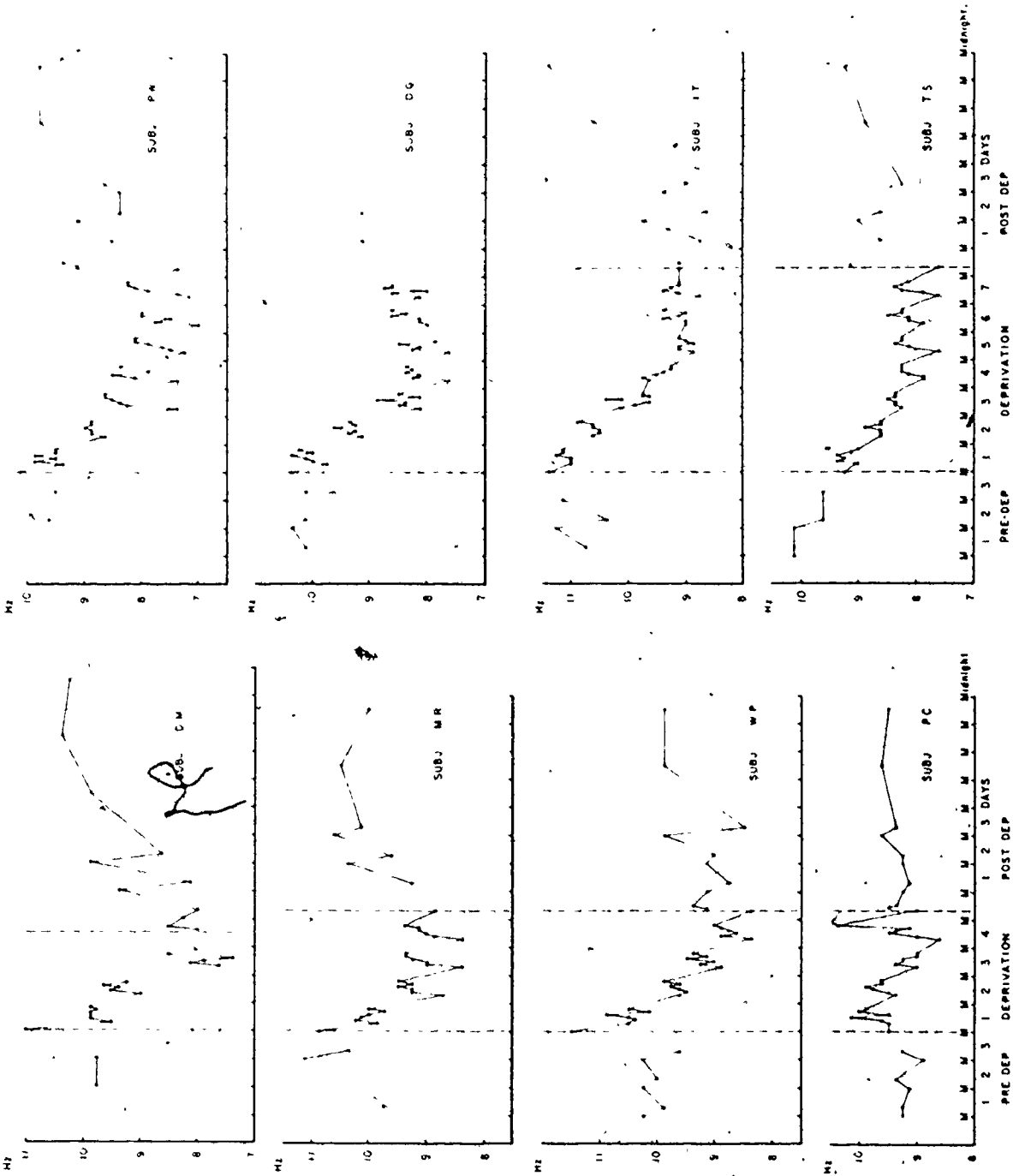


Figure 13

The modal power of the occipital alpha of the four-day and seven-day experimental subjects in the eyes open condition.

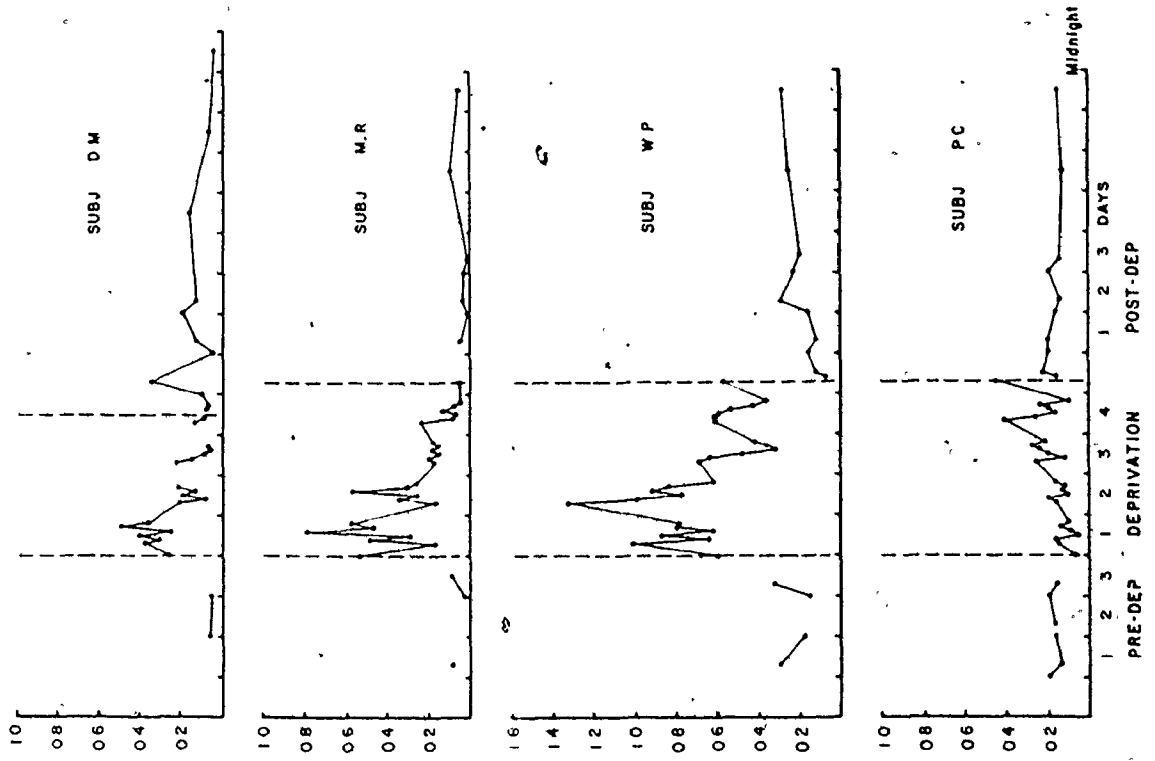
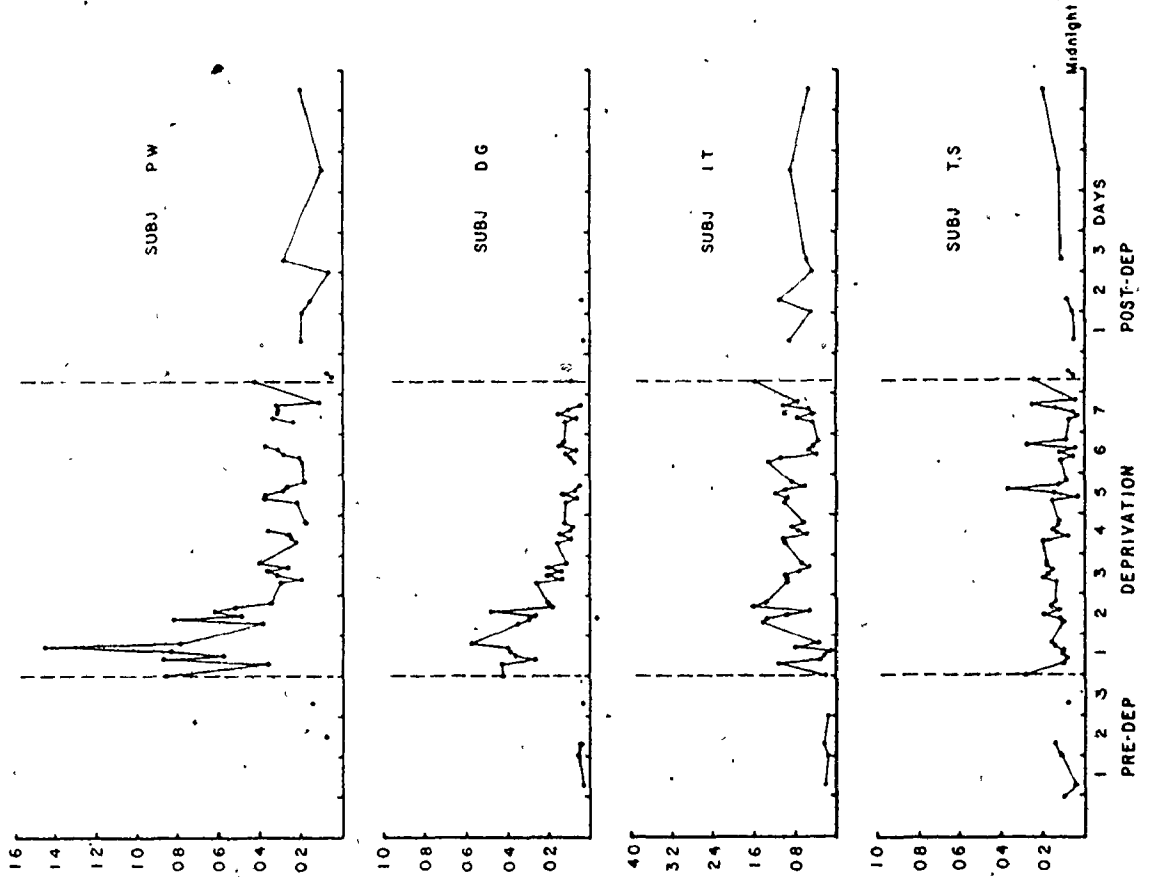


Figure 14

The weighted average frequency of the occipital alpha in the range ± 2 Hz. from the mode of the alpha peak in the power spectrum. Each point represents the average from two samples taken in the eyes closed condition.

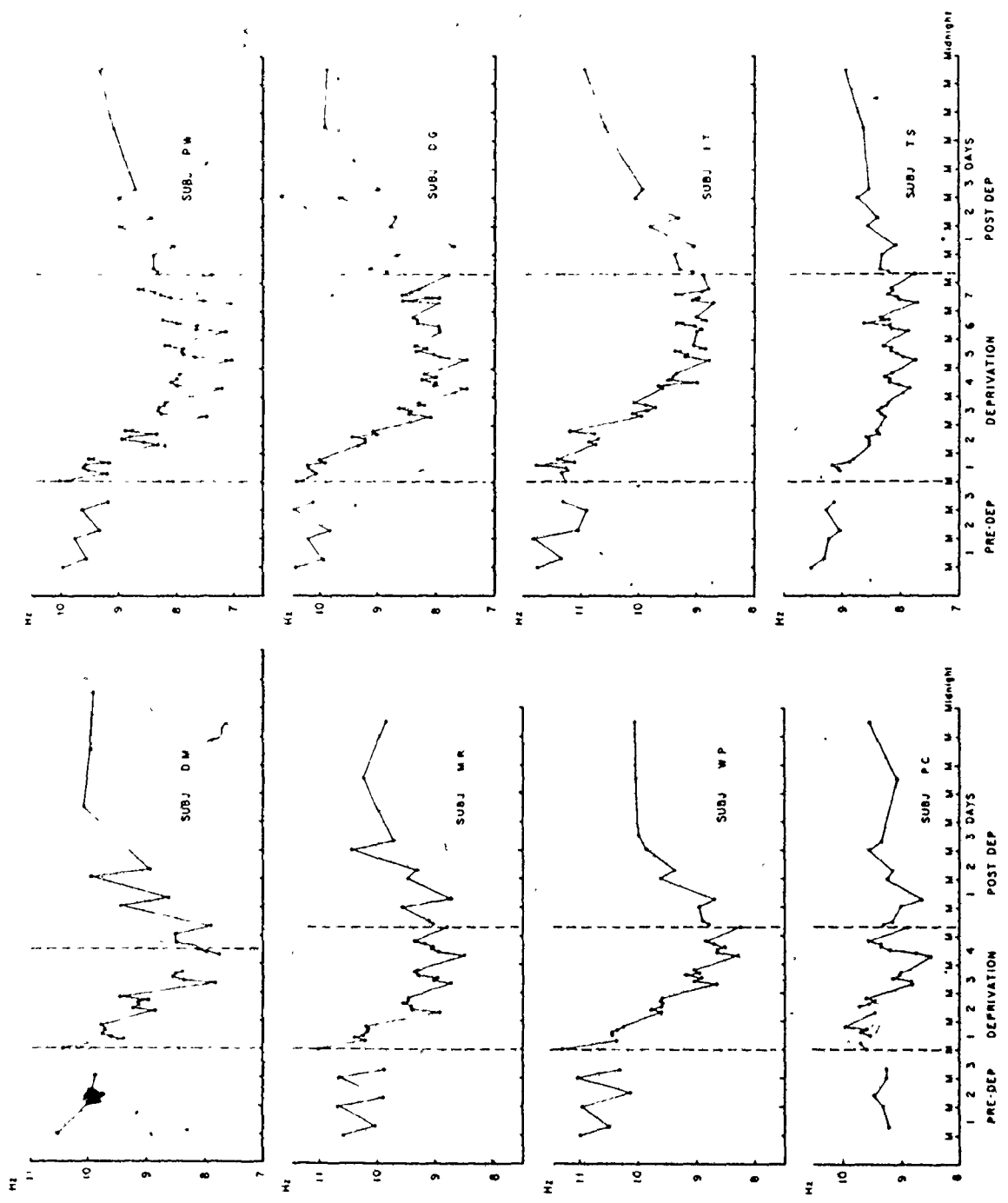


Figure 15

The total power in the spectrum in the range ± 2 Hz. from the mode of the alpha peak. Each point represents the average from two samples of occipital EEG taken in the eyes closed condition.

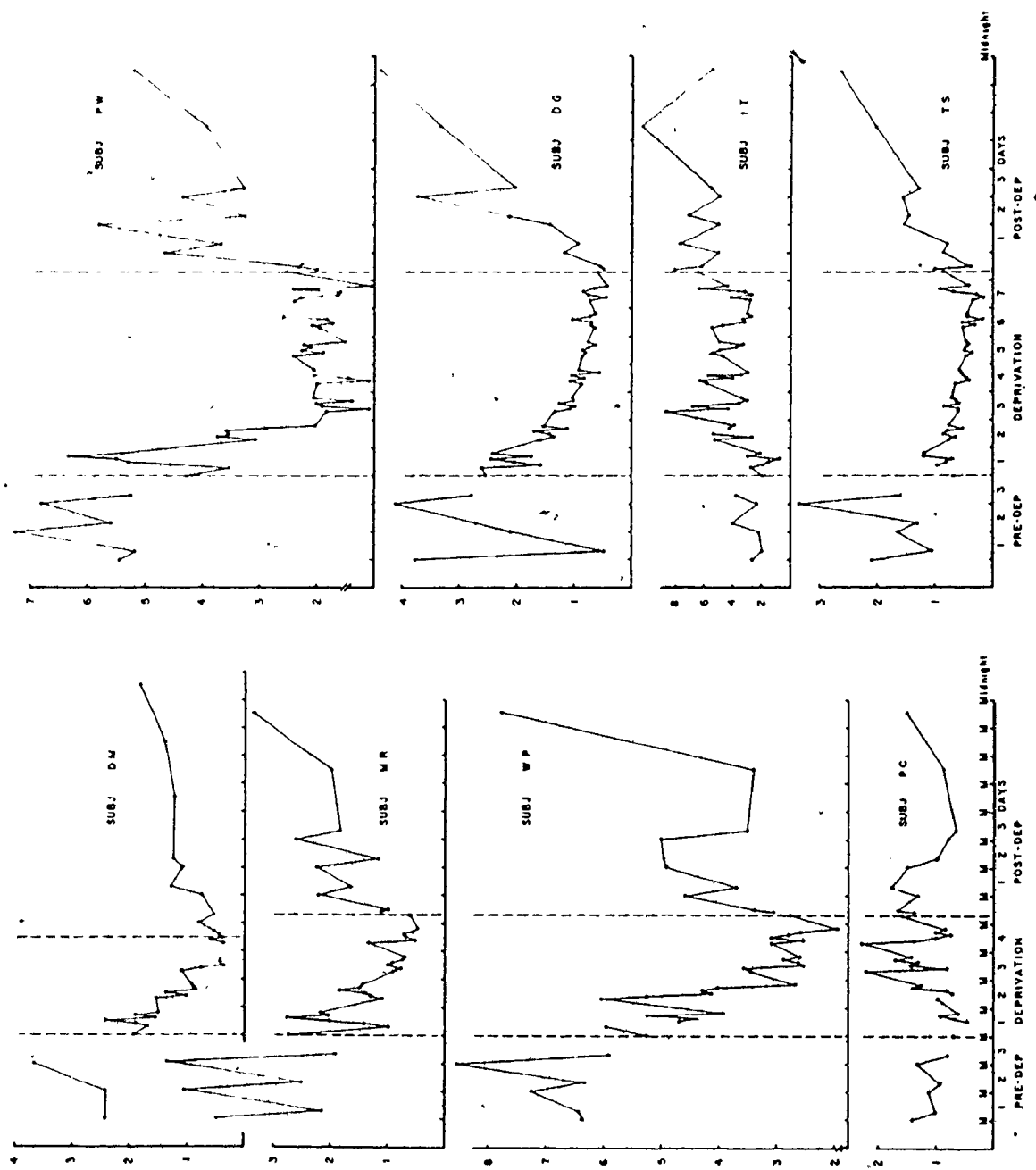


Figure 16

The weighted average frequency of the occipital alpha in the range ± 2 Hz. from the mode of the alpha peak in the power spectrum. Each point represents the average from two samples taken in the eyes open condition.

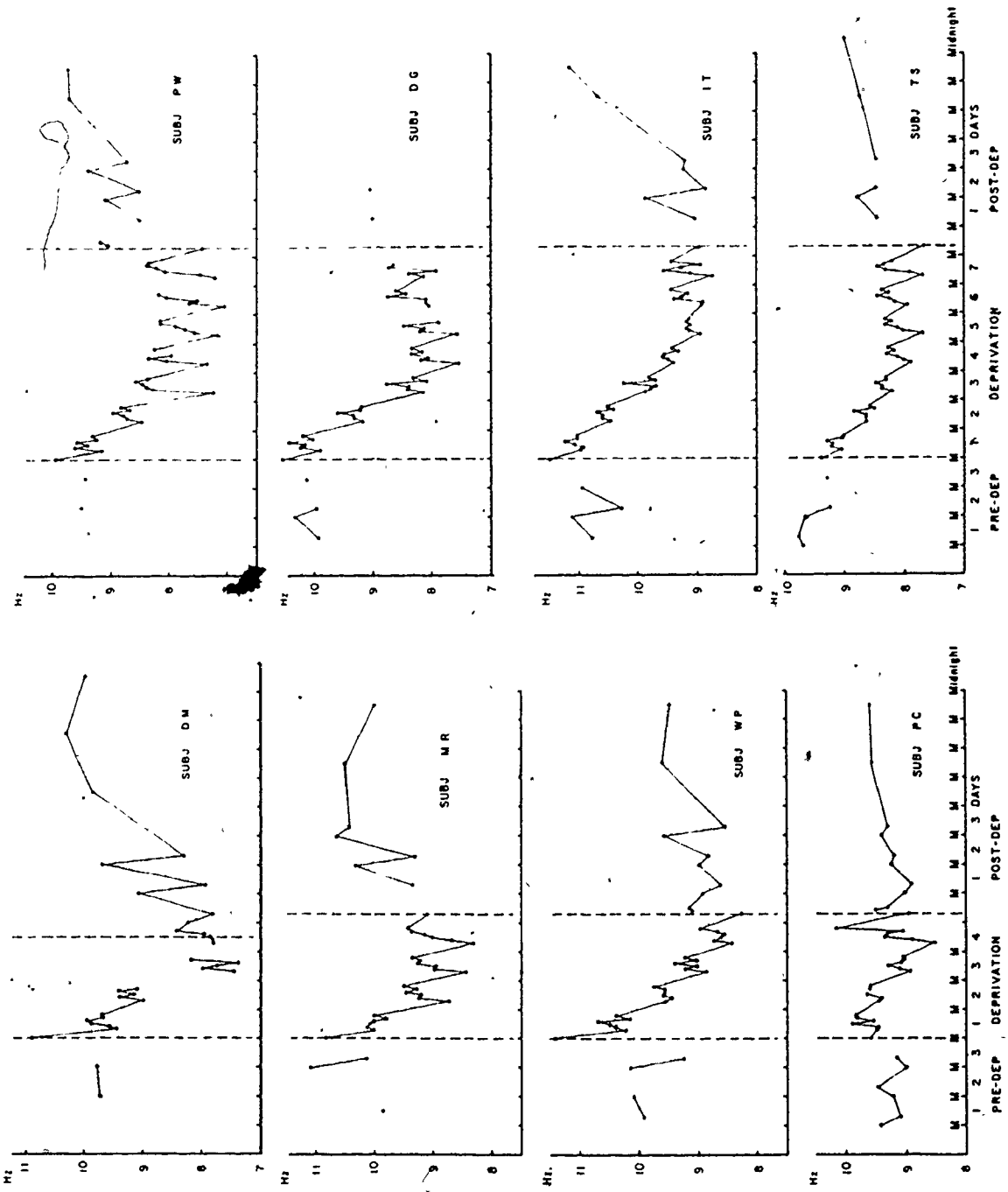


Figure 17

The total power in the spectrum in the range ± 2 Hz. from the mode of the alpha peak. Each point represents the average from two samples of occipital EEG taken in the eyes open condition.

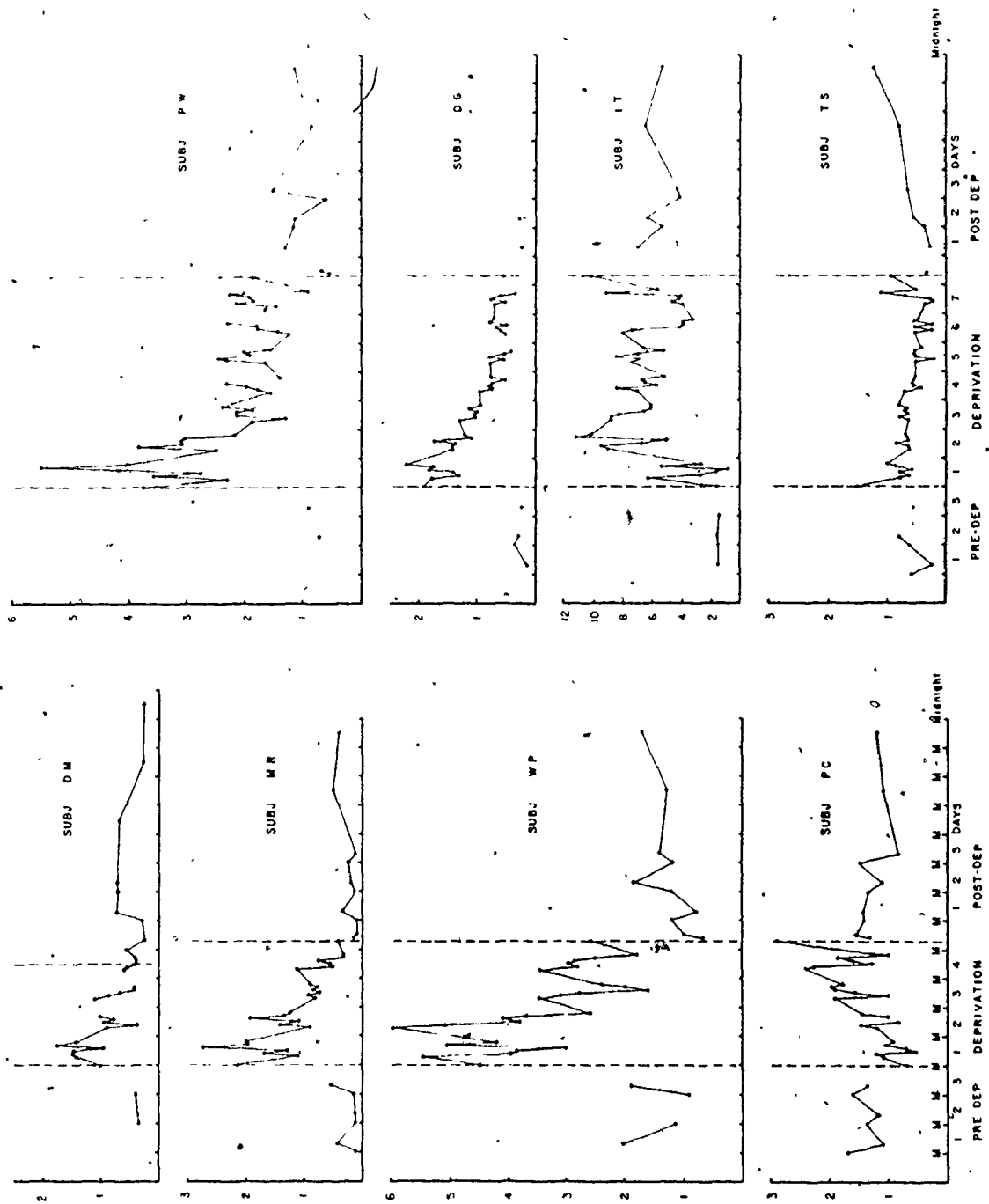


Figure 18

The variance of the distribution of power in the range ± 2 Hz. from the mode of the occipital alpha peak in the power spectrum. Each point represents the average from two spectra taken in the eyes closed condition.

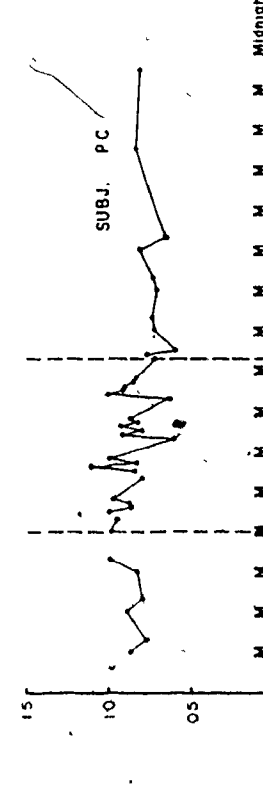
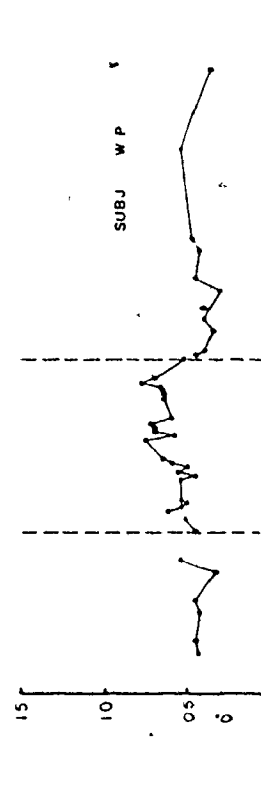
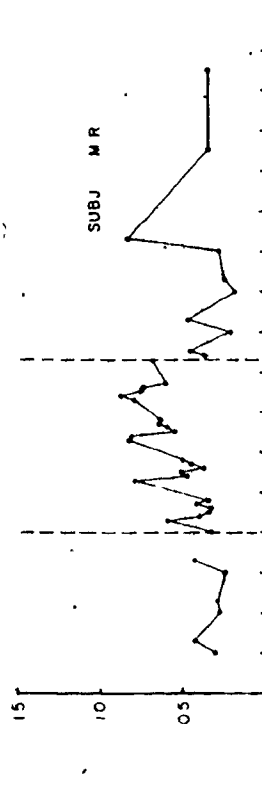
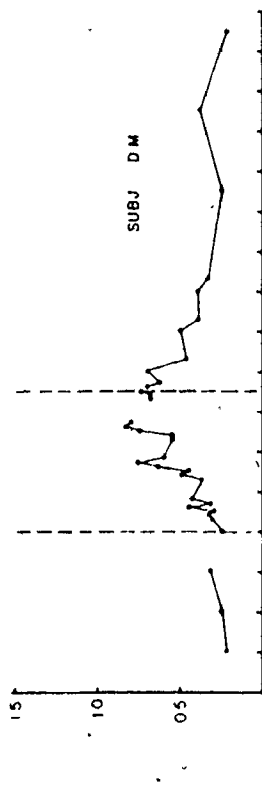
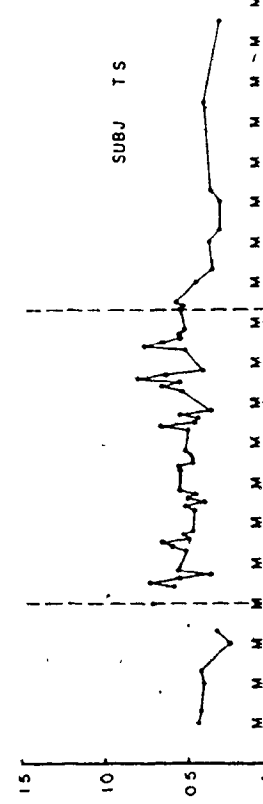
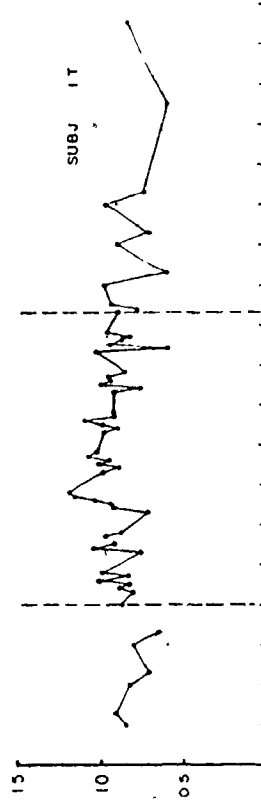
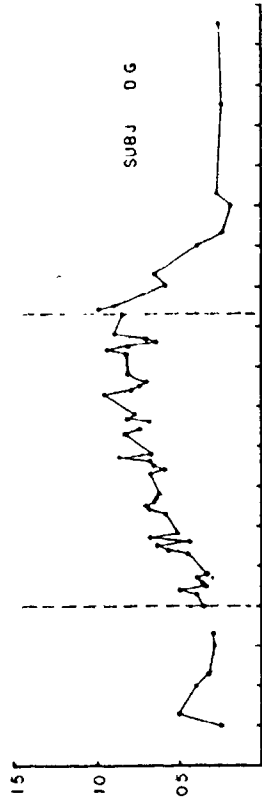
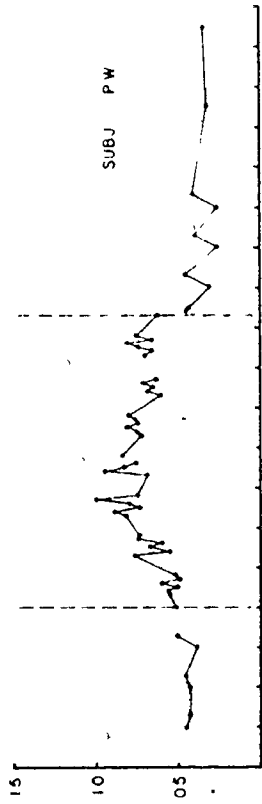


Figure 19

The variance of the distribution of power in the range ± 2 Hz from the occipital alpha peak in the power spectrum. Each point represents the average from two samples taken in the eyes open condition.

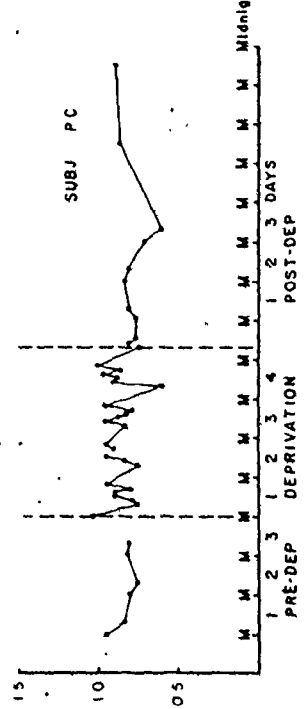
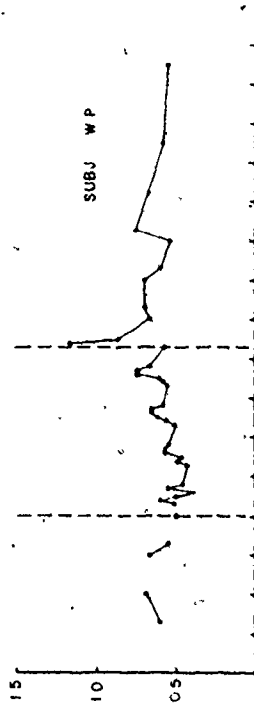
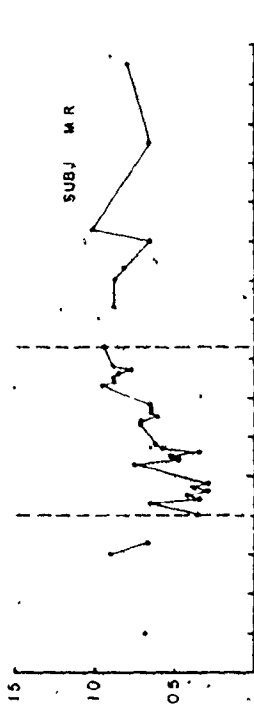
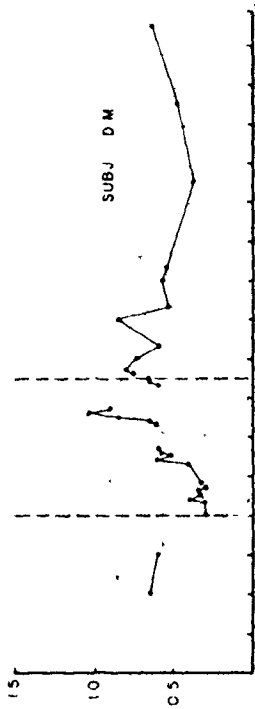
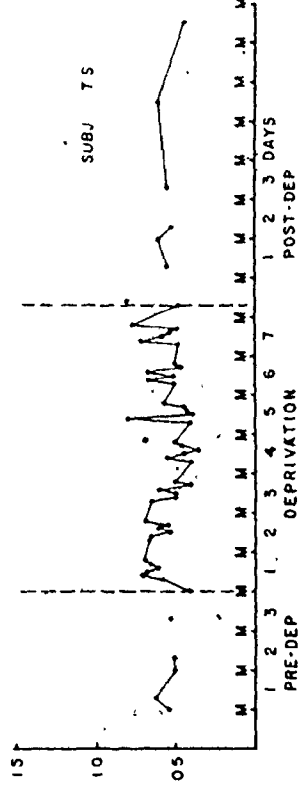
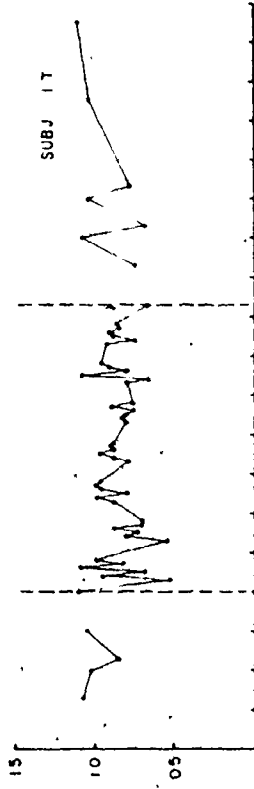
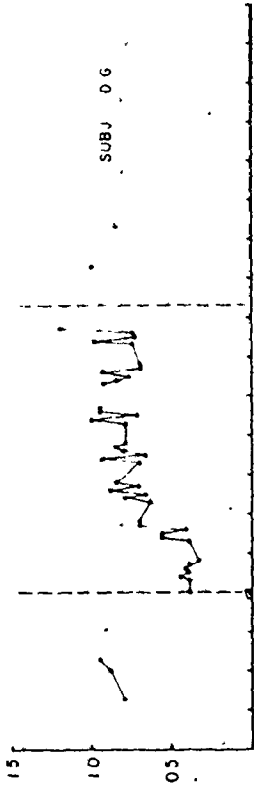
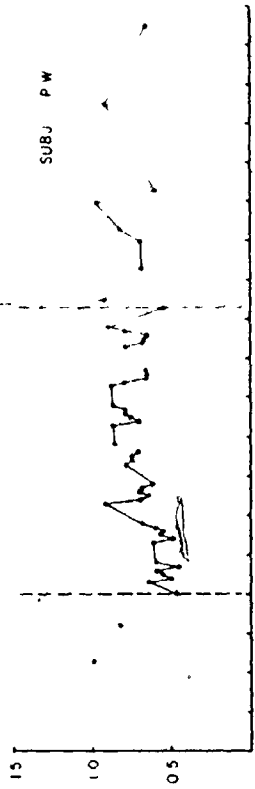


Figure 20

Power spectra produced from 1 minute of the sample of EEG shown in Figure 3. The vertical scale has been magnified by a factor of two as compared to the spectra from the occipital lead shown in Figure 5.

SUBJ. P.W. PRE-DEP I MORNING

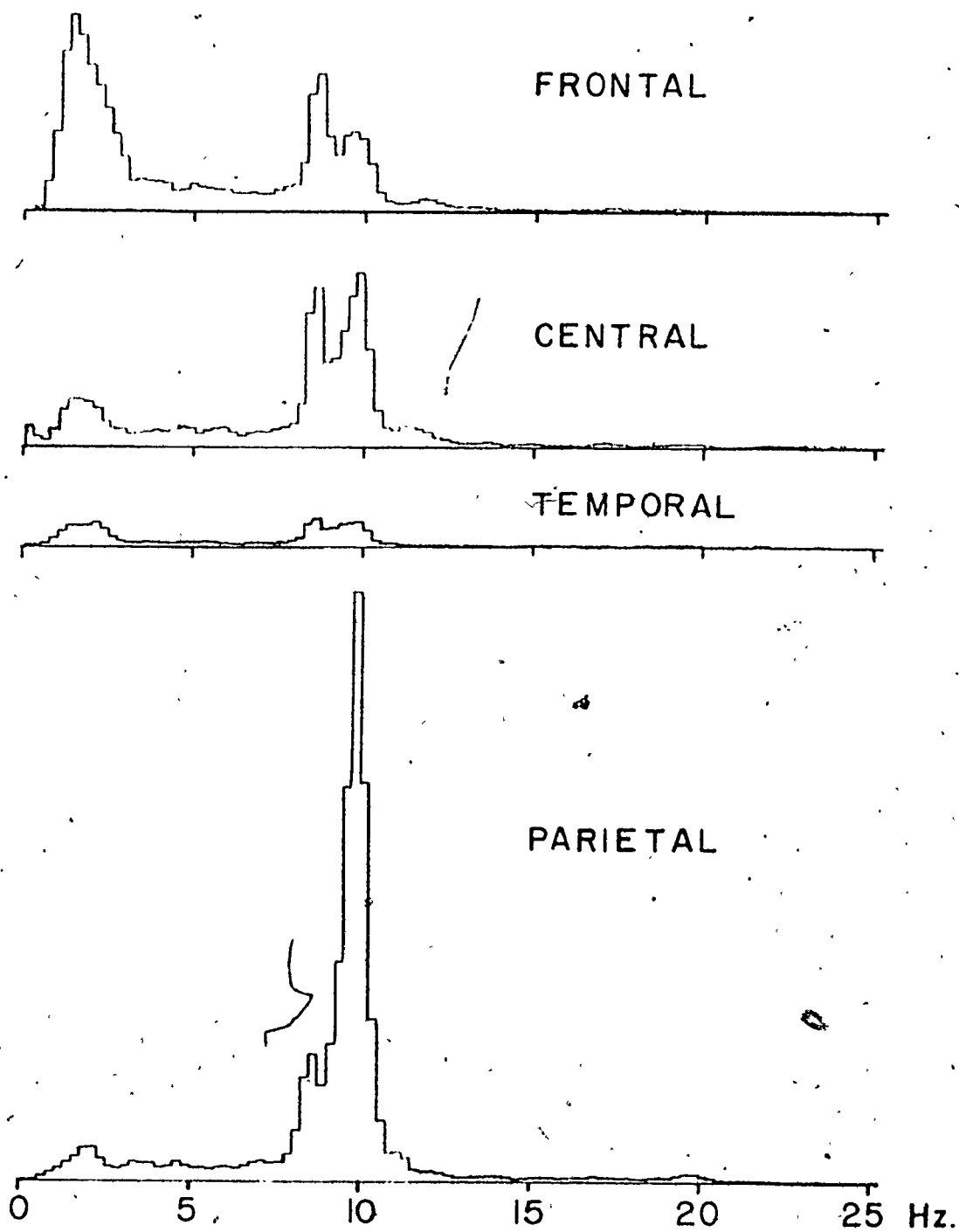


Figure 21

The modal frequency of the occipital and frontal alpha from subjects W. P. and P. W.

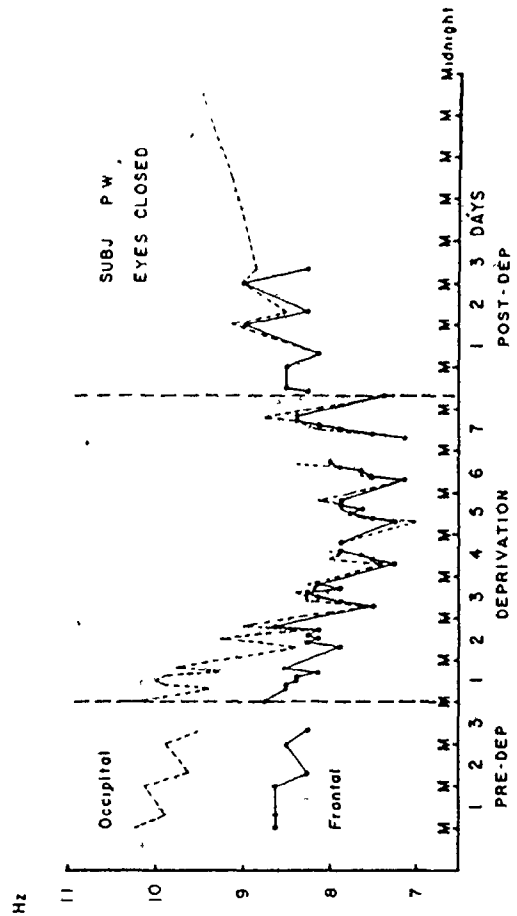
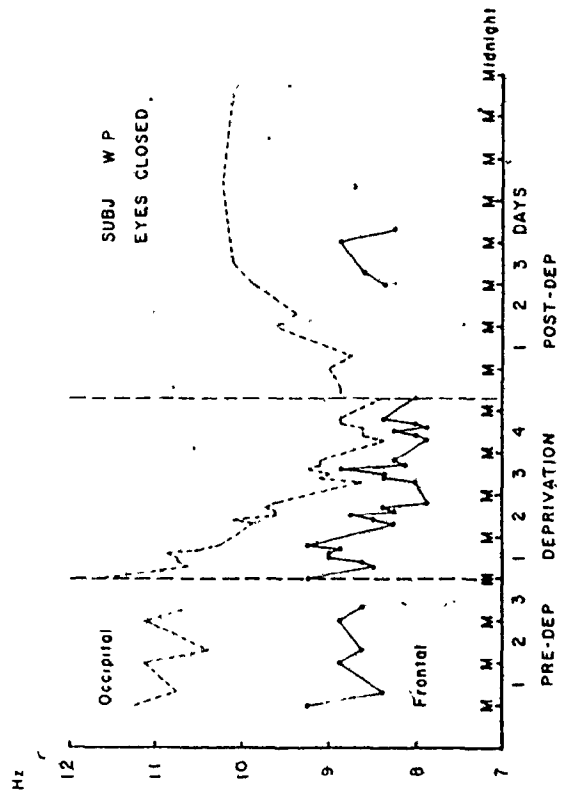
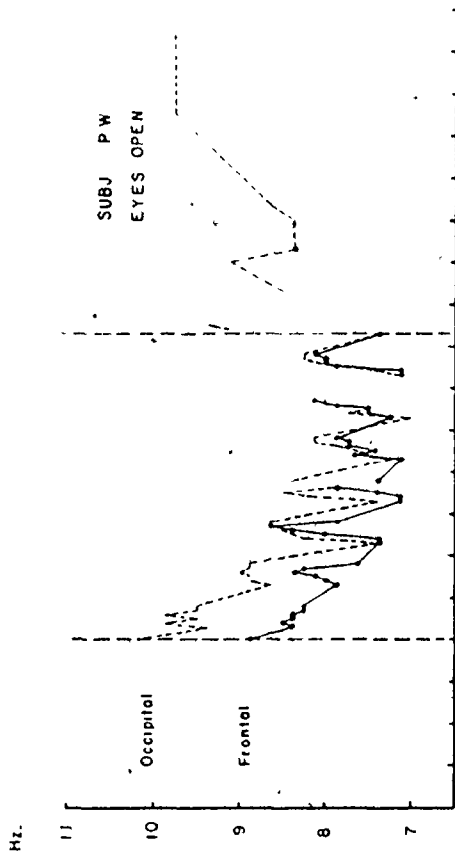
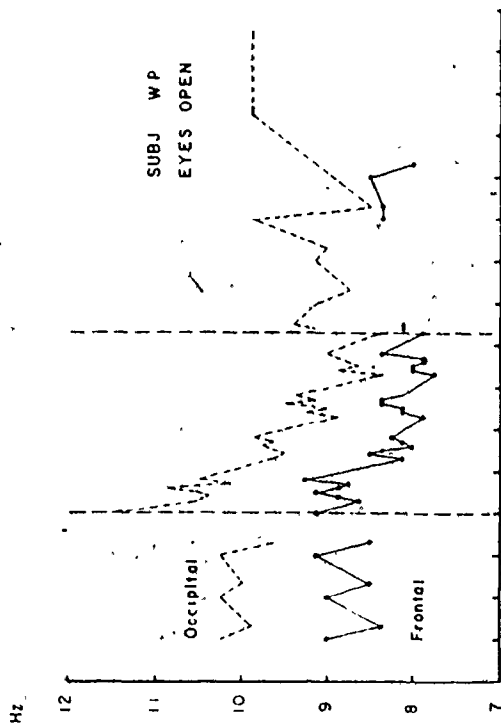
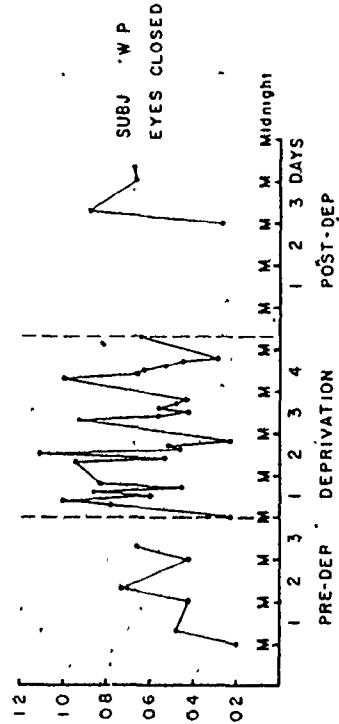
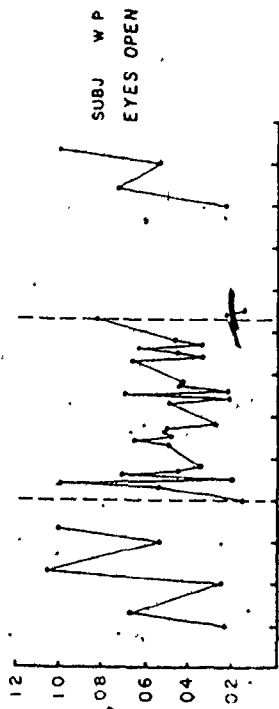
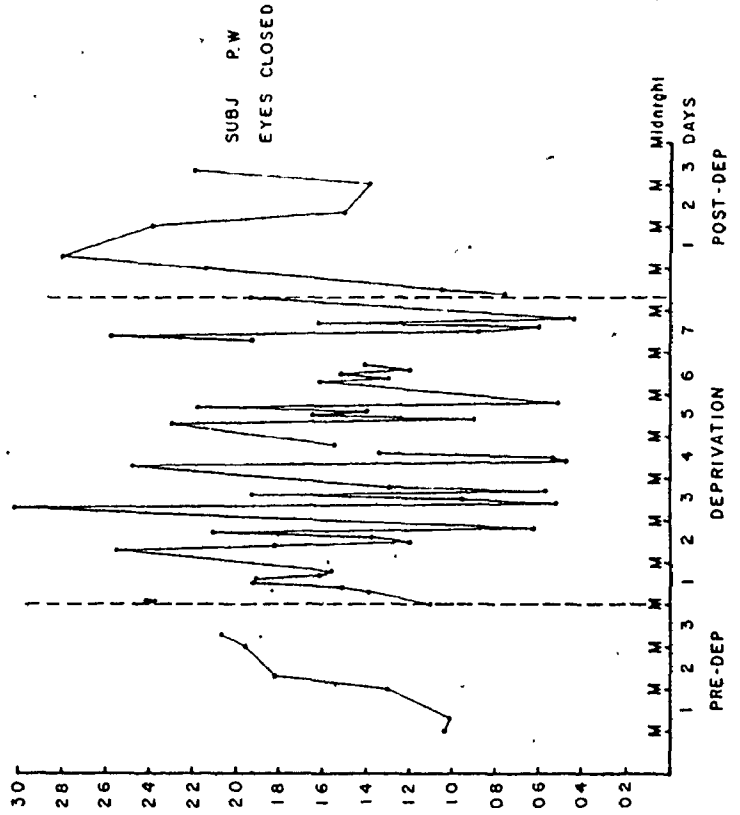
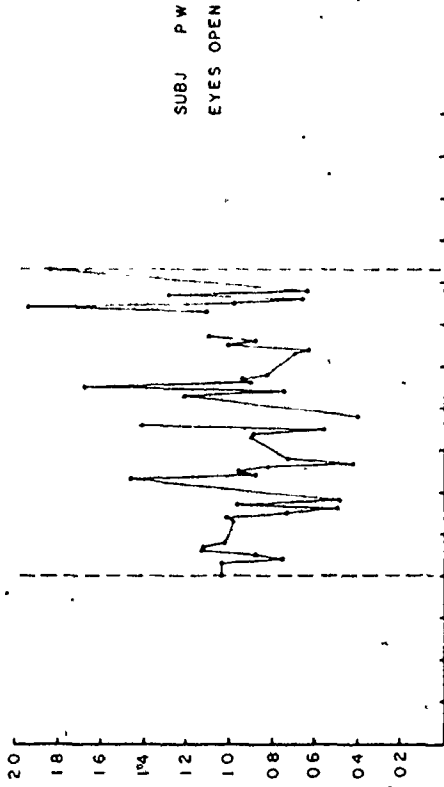


Figure 22

The power of the mode of the alpha peak in spectra of the frontal EEG in subjects W. P. and P. W.



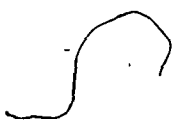
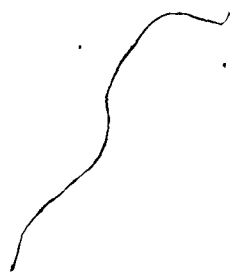
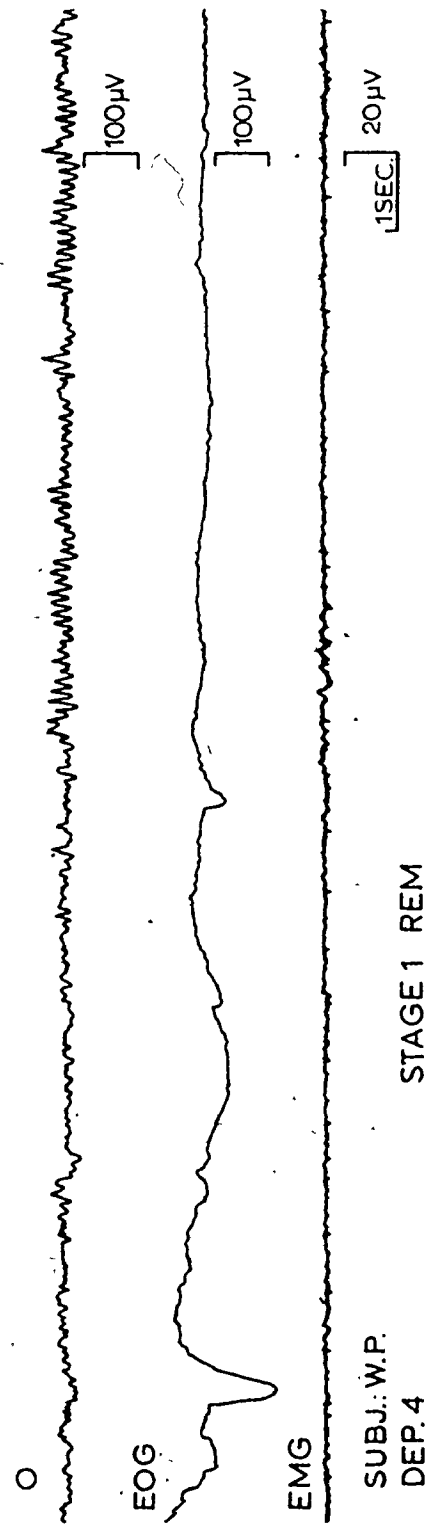
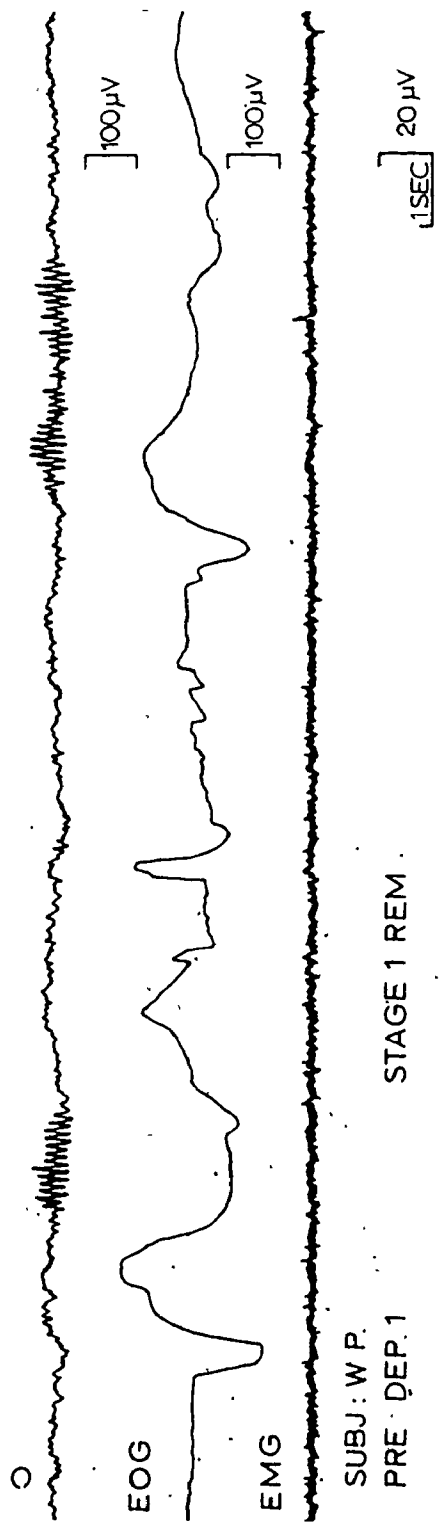


Figure 23

Examples of the occipital EEG, eye movements and EMG during a REM period of subject W. P. on the first pre-deprivation night and on the fourth deprivation night.








Figure 24

Power spectra of 1 minute of the EEG samples shown in
Figure 23.

SUBJ. W.P. REM ALPHA

□ PRE-DEP I
TIME: 0600

■ DEP 4
TIME: 0530

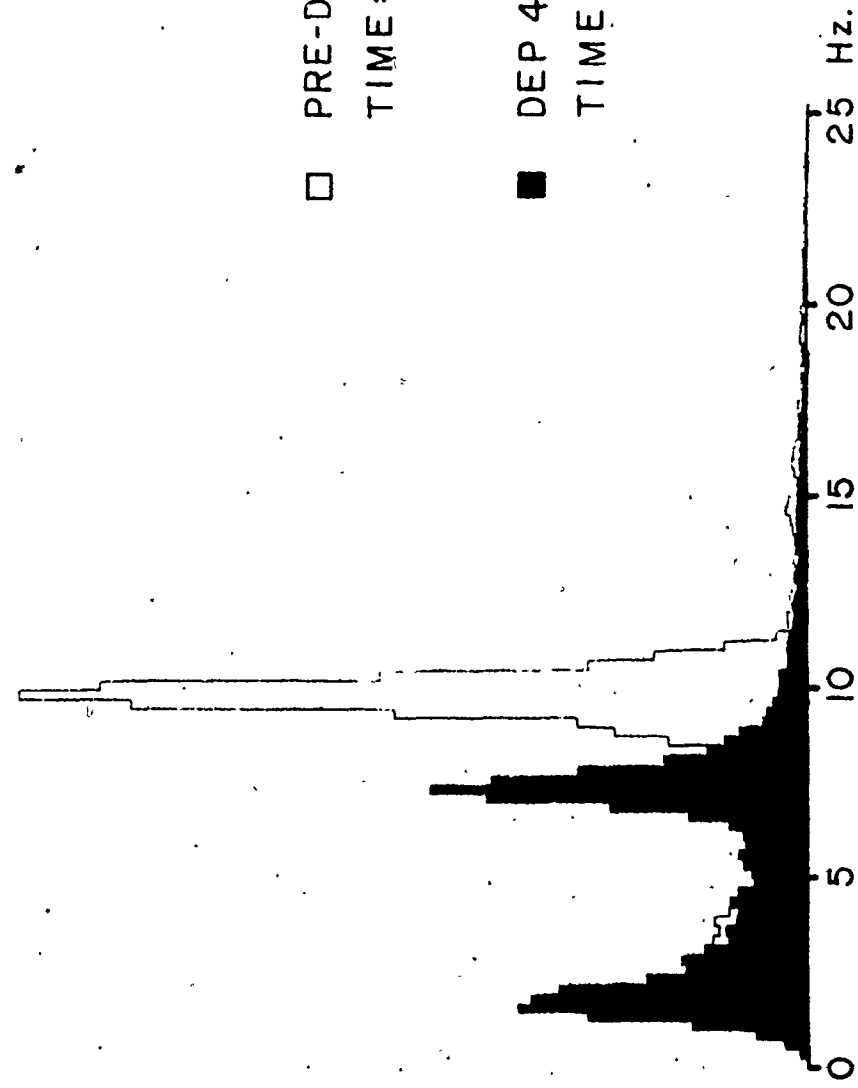


Figure 25

The modal frequency of the occipital alpha during waking and during REM periods for experimental subject W. P. and control subject W. K.

Figure 26 a and b

Examples of power spectra of the frontal, central, temporal, parietal, and occipital EEG of subject W. P. containing peaks in the theta range. The two alpha rhythms (frontal and occipital) can also be clearly distinguished in these examples.

Figure 26a

SUBJ. W.P. PRE-DEP. 2, MORNING
EYES CLOSED

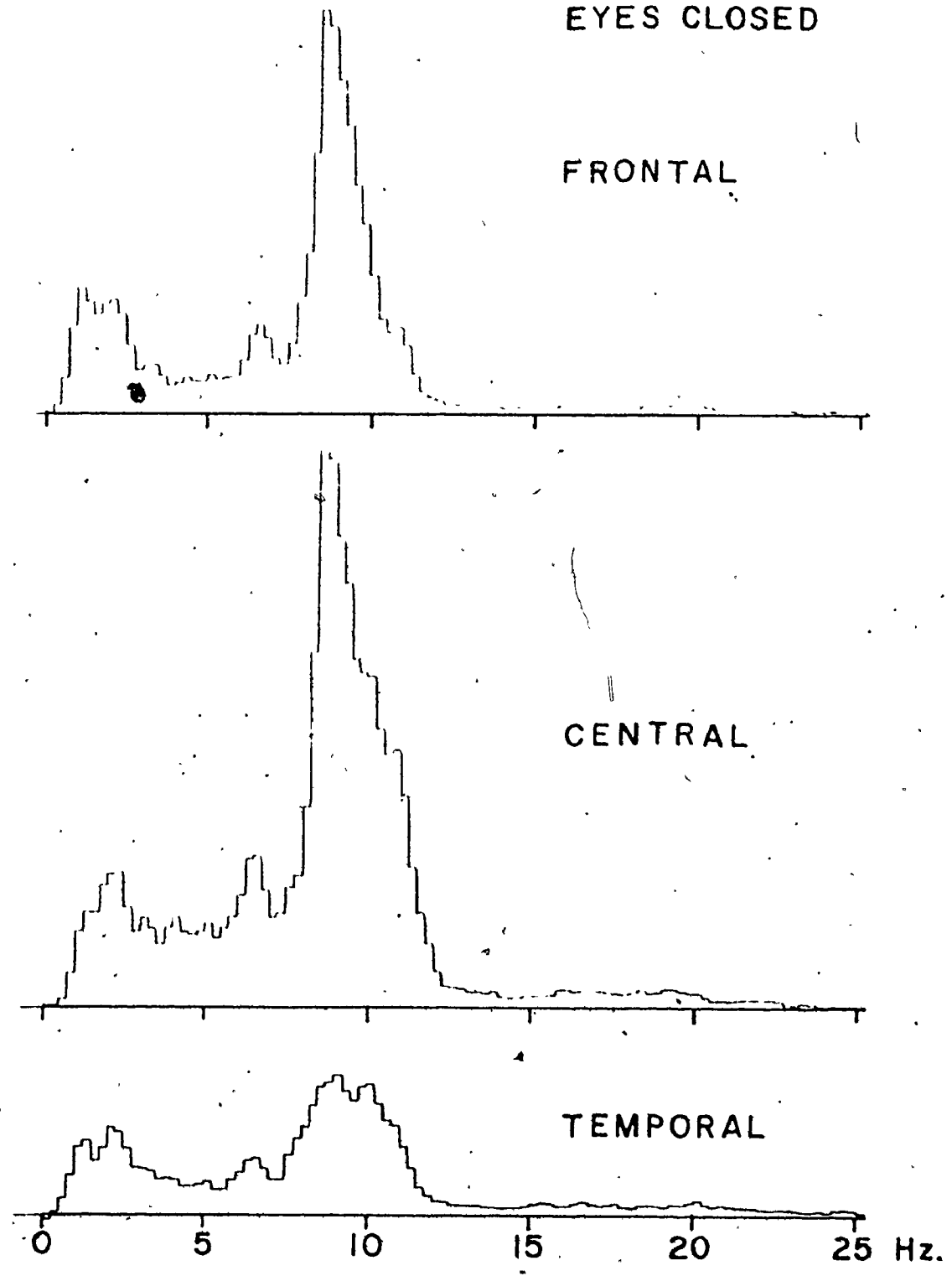


Figure 26b

SUBJ. W.P. PRE-DEP. 2, MORNING
EYES CLOSED

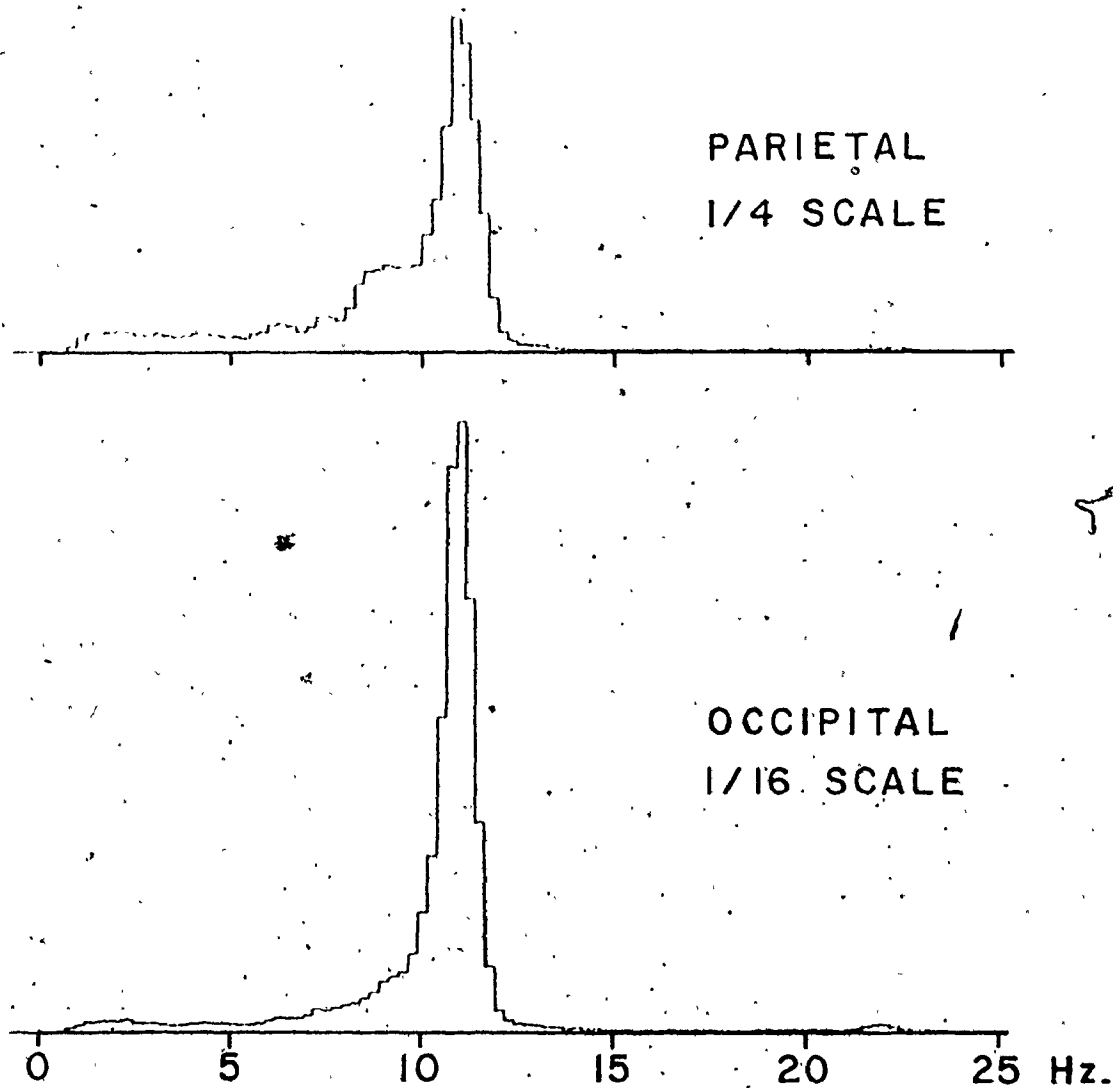


Figure 27

The modal frequency of the theta peak in the frontal EEG of the four-day and seven-day experimental subjects.

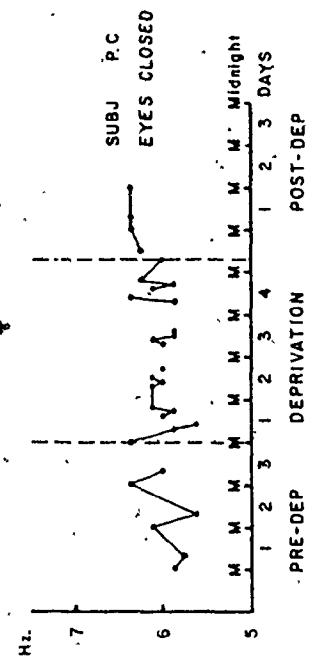
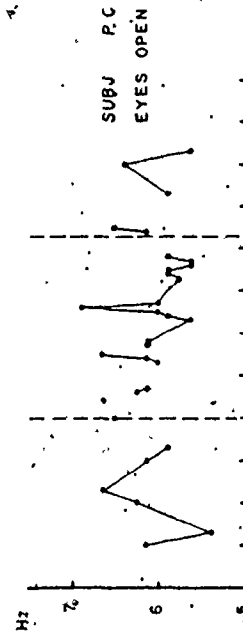
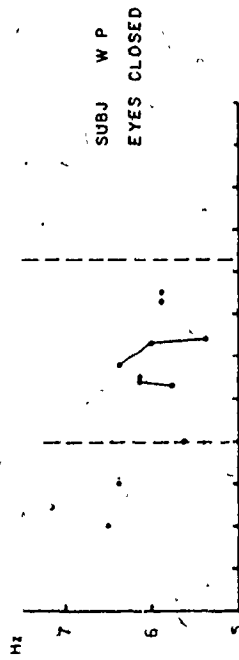
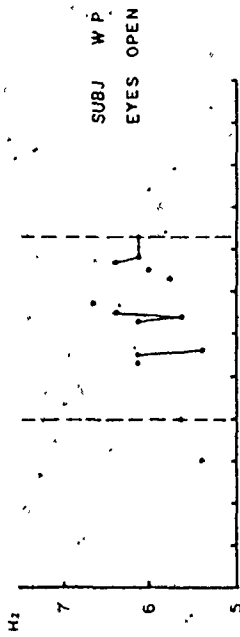
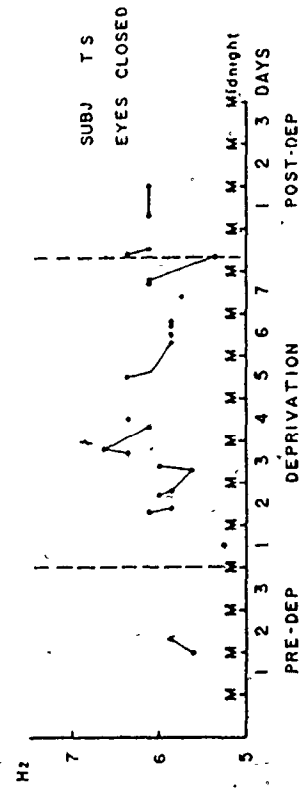
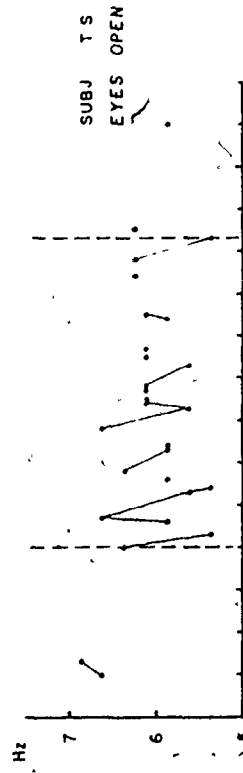
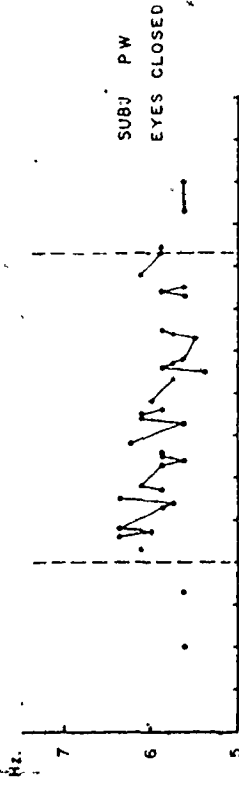
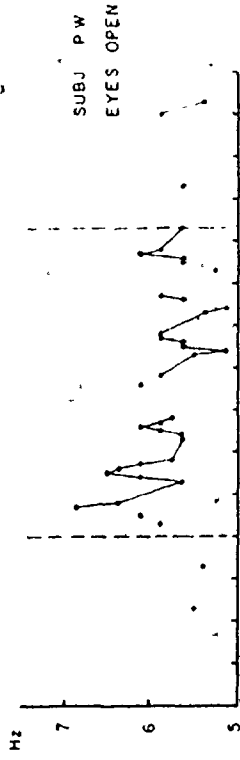


Figure 28

The power of the mode of the theta peak in the frontal EEG of the four-day and seven-day experimental subjects.

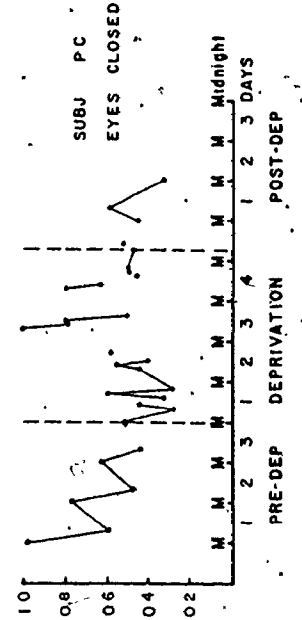
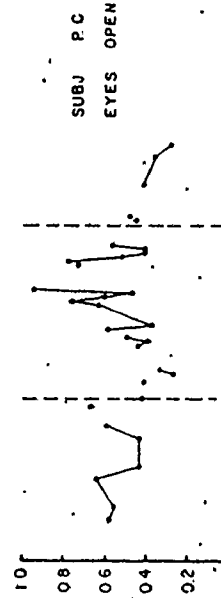
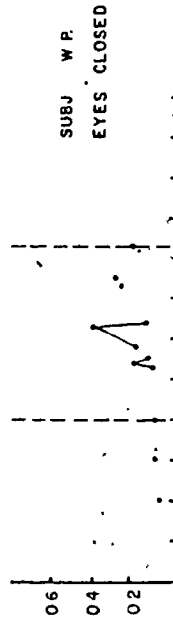
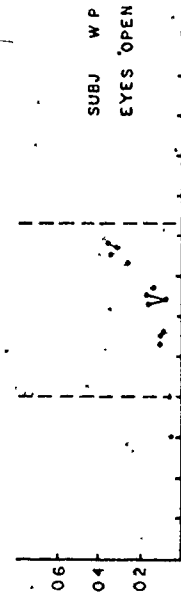
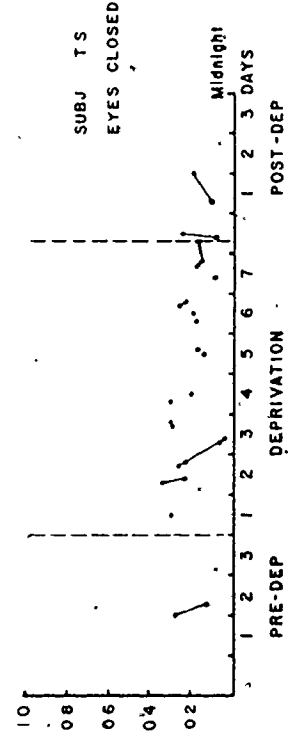
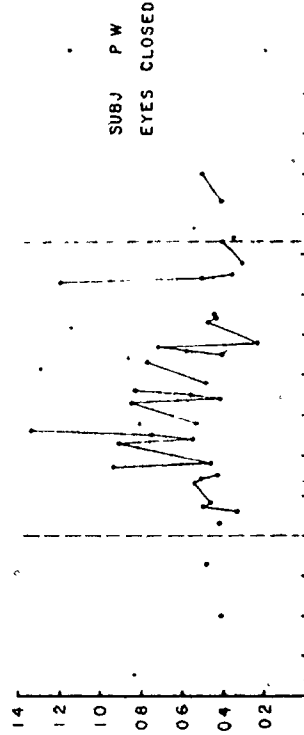
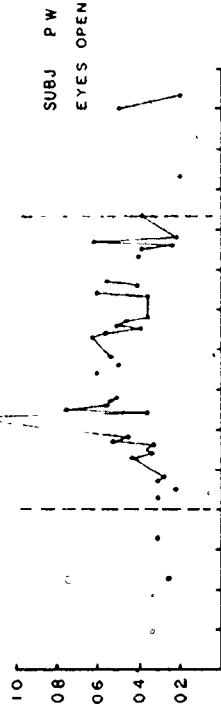
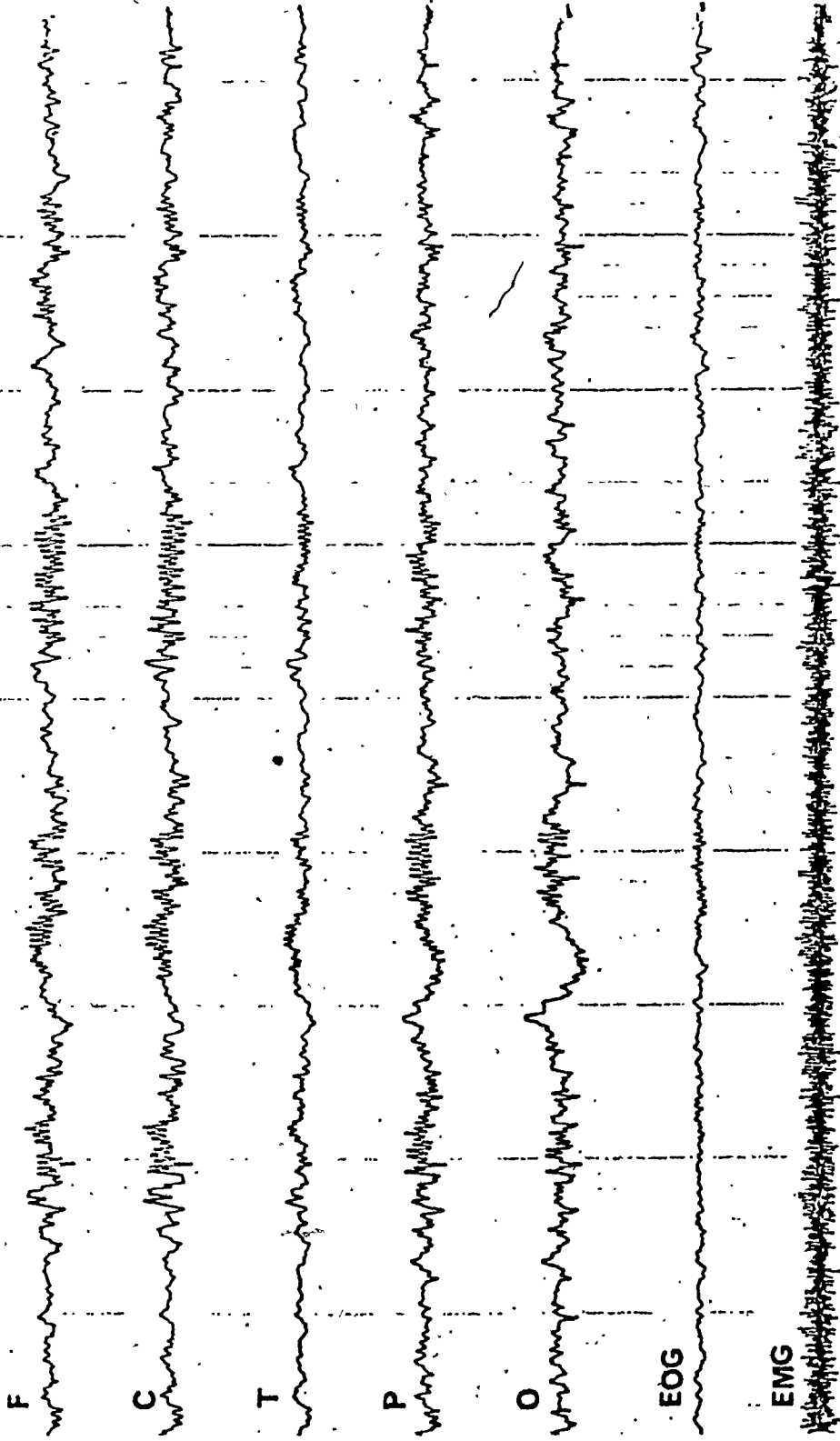


Figure 29

A sample of EEG from experimental subject D. G. recorded during Stage 2 sleep on the third pre-deprivation night.

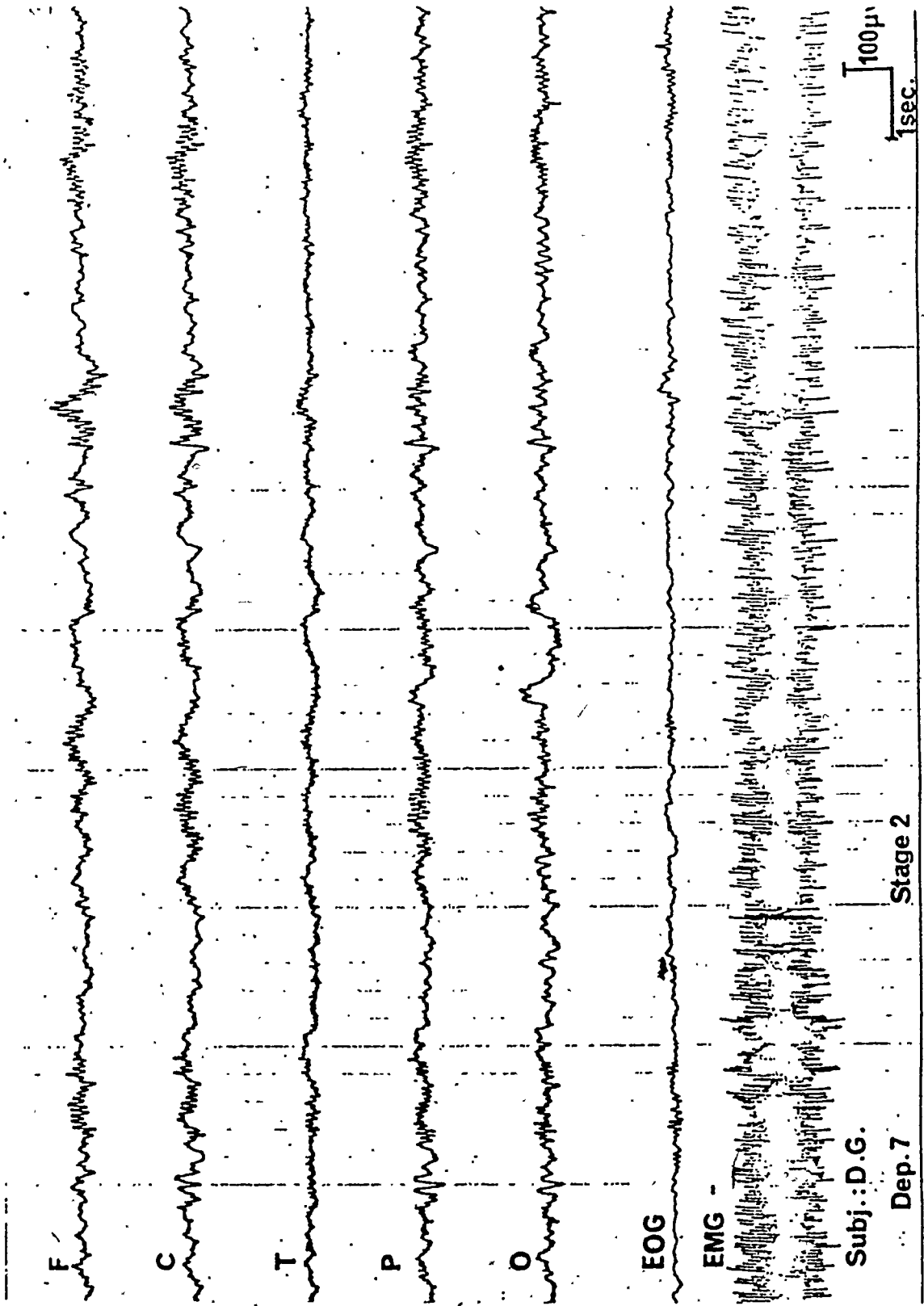


Subj.: D.G.
Pre Dep. 3

Stage 2

Figure 30

A sample of EEG from experimental subject D. G. recorded during Stage 2 sleep on the seventh night of deprivation.



Subj.: D.G.
Dep. 7

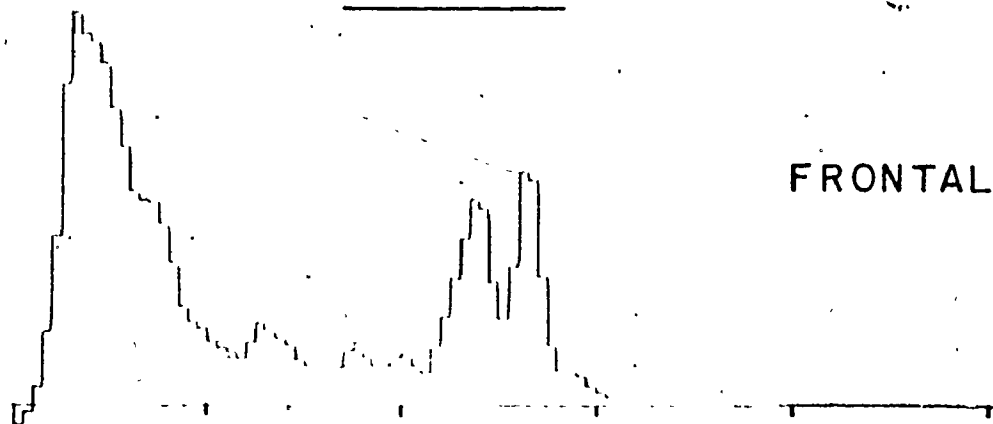
Stage 2

Figure 31

Power spectra computed from 1 minute of the frontal and parietal Stage 2 EEG shown in Figures 29 and 30.

SUBJ. D.G. STAGE 2 SLEEP

PRE-DEP 3



DEP 7

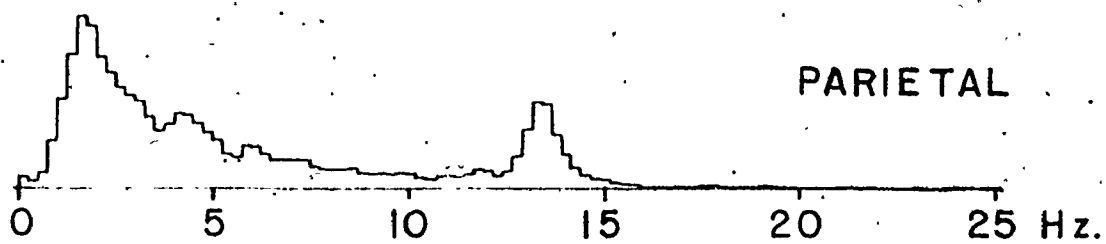
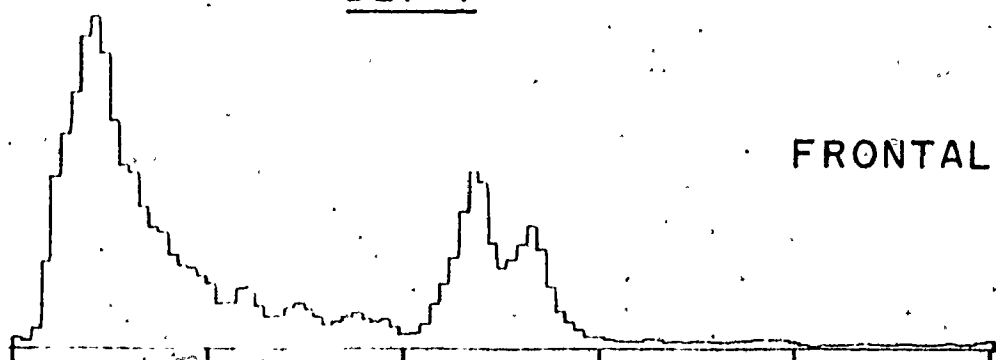


Figure 32

The modal frequency of the low frequency frontal and high frequency parietal spindles occurring during Stage 2 sleep in the experimental subjects.

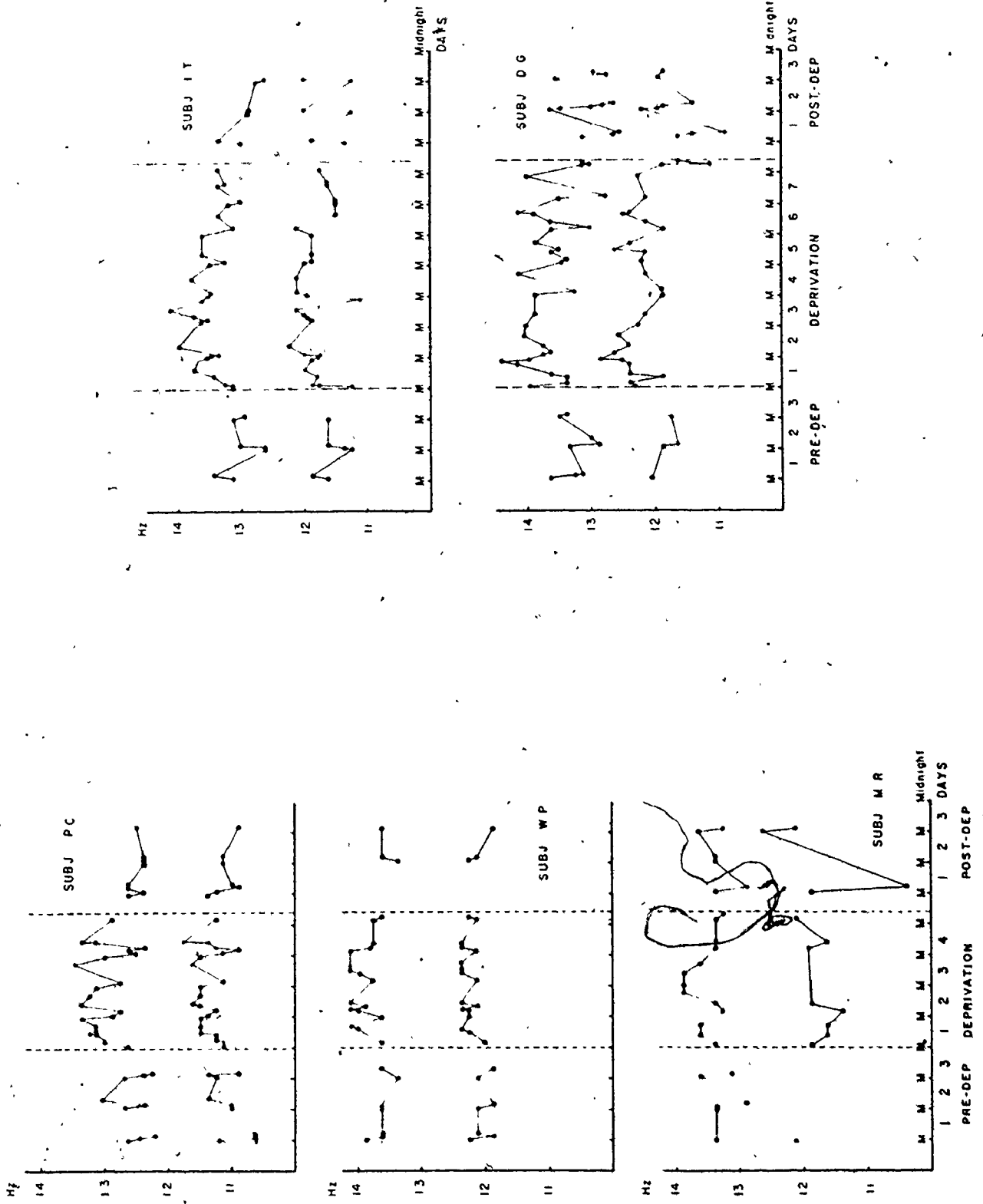
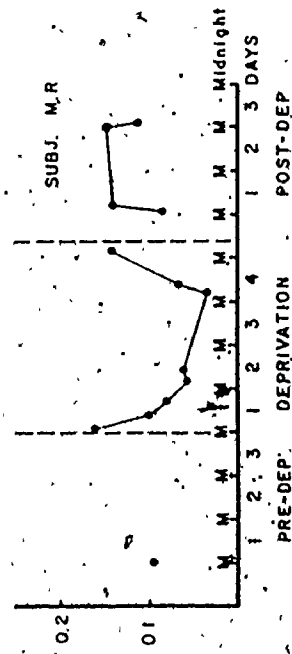
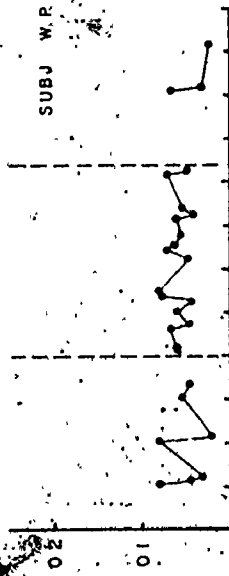
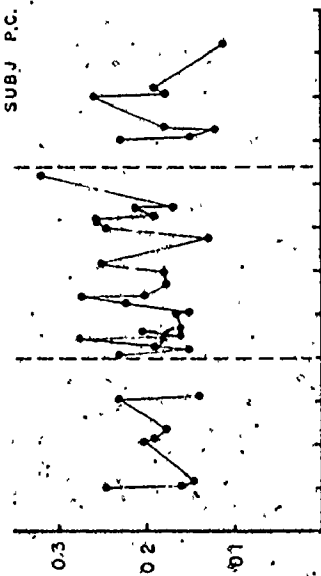
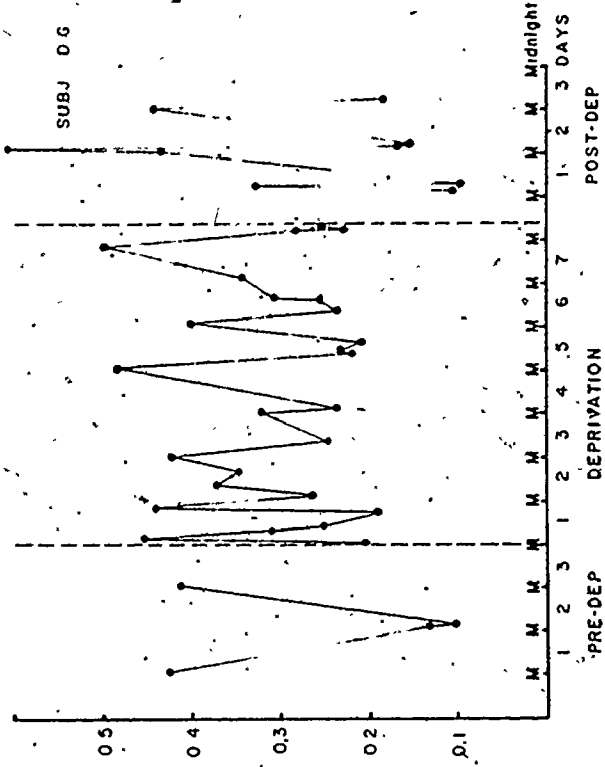
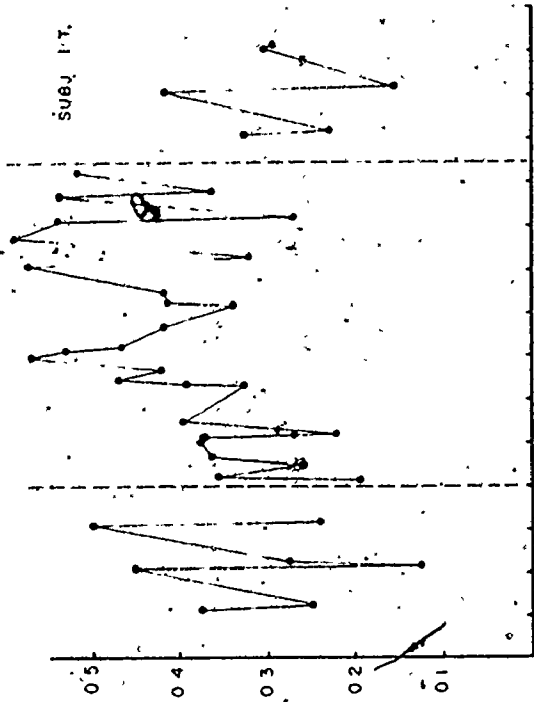


Figure 33

The power of the mode of the low frequency frontal spindle occurring during Stage 2 sleep in the experimental subjects.



sp

Figure 34

The power of the mode of the high frequency parietal spindle occurring during Stage 2 in the experimental subjects.

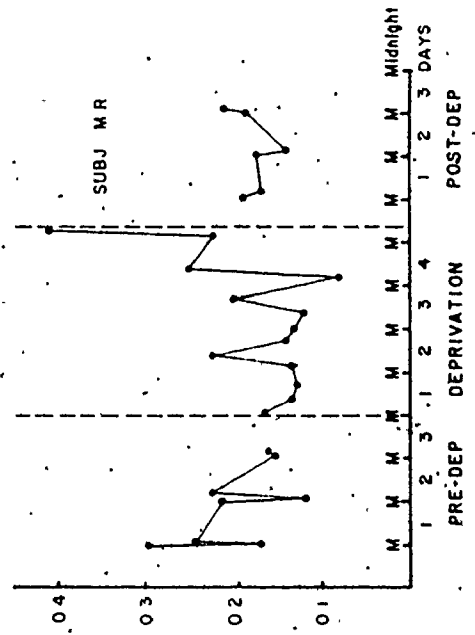
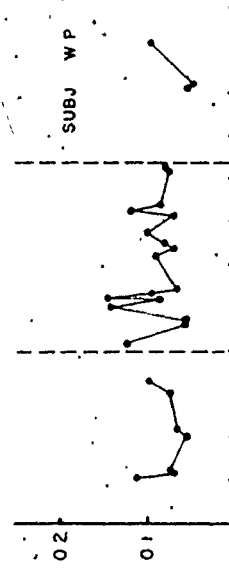
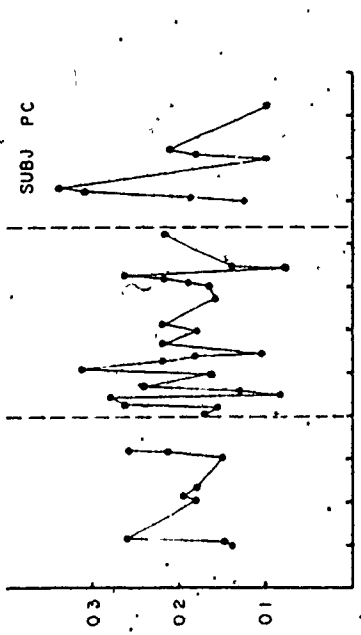
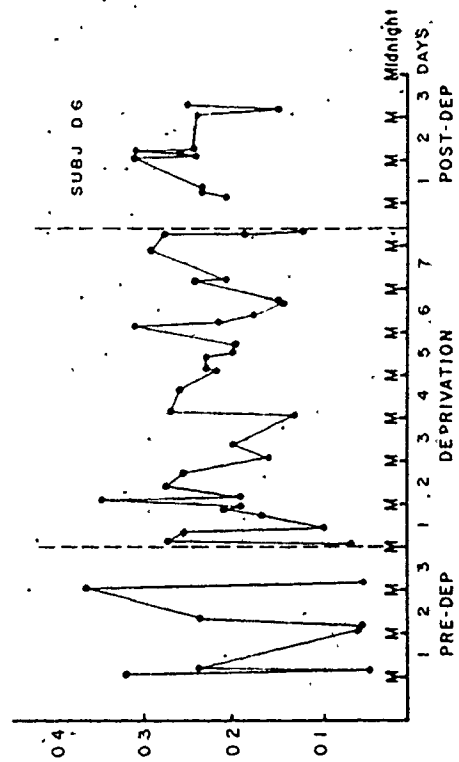
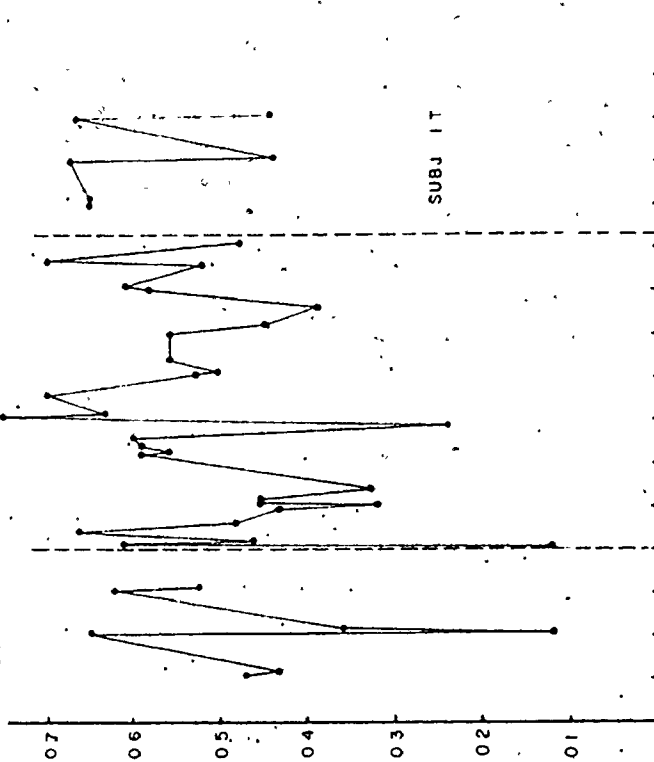


Figure 35

The modal frequency of the occipital alpha of the four-day and seven-day control subjects. Each point represents the average of the modes from two spectra.



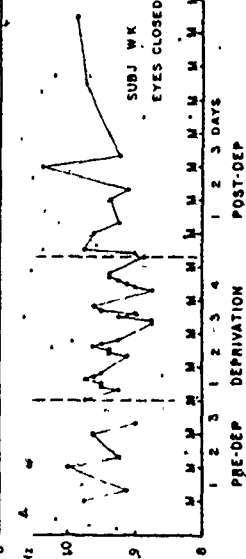
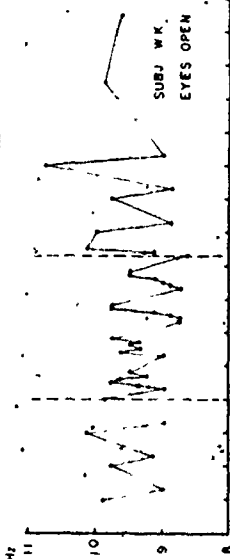
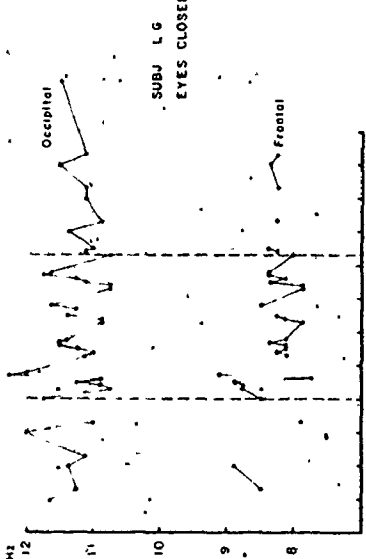
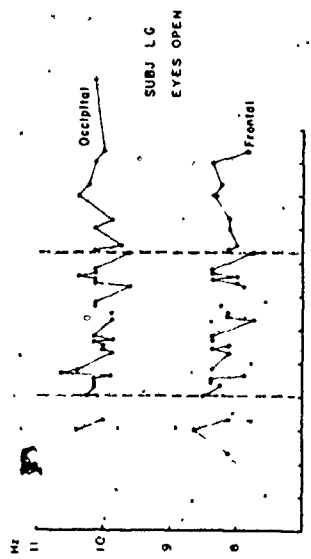
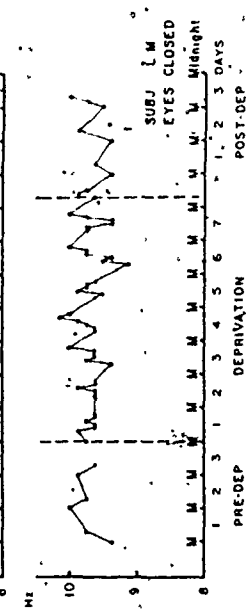
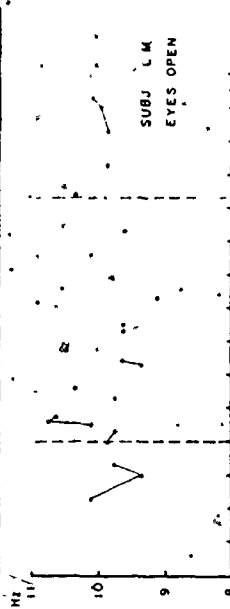
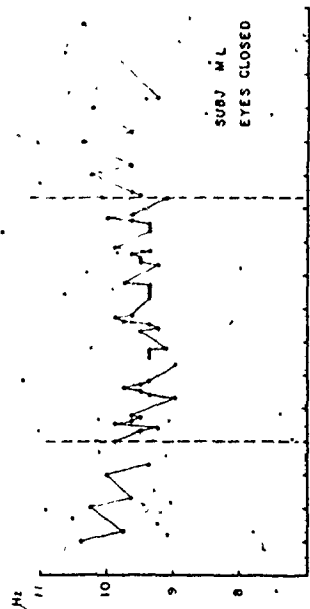
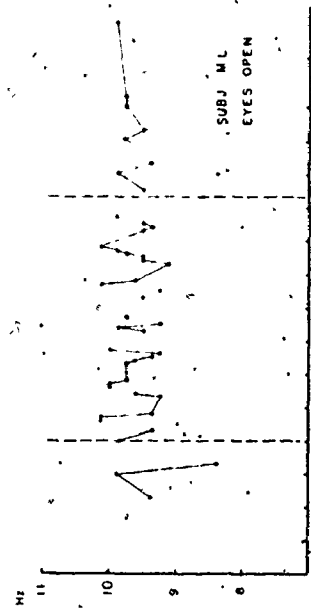


Figure 36

The power of the mode of the occipital alpha of the four-day and seven-day control subjects. Note: The EEG of these subjects was recorded at twice the gain of that used for the experimental subjects. The value of power is thus multiplied by a factor of four.

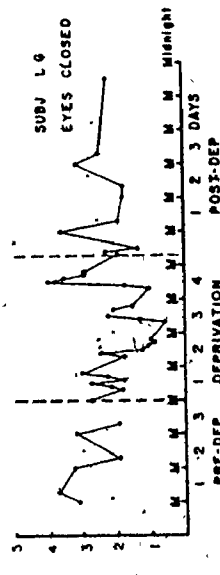
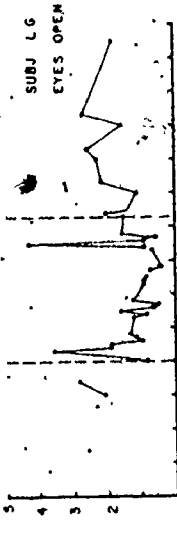
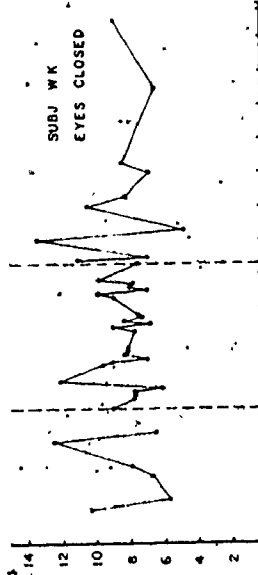
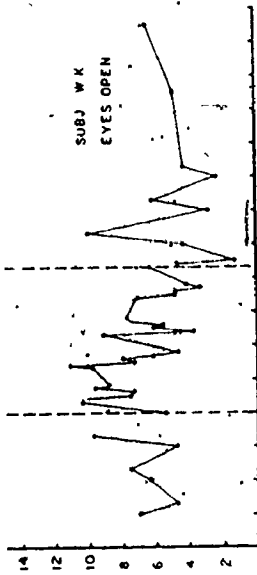
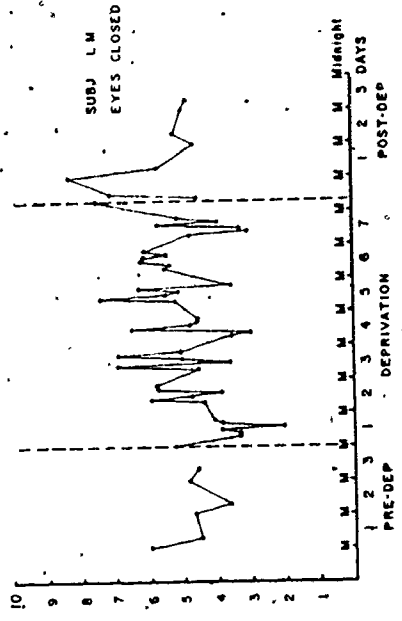
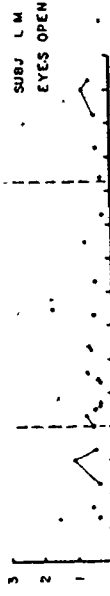
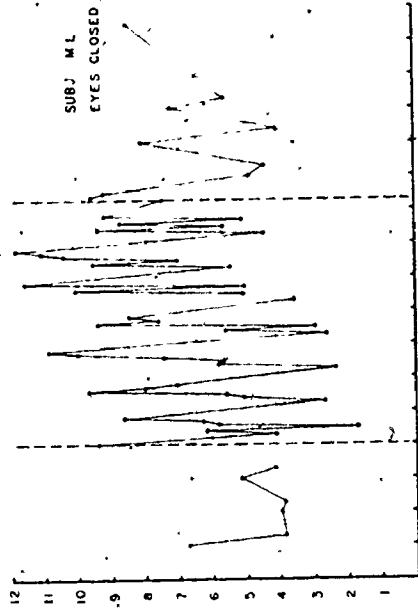


Figure 37

The weighted average frequency of the occipital alpha of the four-day and seven-day control subjects calculated over the range ± 2 Hz. from the mode of the alpha peak.

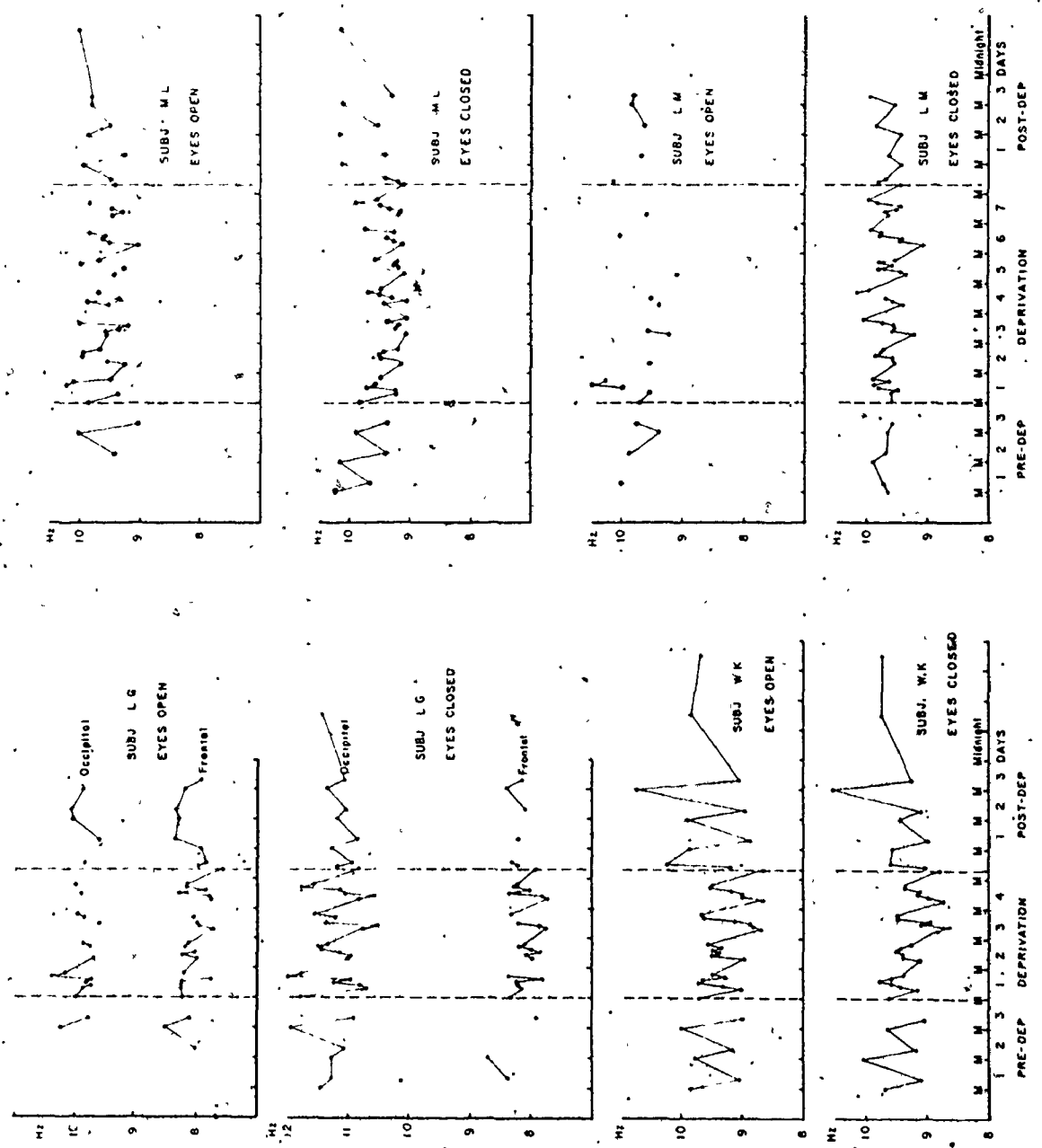


Figure 38

The total power in the occipital alpha peak of the four-day and seven-day control subjects calculated over the range ± 2 Hz. from the mode.

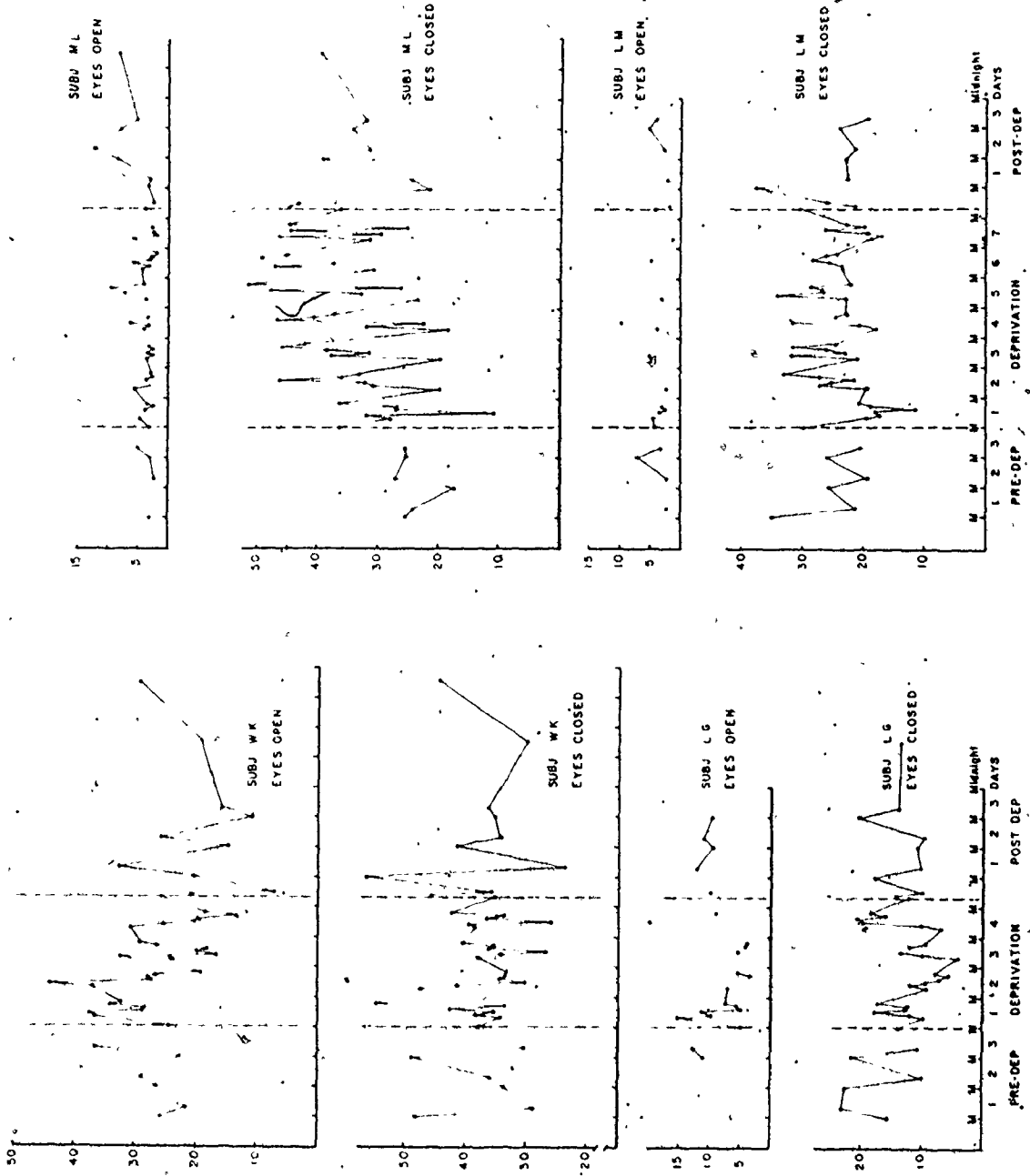


Figure 39

The variance of the peak in the power spectrum of the occipital alpha of the control subjects calculated over the range ± 2 Hz. from the mode.

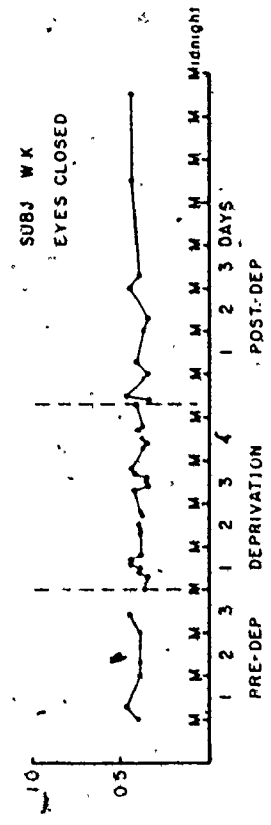
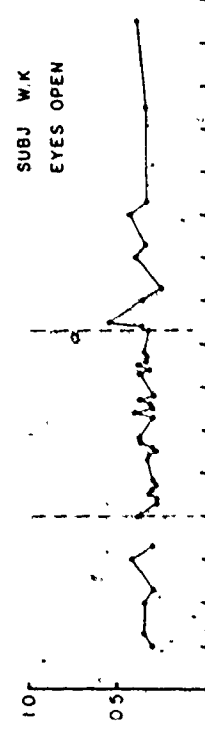
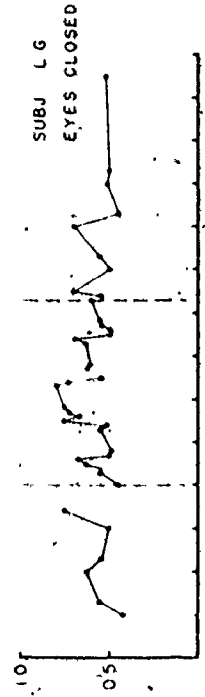
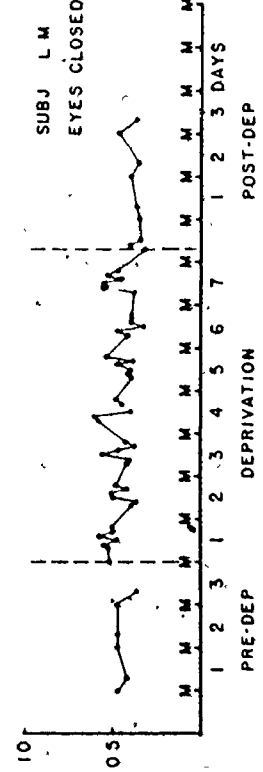
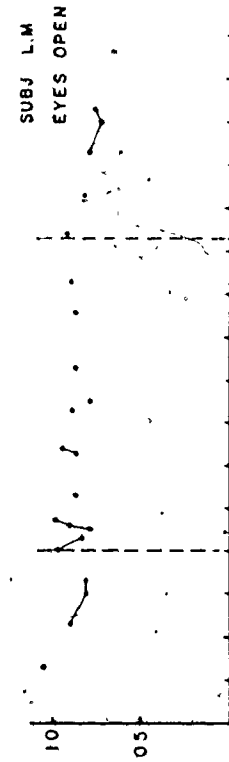
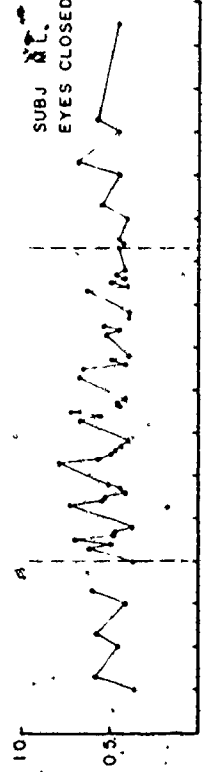
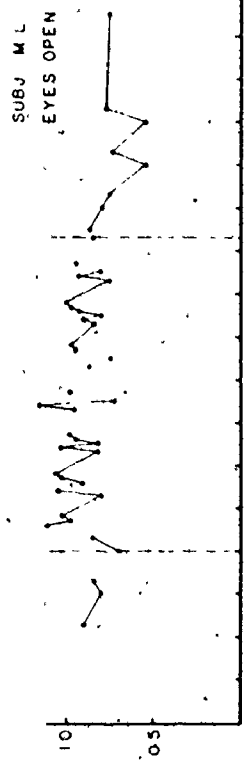


Figure 40

Oral body temperature of the control subjects measured immediately before the alpha recordings.

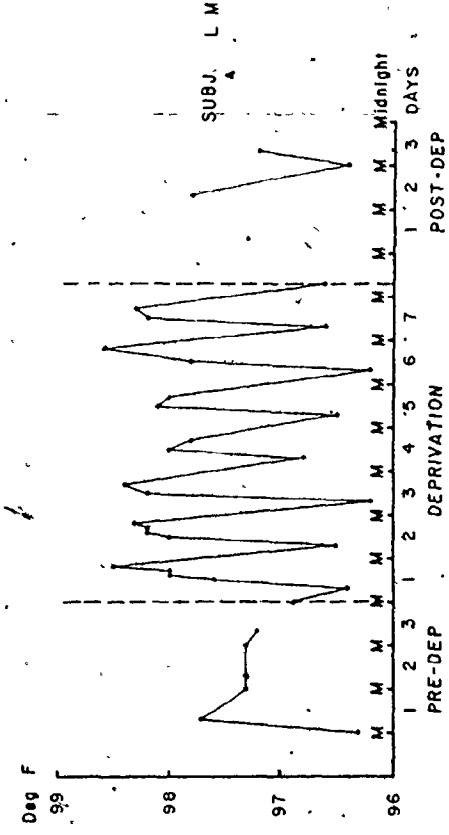
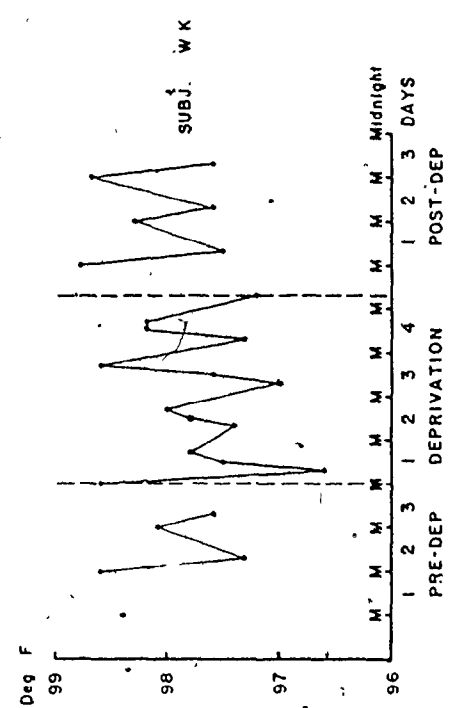
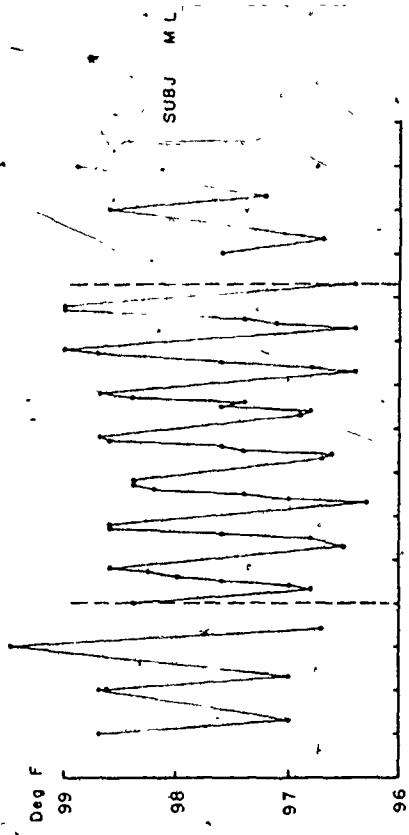
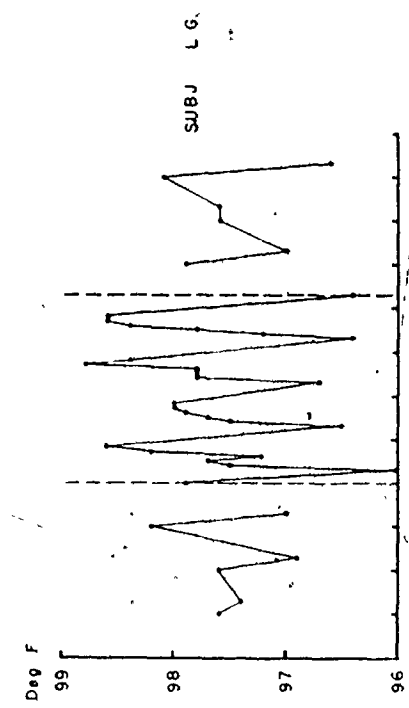
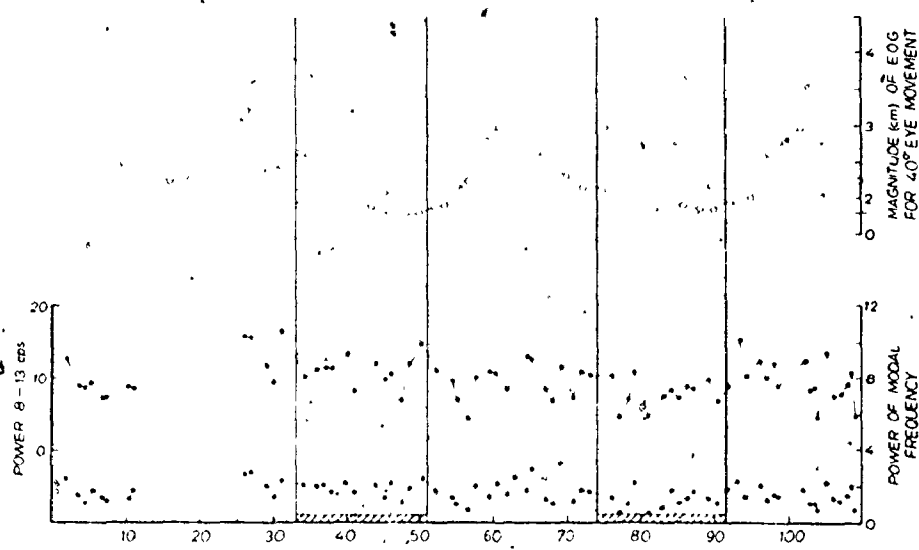
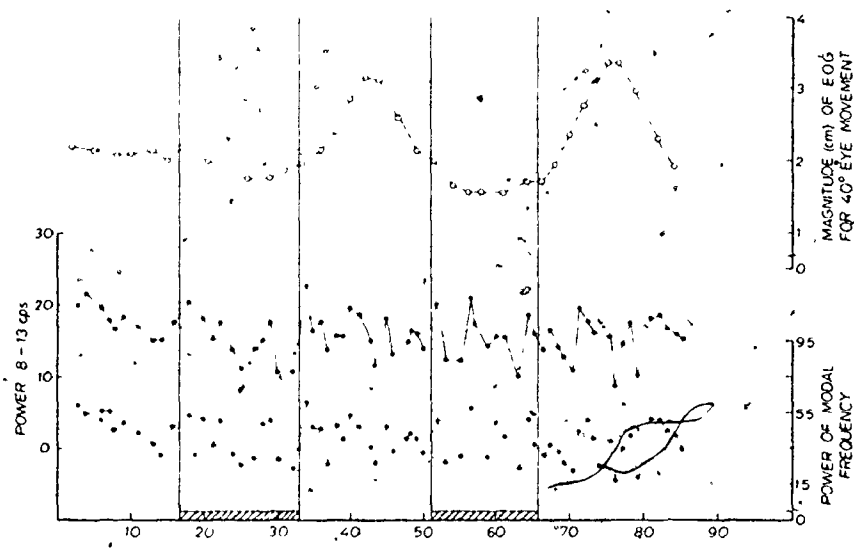
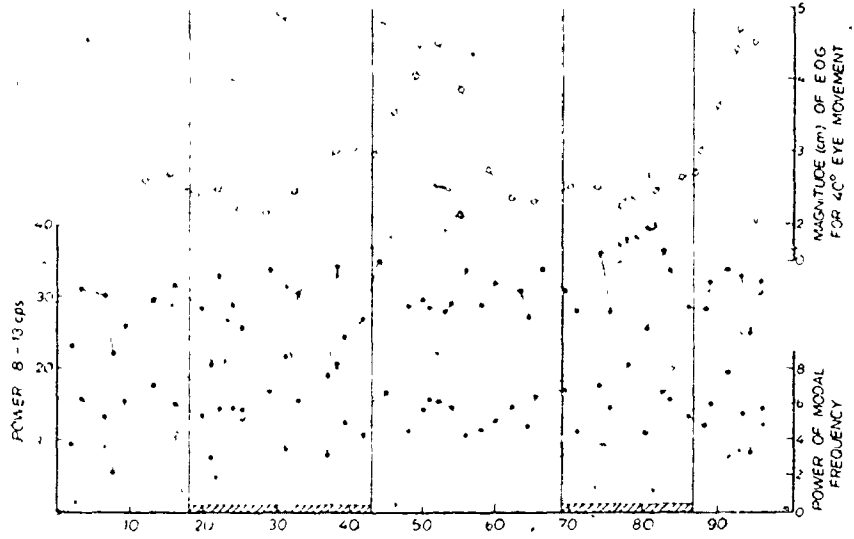


Figure 41

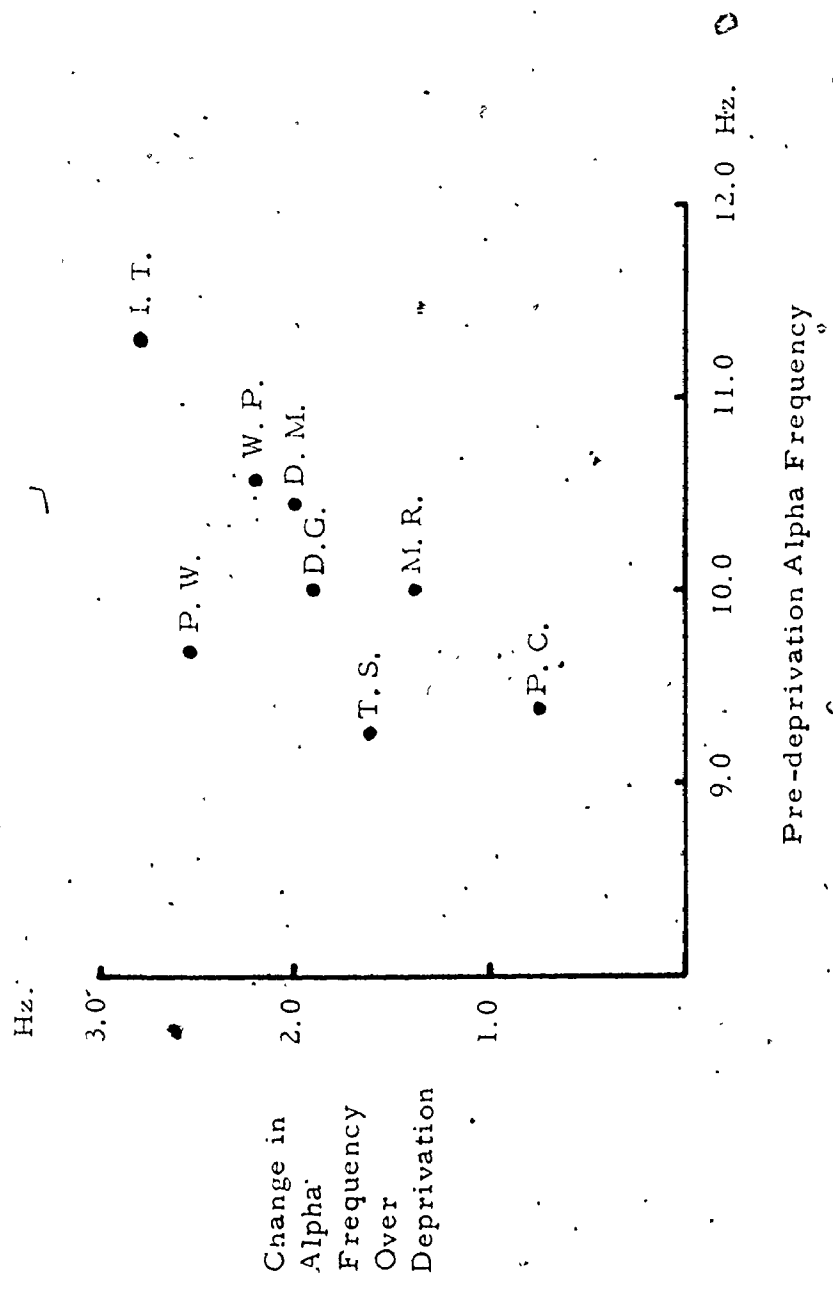
EOG amplitude (o—o), total power in the spectrum of the occipital EEG over the range 8-13 Hz. (●—●), and power of the mode of the alpha peak in the spectrum (●--●) are plotted as a function of time for three subjects. The cross-hatched areas on the abscissa of each graph represent periods during which the subjects' room lights were extinguished.



TIME IN MINUTES

Figure 42

A scatter plot showing the relationship between pre-deprivation modal alpha frequency and the magnitude of alpha shift (Hz.) over deprivation.



Change in
Alpha
Frequency
Over
Deprivation

Pre-deprivation Alpha Frequency

Figure 43

A power spectrum of the occipital EEG of subject W. K. recorded during REM sleep. The higher alpha peak at 7.75 Hz. represents the alpha frequency usually seen in this subject during REM sleep. The lower peak at 9.25 Hz. is the same as the frequency of the alpha during the waking state, but is rarely seen during REM sleep.

SUBJ. W.K. REM ALPHA

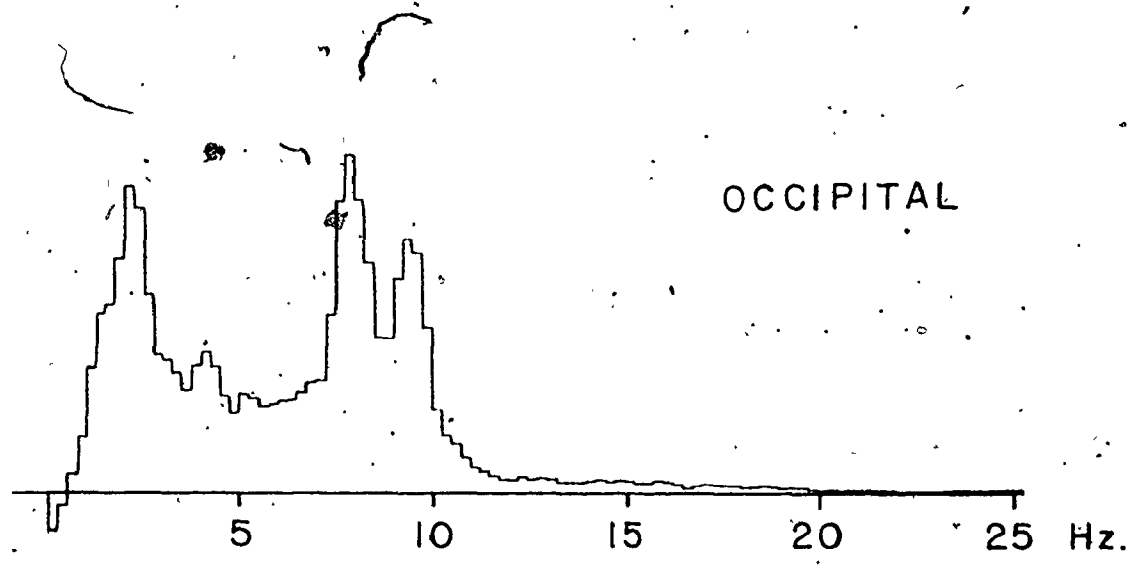
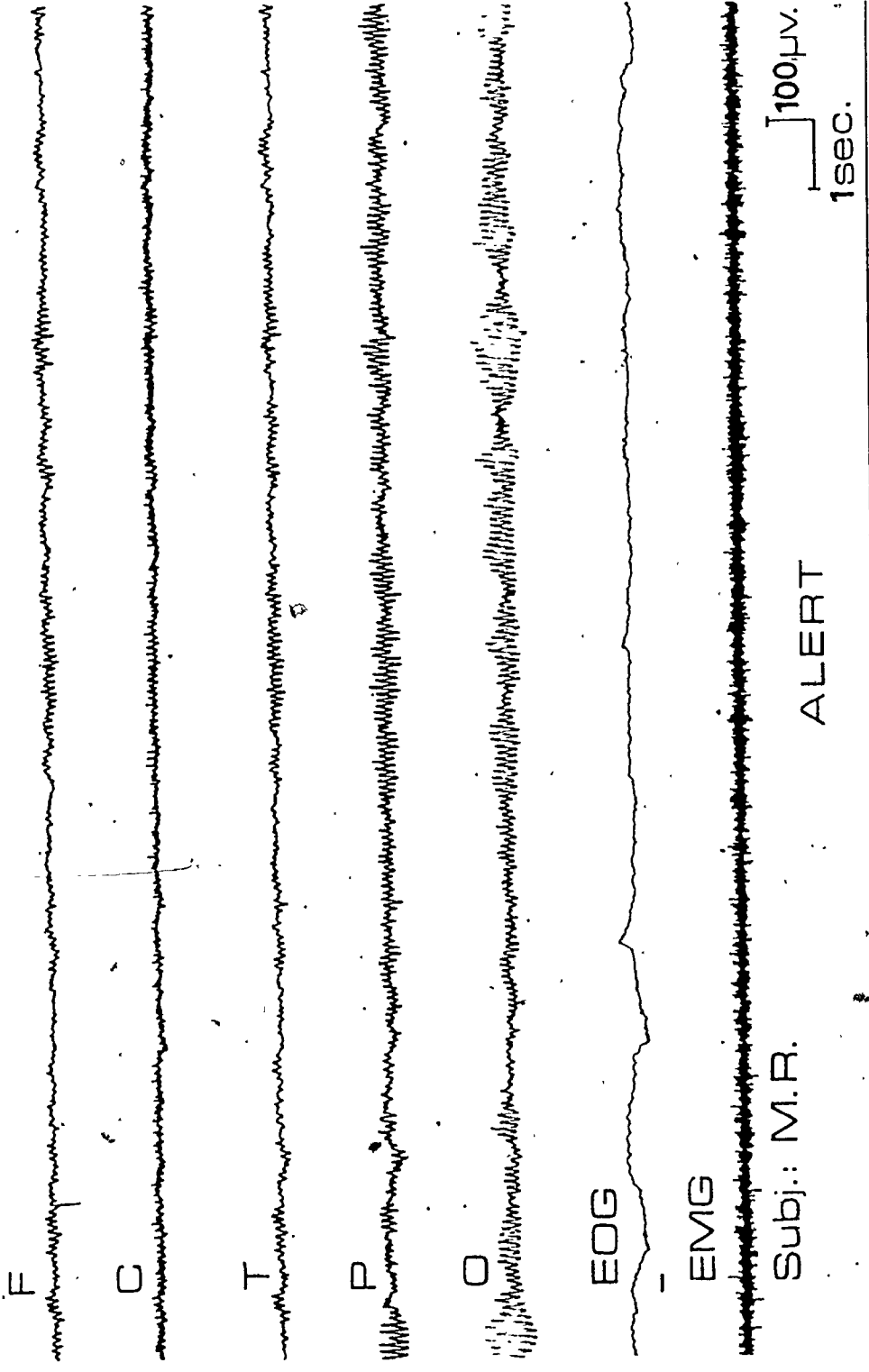


Figure 44

A sample of the EEG of subject M. R. recorded during the eyes closed condition on the evening of the second pre-deprivation night. Continuous alpha activity and rapid eye movements or eye fixation indicate that the subject is alert.

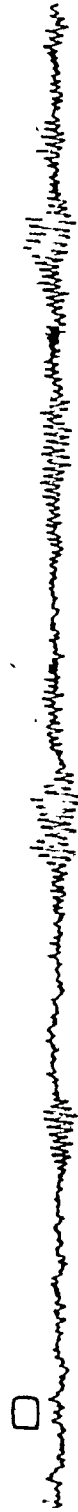
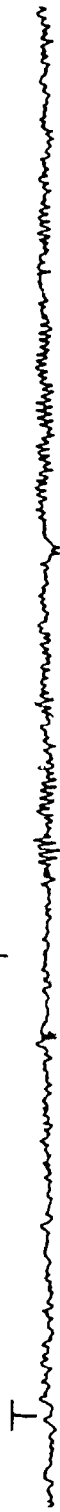
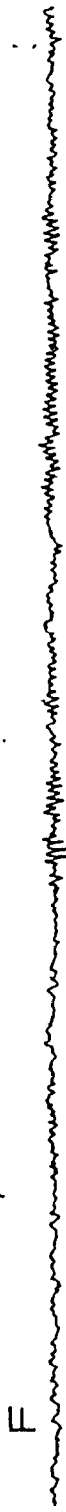


ALERT

Subj.: M.R.

Figure 45

A sample of the EEG of subject M. R. recorded seven minutes after the sample shown in Figure 44. The subject's eyes are closed. The intermittent disappearance of the alpha activity and the slow rolling eye movements indicate that the subject has become drowsy.



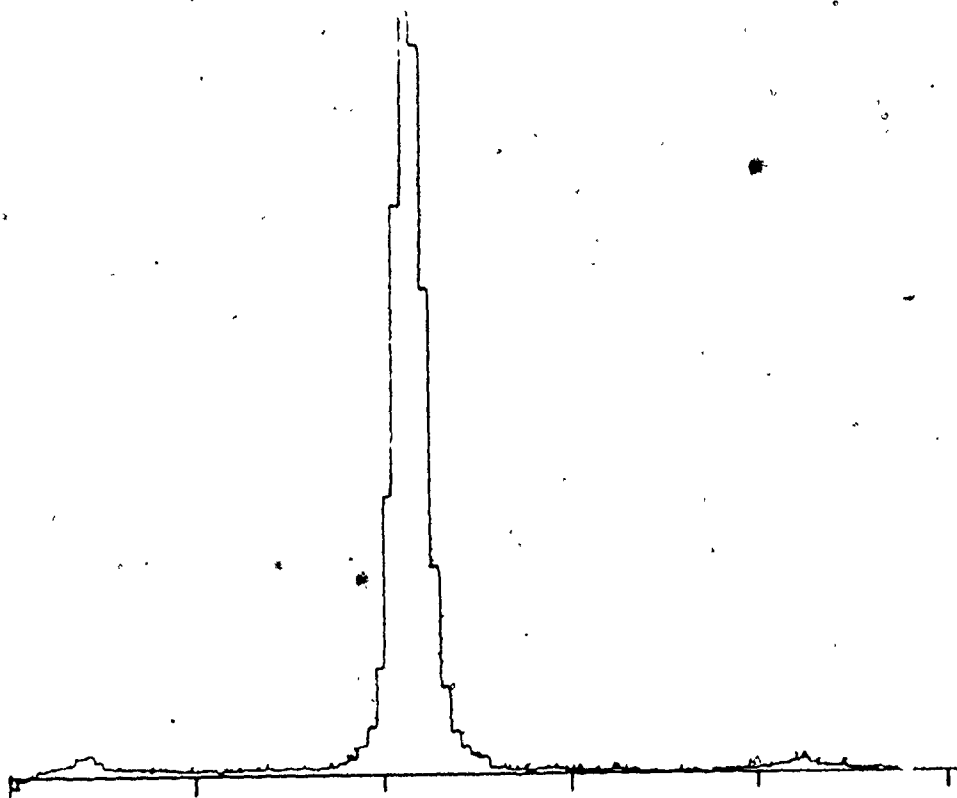
Subj. M.R.: DROWSY

100µv.
1sec.

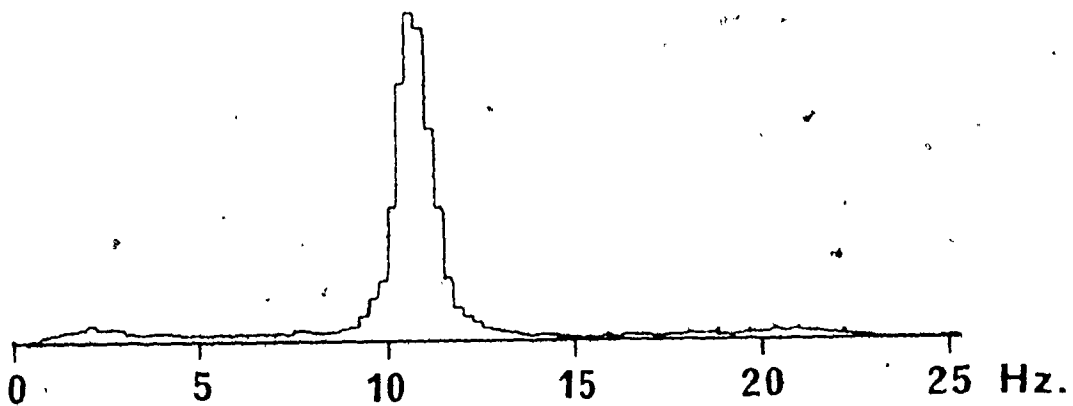
17

Figure 46

Power spectra of the occipital EEG shown in Figures 44 and 45. Although the power of the alpha has diminished in the drowsy state, the modal frequency remains identical.



ALERT



DROWSY

Subj.: M.R. PreDep. 2, Evening

Table 1

The correlation between body temperature and alpha frequency measurements taken at midnight and 8 a.m. on the pre- and post-control nights of sleep, and at 8 a.m., 1 p.m., and 7 p.m. throughout the control confinement period.

Correlation of Body Temperature With Mean Alpha Frequency

Subject	Correlation	Equation
W.K.	$r = 0.74$	$\text{Freq.} = -40.06 + .51 T$
L.G.	$r = 0.69$	$\text{Freq.} = -24.77 + .37 T$
M.L.	$r = 0.57$	$\text{Freq.} = -11.50 + .21 T$
L.M.	$r = 0.62$	$\text{Freq.} = -6.52 + .17 T$

Table 2

The change in modal (Mode) and average (2 Hz.) alpha frequency in the four-day deprivation subjects. The first column represents the average of the values recorded in the morning on each of the three pre-deprivation nights. The change in alpha frequency over deprivation (Hz.) is with reference to this value. Since, for subject D. M. , the EEG was not recorded on magnetic tape in mornings on the pre-deprivation nights, the value in the first column is the average of the evening alpha frequency. The drop in frequency (Hz.) on the morning of deprivation Day 2 is calculated by subtracting the value at that time from the value on the morning of deprivation Day 1, since there is no pre-deprivation morning average. The last alpha frequency measurement was made at noon on the fourth deprivation day for subject D. M. Since this is usually similar to the evening values, it was used as a basis for calculating the change in frequency, subtracting it from the average of the pre-deprivation evening values.

Subject		Pre-Dep. Morning	Dep. 2. Morning	Dep. 4 Morning
D. M.	Mode	10.46	9.00	8.46
	Δ Hz.		-0.63	-2.00
	± 2 Hz. Δ Hz.	10.15	8.88 -0.54	8.17 -1.98
M. R.	Mode	10.00	8.88	8.63
	Δ Hz.		-1.12	-1.37
	± 2 Hz. Hz.	9.96	8.93 -1.03	8.52 -1.44
W. P.	Mode	10.58	9.88	8.38
	Δ Hz.		-0.70	-2.20
	± 2 Hz. Δ Hz.	10.34	9.62 -0.72	8.28 -2.06
P. C.	Mode	9.38	9.63	8.63
	Δ Hz.		0.25	-0.75
	± 2 Hz. Δ Hz.	9.31	9.45 0.14	8.51 -0.80
<u>Average Δ Hz.</u>		Mode :	-0.55 Hz.	-1.58 Hz
		± 2 Hz. :	-0.54 Hz.	-1.57 Hz.

Table 3

The change in modal and average alpha frequency over deprivation for the seven-day subjects.

Subject		Pre-Dep. Morning	Dep. 2 Morning	Dep. 4 Morning	Dep. 7 Morning
P.W.	Mode Δ Hz.	9.67	8.38 -1.29	7.38 -2.29	7.13 -2.54
	+ 2 Hz. Δ Hz.	9.36	8.54 -1.02	7.22 -2.14	7.05 -2.31
D.G.	Mode Δ Hz.	10.04	9.58 -0.66	7.50 -2.54	8.13 -1.91
	+ 2 Hz. Δ Hz.	9.98	9.37 -0.61	7.47 -2.51	7.96 -2.02
I.T.	Mode Δ Hz.	11.30	10.88 -0.42	9.63 -1.67	8.50 -2.80
	+ 2 Hz. Δ Hz.	11.24	10.75 -0.49	9.60 -1.64	8.72 -2.52
T. S.	Mode Δ Hz.	9.25	8.65 -0.62	7.88 -1.37	7.63 -1.62
	+ 2 Hz. Δ Hz.	9.16	8.53 -0.63	7.85 -1.31	7.72 -1.44
	<u>Average ΔHz.</u>	Mode:	-0.75 Hz.	-1.97 Hz.	-2.22 Hz.
		+ 2 Hz.:	-0.69 Hz.	-1.90 Hz.	-2.07 Hz.

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