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SOMATOSENSORY EVOKED POTENTIALS AND THE MODIFICATION OF PHASE TRIGGERED ALPHA ACTIVITY

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CHAPTER I

INTRODUCTION

The human scalp-recorded sensory evoked potential (EP) is a low amplitude, short latency waveform of complex and variable configuration occurring in response to a wide variety of stimuli. Characteristic EPs have been recorded from the scalp of man in response to visual, auditory, somatosensory, olfactory and gustatory stimuli. The overall EP waveform has been found to be sensitive to variations of a wide variety of stimulus parameters, to behavioral, spontaneous and drug-induced physiological changes of the central nervous system, and to the effects of efferent neural activity. The EP has also been used in clinical studies of pathological, organic and functional CNS conditions and as a signal containing information useful in evaluating various aspects of normal human performance. Although there is often disagreement concerning the interpretation of evoked potential changes occurring in response to various experimental treatments, there is no doubt that the EP is an electrophysiological event which can vary systematically with changes in the internal and external milieu of the organism.

However, the scalp-recorded sensory EP is usually a minute signal buried in the ongoing electroencephalogram (EEG). The development of signal averaging techniques, providing a means of enhancing the time locked EP in relation to presumably random background EEG events, has

permitted extensive investigation of the averaged sensory evoked potential. Nevertheless, signal averaging techniques have also served to direct attention away from the fundamental questions concerning the relationships between the EP and EEG. In addition, the study of trial to trial EP variance as an important additional source of information concerning central nervous system processes has been neglected. Furthermore, the temporal demands of signal averaging may result in failure to detect short term systematic changes in EP component parameters (Brazier, 1963, 1964) while long term EP changes may mask the effects of treatment variables where the averaging requirement prolongs experimental sessions beyond the minimum time required to obtain behavioral data. Perhaps most important, the fundamental assumptions underlying signal averaging as a means of enhancing the EP remain largely unsubstantiated even though the technique has become a commonplace laboratory method of studying these time locked electrophysiological phenomena. In the absence of demonstrable relationships between EP and EEG phenomena, the interpretation of certain changes in averaged EP components occurring as a result of specific treatment conditions becomes tenuous if not impossible.

Evoked Potentials and Signal Averaging

Two general models describing the relationship between the EP and ongoing background EEG have been proposed. The simplest of these assumes that the EP is a fixed signal imbedded in a noisy (random) EEG background. This assumption provides the basis for the techniques of signal averaging and summation as means of enhancing the EP relative to background EEG activity. This model asserts that the EP can be described as a time-voltage function which is time locked to the stimulus, i.e., the

EP begins shortly after stimulus onset and follows a prescribed invariant time course given a specific set of experimental conditions (systematic changes or treatment conditions held constant). Thus, on every trial, the EP waveform is invariant while the time-amplitude function describing the EEG which is not time locked to the stimulus appears as a random variable. In this sense, the EEG may be considered to represent a "noise" process described by some distribution of voltage levels around a base line value usually taken to be zero. If such epochs of mixed EP and EEG signals are now summated, the EEG on the average will approach its base line level as EEG values of approximately equal amplitude but opposite polarity tend to cancel out, while the invariant voltages describing the EP monotonically add. Therefore, while the amplitude of the summated random EEG activity diminishes with successive samples, the non-random EP component increases in amplitude relative to the residual noise levels.

It may be easier to understand this process of variance reduction by using a hypothetical example to develop the appropriate statistics. Suppose that the frequency distribution characterizing some random variable had a mean equal to zero and a standard deviation of 20 units. Several samples of 100 observations each were then drawn from this distribution and the means of each sample were used to form a new frequency distribution. The Central Limit Theorem predicts that the resulting sampling distribution will be Gaussian (normal) with a mean equal to that of the parent distribution and a standard deviation or standard error (S.E.) which is related both to the population variance and to the number of observations which were used to derive each of the

sample means. Specifically, the S.E. of the sampling distribution is the population standard deviation divided by the square root of the number of observations per sample. For the present example, the population distribution had a mean of zero and a standard deviation of 20 units. Since each of the sample means was based on 100 observations, the resulting sampling distribution would be expected to have a mean of zero and a S.E. of 20 divided by the square root of 100 (i.e., 10) or 2 units.

Whenever the mean and standard deviation of the population distribution is known or can be estimated, confidence limits may be specified for the means of samples drawn from the distribution. For the previous example, 95 percent of the sample means would be expected to fall within the confidence limits of zero ±1.96 S.E., i.e., between 3.92 and -3.92 units. These fundamental principles of statistical sampling and estimation have also been used to establish confidence limits for the residual (mean) EEG following signal averaging. If the distribution parameters for EEG amplitude have been estimated by measuring the amplitude of the EEG (relative to zero potential) at a large number of sample points, then the resulting mean (base line) and variance parameters would characterize the amplitude behavior of the EEG for a particular state of the organism. The confidence limits for the mean of a small sample of EEG amplitude observations drawn from this EEG distribution can then be specified.

The simple signal averaging model which was proposed earlier assumes that invariant EP processes do not modify EEG characteristics. Therefore, the confidence limits for EEG samples derived from the non-stimulus EEG distribution are also appropriate for samples of EEG activity

which occur during the evoked potential sample period. If the values of the residual waveform of the averaged evoked potential fall outside the estimated confidence limits for the residual (mean) EEG, it may then be concluded that the observed deviation from the base line EEG level was due to the presence of the evoked potential waveform. Knowledge of EEG distribution characteristics could be used a posteriori to statistically confirm the existence of an observed EP component or a priori to select an appropriate sample size to assure detection of low amplitude components or to equate signal to noise ratios between conditions in which background EEG characteristics are likely to be different.

A more general signal averaging model is based upon the assumption that both the EP and the EEG have inherent variance. EP variance is less than that of the background EEG activity but irreducible for a specified set of experimental conditions, i.e., even if all variables known to affect EP variance could be held constant, there would be a small residual trial to trial variance. Walter and Gardiner (1970) refer to such variance as a "fundamental indeterminancy" of the EP analogous to the uncertainty of measurement in the physical sciences. The assumption that there is a fundamental EP variance is based on a particular model of CNS (cortical) organization, specifically that there is a large population of neurons and neuronal processes which may participate in the generation of the EP and that for any given afferent volley membership in the participating sub-population is governed in part by random processes. There are, of course, conditions under which the total number or percent of participating elements may be manipulated. For example, the observation that trial to trial EP variance decreases as stimulus intensity increases

(Brazier, 1963; Adey, 1965; Horvath, 1969) provides empirical support for these hypothesized stochastic EP processes, i.e., with increased stimulus intensity a greater proportion of the total population of EP generating elements becomes involved and, consequently, there is less latitude for trial to trial variation in the participation of individual generators.

The expected mean value of the variable EP component of the combined EP, EEG signal is equal in amplitude to that of the fixed EP amplitude model and the distribution properties of the background EEG remain unchanged (Walter and Gardiner, 1970). The "irreducible" EP variance appears as a presumably small error variance in the combined EP and EEG variance. Note, however, that with this model no "real" EP model emerges as a result of averaging because the averaged signal may or may not represent any of its constituent members. Most important to the present proposal is the fact that for both signal averaging models, the EP and EEG amplitude functions are presumed to combine additively and to be independent to the extent that neither signal significantly modifies the other during the sample period. Unfortunately, there is little reason to believe that evoked potentials do not modify the amplitude characteristics of the background EEG during the time the EP is observed. The nature of these EEG modifications must be evaluated before their influence on the averaged EP can be specified.

EEG and Signal Averaging

Spontaneous EEG has been characterized as continuous wave activity varying in amplitude, frequency and phase relations to the extent that the overall characteristics are similar to those of random noise (Elul, 1969). Of particular interest to the present discussion are the

EEG amplitude properties and their relation to the signal averaging process.

Although there have been relatively few investigations in which the EEG amplitude distribution per se (not to be confused with amplitude integration) has appeared as a dependent variable, those data which are available (Lion and Winter, 1953; Kozhevnikov, 1958; Saunders, 1963; and Elul, 1969) suggest that even the assumption of a Gaussian amplitude distribution may be valid only under certain conditions and that changes in the variance of EEG amplitude distributions in response to treatment variables may be substantial. These data are by no means surprising since EEG changes have been measured in numerous ways for a wide variety of behavioral and physiological conditions. However, any change in the overall amplitude characteristics of the EEG may also require a readjustment in the sample size of the averaged EP in order to obtain appropriate reductions in the magnitude of the residual EEG component. A failure to do so may adversely affect EP detectability.

It has been proposed (Schimmel, 1967; Walter and Gardiner, 1970, and others) that the influence of the EEG component in the averaged signal can be determined by characterizing the pre-stimulus EEG in terms of amplitude, spectrum, etc. This is correct only if the EP and EEG amplitude processes are independent during the period in which the EP is sampled. Suppose that the assumed independence is invalid under certain conditions as would be the case if the afferent volley were to desynchronize the EEG over widespread cortical areas. The net effect might be a sudden brief reduction in EEG amplitude variance, coincident with the EP sample period, which would not be predicted on the basis of prestimulus EEG

analysis. Further, it is possible that contrasted experimental treatments might differentially affect EEG only during the period immediately following the stimulus. For these reasons, a method is needed for studying the nature of EEG changes which might occur only during the evoked potential sample period. One approach to this problem is to cause the evoked potential to appear against a background of non-random rather than random EEG. It will be shown later how such controlled EEG may be used to detect and identify certain changes in the background EEG component of the averaged evoked potential.

Thus far, background EEG has been considered as a random process with many characteristics of noise. But it is obvious even from casual observation of the on-going EEG that there is considerable structure to certain events such as spindles, trains of alpha activity, brief bursts of beta or theta frequencies and other events. These phenomena may even characterize specific physiological states or recording sites. It may be possible, therefore, to predict the time course of the EEG with reasonable accuracy so that the EEG waveform becomes non-random within the accuracy of prediction. The ability to predict the time course of EEG activity over that period of time corresponding to the sampling period of the EP has two important consequences. First, if the EEG function is known and can be manipulated in a controlled fashion, specific predictions can be made concerning changes in expected values of the resulting combined EP and EEG signals under the conditions of the traditional signal averaging models. Departures of the combined signal amplitudes from the predicted values would imply violations of these basic assumptions. It may then be possible to determine the nature of the relationships between EP and EEG

processes under these conditions and to reformulate signal averaging models appropriate to particular experimental conditions.

A second consequence of averaging EPs against an EEG background with a known time course is that the variance due to EEG may be substantially reduced. If the waveform of the EEG component of the combined EP, EEG sample were known precisely, it could be subtracted from observed sum of the signals on any given trial leaving only the EP waveform. Under less ideal conditions, the time course of the EEG will not be predicted accurately on every trial so that some averaging may still be required to extract the EP waveform. Nevertheless, the amount of averaging should be substantially reduced if a large portion of the normal amplitude variance of the EEG is successfully removed.

One method of gaining control over the variable properties of the EEG is based upon a detailed statistical analysis of its amplitude behavior as a function of time. For example, the application of multivariate regression analysis to the prediction of the EEG waveform characteristics has been described in some detail by Fenwick, et al. (1969), and Walter and Gardiner (1970). Following an analog to digital conversion of the EEG, a regression analysis is performed in order to determine the optimal combination of EEG amplitude values obtained during brief sample epochs for predicting the value of a specific amplitude point. Successive sets of predictors are then derived to predict later EEG values over a time period corresponding to the EP sampling period. Given these sets of EEG predictors and the observation of EEG amplitudes immediately preceding stimulus delivery, the time course of the background activity can be predicted. According to the averaging models, if the predicted

EEG component is then subtracted from the observed EEG plus EP signal, the residual signal should represent EP processes alone plus some error variance. The magnitude of the error variance will depend primarily upon the accuracy with which the background activity has been predicted.

The success of autoregressive predictors depends on stationary EEG processes, i.e., state changes which affect EEG properties will invalidate predictors derived under dissimilar EEG conditions. Further, if such predictors are to be used to enhance single trial EP data by means of the subtraction process, then it must also be demonstrated that the EEG component of the combined signal is not altered during the time in which the EP is sampled. Specifically, changes in background activity may occur coincident with EP processes but not be evident from inter-stimulus samples in which EEG state changes are monitored or during which EEG predictor data are obtained. Finally, the autoregressive analysis requires analog to digital conversion, extensive digital computation, and storage of digital predictor data characterizing pre-stimulus EEG samples. This procedure is therefore limited in application to those facilities where high-speed large capacity computers are available.

Some EEG events such as trains of alpha activity are sufficiently invariant and of long enough duration that relatively simple measurements can detect them, thus permitting prediction of subsequent EEG events over short periods of time. Remond (1968) used three parameters to characterize alpha events: polarity, amplitude and half-wave width, i.e., the time between the two base line zero-crossings of the half-wave. A device which incorporates these parameters is essentially a pattern recognition system which has been set to detect alpha events with specified properties.

Remond used such a device to study the spatio-temporal organization of the alpha rhythm by averaging the EEG which occurred during a several hundred millisecond time "window" beginning when the programmed criteria defining the triggering alpha event were met. He found that the average of EEG activity following the triggering alpha event was generally the alpha rhythm. This might be expected since the alpha rhythm rarely occurs as a single isolated event but rather as a burst of synchronous activity of variable, often extended, duration.

A modification of Remond's method of pattern recognition of the alpha rhythm was selected for use in the present investigation for two reasons. First, the relative simplicity of the pattern recognition system needed to detect alpha activity was an important consideration in the decision to use this method of controlling EEG parameters. Second, the predictable properties of the alpha rhythm, together with the observation that alpha activity is a prominent feature of EEG recorded from widely separated sites under a variety of states, led to the selection of alpha activity as a representative type of "background" activity. The manner in which controlled alpha activity may be used to investigate the relationship between EEG and EP processes during the averaging process is discussed later.

Evoked Potentials and the Alpha Rhythm

There are at least two ways in which selection of alpha rhythms as a type of EEG background activity is likely to influence the observed relationship between EEG and EP processes. First, by selecting alpha activity, one is also sampling the states of CNS organization which produce alpha-like EEG, i.e., such events may in a general way be

indicative of the state of "arousal" or "vigilance" (in the Hebbian sense) of the central nervous system. It may be that the relationship between EP and EEG processes is different under conditions in which beta or some other type of activity is the predominant feature of the EEG. However, activity within the alpha band can be a prominent feature of the EEG under a wide variety of conditions so that by careful selection of predictor parameters, it may be possible to define a number of such states for separate study. A second influence of the alpha rhythm on the EP waveform may occur when stimuli are phase locked to EEG events as a result of the so-called alpha excitability cycle. For example, Remond (1968) and others have argued that different phases of the occipital alpha rhythm also represent different physiological states so that photically induced afferent volleys which reach the cortex at different phases of the alpha cycle will produce different EP waveforms attributable to altered EP generator states. Remond's data appear to support his position. However, when stimuli are delivered at specified alpha phases, the resulting signal will also contain a large-amplitude non-random alpha component which would be expected to be different for various phases of the EEG whether or not the EP is also different.

Callaway and Layne (1964) obtained both visual EP and non-stimulus EEG samples (250 millisecond sample length) for various phases of the occipital alpha rhythm and by subtracting the EEG average from the EP average of the same phase, they found that the resulting waveforms were similar for all phases of the alpha rhythm. Therefore, most of the phase related signal changes observed by Remond during the first 250 milliseconds of his EEG samples were probably due to the addition of the

non-random alpha components to otherwise similar EP signals. Callaway and Layne did find small phase-related residual differences in the amplitude and latencies of the visual EPs after EEG subtraction which they attributed to changes in the EP waveform. However, this conclusion does not necessarily follow from their data since the background alpha activity occurring during the EP sample period may be slightly altered with respect to amplitude, phase or frequency. Thus, the no-stimulus alpha averages which were subtracted from the combined EP, EEG signals could have produced a systematic source of error in the residual signals.

There is, of course, evidence that the EEG background can change during the time course of the EP. One example of such a change is the well-known phenomenon of "alpha blocking." Ciganek (1969) found a marked decrease in the variance of single trial visual EP (plus EEG) amplitude occurring approximately 80 milliseconds after stimulus onset and proposed that this reduction in variance below pre-stimulus EEG levels corresponded to the onset of the alpha block. It has been observed that alpha blocking habituates with stimulus repetition and in general that cortical EEG desynchronization in response to peripheral stimulation becomes localized to the primary receiving area with successive stimuli (Sokolov, 1963; Sharpless and Jasper, 1956). Such spatial changes in EEG desynchronization may also alter the ratio of EP to EEG amplitudes and therefore the detectability of EP components. At a remote recording site, i.e., one in which the source of the EEG is not the source of EP activity, desynchronization should be greatest early in a series of stimuli and become less pronounced with stimulus habituation. Therefore, EP components (with latencies greater than the onset of EEG modification) should be more

detectable early in the stimulus series when the amplitude of the desynchronized background activity is lowest. However, as widespread desynchrony diminishes with habituation, EEG activity at the remote site should be less affected, EEG "noise" should increase and EP component detectability should decrease. Thus, in the absence of information concerning the nature of possible EEG modifications occurring during the EP sample period, it may be erroneous to attribute changes in EP component detectability or variability to changes in the evoked potential or to those neurophysiological mechanisms which are believed to mediate EP components. Similar interpretive difficulties arise wherever experimental variables are likely to alter EEG characteristics between treatment conditions. Knowledge of pre-stimulus EEG parameters does not necessarily provide a solution to these problems unless it can be demonstrated that estimated EEG parameters continue to be valid during the EP sample period.

Stimulus-provoked alpha activity is also a common phenomenon under conditions of EEG desynchrony, drowsiness and low behavioral task demands (Morrell, 1966). Although stimulus-provoked alpha can be recorded over widespread areas of the scalp, the long latency of .5 to 1.5 seconds indicates that this phenomenon is not a likely contaminant of the averaged EP within the usual half-second sample period. However, if stimuli are closely spaced and regularly presented, other forms of EEG entrainment may produce serious distortions in the resulting averaged signal. Entrainment refers to the synchronization of EEG activity by stimulus events. As an example, a well developed period of alpha-like activity ("ringing" or "after-activity") may develop several hundred milliseconds following stimulation. Although the relationship between ringing and normal alpha

EEG may be debatable (see Chapter 4), the possibility of interactions between these stimulus synchronized events and subsequent EP samples should be considered. If stimuli are presented with a constant inter-stimulus interval within this period of synchrony, the net effect may be that EEG processes become non-random with respect to stimulus processes. The resulting averaged waveform may be additively modified by the non-random background activity carried over from each successive sample.

The nature of the various interactions between EEG and EP processes may depend on the proximity of the recording electrode to the EP generator sites and/or the coherence of the electrical activity between these sites. For an active electrode which is close to the EP generator, it might be predicted that many of the elements which are generating EEG activity during a pre-stimulus epoch will be required to process sensory information following stimulus delivery. Consequently, during the EP sample period, the background EEG activity will be altered to the extent that the role of some subgroup of EEG generators has changed to meet the functional demands of the stimulus situation. If a substantial proportion of the total number of generator cells remains unaffected by these demands, the pre-stimulus EEG will continue with little change during the time period in which EP processes are being developed by the remaining cells. For any electrode site where the underlying cortex is functionally involved in both EP and EEG processes, the traditional independent process signal averaging model is probably invalid but new models might be developed where the nature of the EEG change can be determined.

The relationship between EP and EEG processes at a recording

site which is remote with respect to the EP source should most closely approximate the conditions of the independent process model, i.e., the underlying cortex at the recording site generates background EEG which is not substantially influenced by the EP signal being volume conducted from a distant location. There are, however, certain conditions under which interactions between EP and EEG signals might occur even though the underlying processes were functionally independent at the recording site. Specifically, if EEG features are used to control sample selection as in the proposed study, there may be circumstances in which the EEG at the recording site is highly correlated with EEG activity at the EP source. Under these circumstances, selection of EEG events at a remote site also selects to some extent the functional state of the EP generators. If functional interactions between the EP and EEG processes do occur at the EP source, they may also appear at the distant recording electrode. Such inter-electrode interactions could be studied by comparing data obtained from periods of high EEG coherence with those obtained from trials in which EEG activity derived from the EP source was essentially random with respect to EEG recorded at a distant point.

Phase Triggered Alpha Activity and Signal Averaging

The method which was selected for the study of stimulus modification of background alpha activity during the EP sample period depends upon the non-random characteristics of samples of phase triggered alpha activity. Suppose that separate samples of the alpha rhythm were initiated as the EEG crossed the base line in the positive and negative directions. If the samples of each type of alpha activity were then averaged together to reduce some of the inherent variability in the alpha rhythm, the result

would be two alpha rhythm averages which were 180 degrees out of phase. The amplitudes (relative to EEG base line) of the resulting averages might be measured at any number of points along the waveforms and the amplitude differences between the two phase triggered averages at successive time points could be computed. These differences would be greatest at points corresponding to the peaks of the averaged alpha activity. If a number of pairs of averages of the phase triggered alpha rhythm were thus compared, a distribution of difference scores could be obtained for each sample point of the averaged waveforms.

If a stimulus were then delivered at the points at which the phase triggered samples of alpha activity were initiated, the resulting averages would be a combination of the alpha rhythm and the EP waveform. According to the assumptions of the previously discussed signal averaging models, the presence of the stimulus produced evoked potential should not alter the characteristics of the background alpha rhythm (even though the overall appearance of the averaged alpha rhythm waveforms would be distorted due to the addition of the EP waveform). Therefore, the amplitude differences between these phase triggered EEG plus EP averages should be the same as those which were obtained under the non-stimulus conditions assuming that interactions between EP and EEG processes did not occur. However, it was also suggested that modifications of background alpha activity are likely to occur under some circumstances. Consequently, there may be discrepancies between the phase related difference data obtained under non-stimulus and stimulus conditions. The quantification and interpretation of the various difference measurements will be discussed in greater detail in the remaining chapters.

The somatosensory evoked potential was selected for use in this study because its components are believed to arise exclusively from the primary sensory and adjacent association cortices. For stimulation of the Median Nerve on the right, these cortical areas are located almost directly beneath the C3 recording position of the international 10-20 system of electrode placement. Therefore, the C3 recording site provides an opportunity to examine the behavior of EEG recorded very near the source of the EP. The vertex (Cz) was selected as a second recording site where EEG processes might be expected to be relatively less affected by those processes generating the somatosensory EP. This arrangement of recording electrodes relative to the presumed source of the EP might permit the detection and evaluation of some of the previously discussed interactions.

Purpose

The purposes of this investigation were, first, to obtain reliable non-random alpha-frequency averages from central C3 and Cz scalp recording sites and to examine some of the properties of these signals; second, to determine whether or not Median Nerve stimulation significantly modifies these properties of the non-stimulus alpha averages and, if so, to what extent such modifications occur within and between the two recording sites; and finally, to evaluate possible stimulus modifications of background EEG as they may relate to the process of signal averaging as a means of extracting evoked potential information from the scalp recorded EEG.

CHAPTER II

METHOD

Subjects

Subjects participating in the experiment were eight male, paid volunteers aged 24 to 33 with previous experience in EEG and EP studies. None of the Subjects was selected on the basis of having abundant alpha activity.

Apparatus

All EEG data, stimulus markers and EEG (alpha rhythm) zero-cross data were recorded for visual monitoring through a Grass Model 6 Electroencephalograph and permanently stored on magnetic tape through an Ampex Type 1300 FM magnetic tape recorder for subsequent electronic analysis. EEG was recorded with Grass Instrument gold electrodes from C3 and Cz referenced to the linked earlobes. In addition, Subjects were grounded at the right wrist proximal to the site of stimulation to reduce the influence of shock artifact and stray electrical signals. During all recording sessions, Subjects rested on a bed located within a dimly illuminated, sound attenuated, electrically shielded room. A 40db white noise was provided to further reduce the effects of extraneous noise originating outside the recording room.

A Fabri-Tek Instrument Model 1062 Instrument Computer was used

for on-line monitoring and later reproduction of averaged evoked potentials and phase triggered EEG samples, and for subtraction of empirically derived EEG functions from the average of combined EEG plus EP signals. Averaged EPs, EEG samples and corrected EP averages were written out graphically through a Hewlett-Packard 7004B X-Y Recorder for further analysis.

Stimuli consisted of 1/2 miilisecond square wave pulses delivered to the right median nerve at the wrist by a Grass S8 Stimulator through a Grass SIU5 Stimulus Isolation Unit. Shocks were delivered to the median nerve through two adjacent electrodes with the cathodal lead proximal to the anode. Stimulus intensity was adjusted to produce a barely perceptible thumb twitch and a sensation in those parts of the hand mediated by the median nerve. The rate of EEG sampling and stimulus presentation was regulated by the EEG control system in conjunction with a paper tape program read by a BRS, TRS-3 Tape Reader.

Pattern Recognition of the Alpha Rhythm

A pattern recognition system which monitored the frequency and amplitude characteristics of the EEG was used to detect the presence of alpha rhythm and to initiate EEG samples when appropriate pre-programmed conditions were met. The operating principles of the pattern recognition system are explained in reference to Figures 1 and 2 below.

Figure 1 depicts an isolated segment of EEG consisting of a series of large components upon which there is a small amount of "riding" activity. The time between successive base line crosses (zero voltage in figure 1) may be used to derive one measure of EEG frequency. Frequency (Hz) is equal to 1/period, where period designates the amount of time

Figure 1. Alpha rhythm frequency and amplitude measurements. Alpha activity was said to be present whenever the time between successive base line crossings fell within the pre-determining range of half wave zero-cross values corresponding to 7.5 to 13.5 Hz EEG activity. EEG amplitude measurements were made relative to a zero-volt base line value. The vertical arrows indicate the points at which peak to base line amplitude measurements were made.

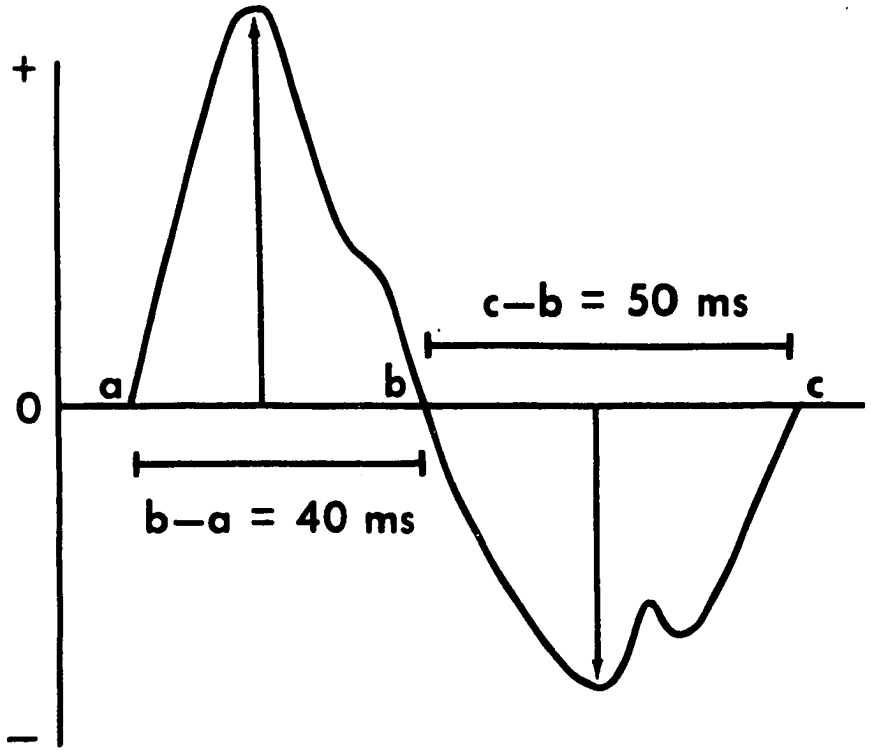

Figure 1. Alpha rhythm frequency and amplitude measurements.

required to complete one full cycle. In Figure 1, a full cycle is represented by the segment of waveform between points "a" and "c". If the elapsed time between "a" and "c" were 90 milliseconds, i.e., .090 seconds, then the frequency derived from these base line crossings is Hz = 1/.090 or 11.1 Hz. EEG frequencies may also be estimated from the half-wave segments "b-a" and "c-b" from the relationship, Hz = 1/2(half-period). Where b-a = 40 ms., Hz = 12.5 and for c-b = 50 ms., the frequency is 10 Hz. Frequency estimates derived from successive base line crossings (half-waves) were used in this study as a means of detecting the presence of alpha activity. Specifically, the alpha rhythm was defined as having a frequency range of 7.5 to 13.5 Hz, and a period range of 133 to 74 milliseconds. The corresponding range of half-periods or time between successive base line crossings was therefore 66.5 to 37 milliseconds. In this experiment, a counting sequence was begun with each base line transition of the EEG and terminated with the following base line crossing. If the accumulated millisecond count exceeded 37, but was less than 66 milliseconds, the EEG half-wave was identified as alpha activity.

Alpha activity which may be detected from base line transitions may be further categorized according to the base line to peak amplitude which the half-wave attains during the time between base line crossings (Figure 1). Although base line to peak amplitude per se does not identify alpha activity, this variable in combination with the frequency data may permit the detection of alpha activity which is more likely to be followed by several additional cycles of alpha rhythm. By appropriate adjustment of the amplitude threshold criterion, it was possible to detect the higher amplitude alpha activity which frequently accompanied

short periods of sustained alpha rhythm while ignoring the occasional low amplitude, isolated EEG events of alpha frequency which occurred during periods of EEG desynchronization.

From pilot studies it was found that a simple EEG half-wave pattern detection system based upon frequency and amplitude criteria was not always satisfactory in reliably detecting events which were followed by alpha activity of any appreciable duration. This was due to the fact that alpha recorded from the central regions used here was not generally as abundant as that recorded from more posterior sites. Consequently, for centrally recorded EEG, isolated events fulfilling programmed criteria were likely to trigger samples which were followed by predominantly non-alpha EEG. By increasing the amplitude criterion to approximately one-half the average peak to base line amplitude of the Subject's alpha rhythm, some improvement in the detection of longer epochs of alpha activity was attained. If higher amplitude criteria were used, the inter-sample interval became unacceptably long for purposes of the present study. By imposing the additional requirement that more than one consecutive alpha event of specified frequency and amplitude be observed before initiating a sample, a further improvement in detecting sustained bursts of alpha was realized.

The alpha rhythm pattern detection criteria which were finally selected were general enough that a re-definition of these parameters was not required for each Subject. Specifically, the system recognized as alpha activity any EEG events with a frequency (measured from successive base line crossings) of 7.5 to 13.5 Hz. Positive and negative amplitude thresholds were generally set at 50 per cent of the maximum peak to base

line excursions of the alpha rhythm. Further, EEG samples could be initiated only when three consecutive alpha events of appropriate frequency and amplitude occurred.

A sequence of EEG events leading to the initiation of an EEG sample is illustrated in Figure 2. In this illustration, the positive and negative amplitude thresholds are indicated by the broken lines above and below the indicated base line. The EEG base line crossings identified by dots along the zero potential line were used to estimate the frequencies of the successive half-waves of EEG. The first three half-waves of the illustrated EEG segment failed to meet amplitude and frequency conditions and were therefore not recognized by the pattern recognition system. The characteristics of the following negative half-wave (number 4) did meet the programmed criteria causing the pattern recognition system to record the presence of this event and to begin observing sequential properties of the EEG. Because the system was not designed to analyze riding activity occurring between base line crossings, waves a and b of Figure 2 had no influence on the decisions of the pattern recognition system. Wave 5 failed to meet criterion conditions and according to pre-programmed instructions, the sequential counting operation of the recognition system was stopped and reset to zero. Finally, the sequential count began again following wave 6 which was recognized as an alpha wave of appropriate amplitude and continued since waves 7 and 8 also met criterion conditions. The occurrence of three consecutive half-waves of alpha activity of appropriate characteristics resulted in the initiation of an EEG sample coincident with the final zero-cross of wave 8. For this particular sample, the alpha rhythm persisted for several hundred milliseconds.

Figure 2. Pattern recognition of the alpha rhythm. EEG events were recognized as alpha activity if the time between zero crossings was greater than 37 but less than 66 milliseconds. EEG samples were initiated only when three consecutive half-waves of alpha activity of appropriate amplitude (indicated by the broken lines above and below the zero-volt base line) were observed. Because waves 6, 7, and 8 were of appropriate frequency and amplitude a sample of indefinite duration was initiated at the point indicated by the arrow.

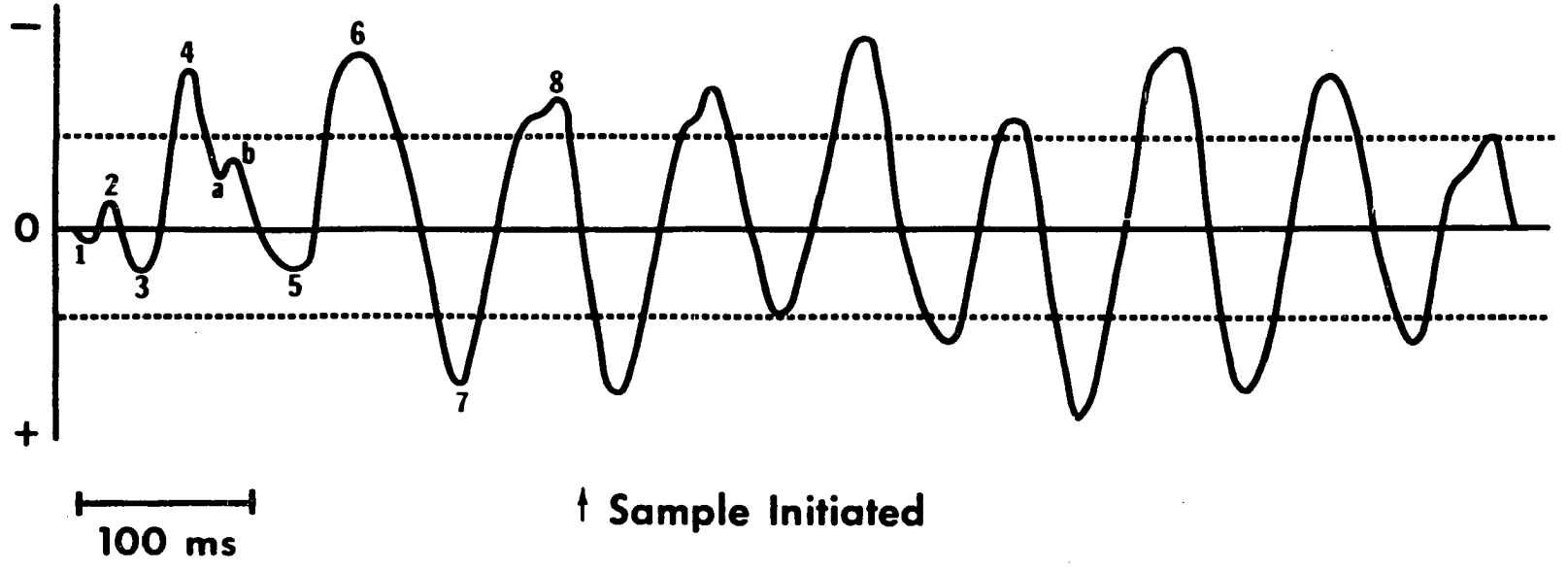

Figure 2. Pattern recognition of the alpha rhythm.

The polarity of the first wave of the EEG triggered alpha rhythm sample illustrated in Figure 2 was positive. This is, of course, due to the fact that the last wave of alpha activity of the pre-sample EEG was negative. By instructing the pattern recognition system to initiate samples only when the last observed wave in the pre-sample EEG was of specified polarity, it was possible to manipulate the phase of the resulting EEG sample. If the last pre-sample wave of alpha activity in Figure 2 had been positive, then the subsequent EEG sample would have begun with a negative wave. This feature of the pattern recognition system was externally programmed so that the order of occurrence of initially positive or negative EEG samples could be manipulated to fit the needs of the experiment.

In addition to controlling the phase of the EEG sample, it was also required that Median Nerve stimuli be delivered on half of the samples of each phase. There were, therefore, four possible sample types depending on whether or not the pattern recognition system initiated initially positive or negative EEG samples and on whether or not the sample was or was not accompanied by stimulus delivery. These various signal combinations are summarized in Table 1. The positive and negative trigger conditions indicate that the criterion conditons were met and that a sample was initiated during a positive or negative transition of the alpha wave. The trigger conditions were such that the resulting positive and negative alpha samples were 180 degrees out of phase. The first alpha peak for a "+trigger" sample was positive while that of the "-trigger" condition was negative when the appropriate trigger conditions were met. The use of either the Cz or C3 recording sites as a source of

EEG information to be processed by the pattern recognition system was distributed among the eight Subjects. For the first five Subjects Cz EEG data were used for this purpose. For the last three Subjects C3 EEG data were processed by the system. In either case, the EEG data from both recording sites were simultaneously sampled and data from both recording sites were subjected to all subsequent analyses.

TABLE 1

EEG AND EP SAMPLE CONDITIONS

Stimulus	Alpha Rhythm Phase	Recording Site	
		Cz	C3
no stimulus	+ trigger	Cz, +EEG	C3, +EEG
	- trigger	Cz, -EEG	C3, -EEG
stimulus	+ trigger	Cz, +EP	C3, +EP
	- trigger	Cz, -EP	C3, -EP

The nomenclature presented in Table 1 will be used throughout the remaining chapters to identify the type of sample, i.e., EEG or evoked potential plus EEG, and the trigger conditions which initiated the sample. It should be noted that "+EP" or "-EP" designates a signal consisting of an evoked potential and some non-random EEG component. The notation "±EEG" or "±EP" will be used when reference is made to the averages or samples obtained under both the positive and negative (i.e., ±) sampling conditions.

The order of occurrence of the four types of samples was randomized within the various trials under the condition that any particular sample could not be repeated until each of the remaining three sample types had been obtained. A minimum inter-sample interval of three seconds was also imposed so that the minimum programmed inter-stimulus interval (combining both trigger conditions) was three seconds with a range of 3 to 9 seconds. The actual inter-sample intervals were somewhat greater since they were also contingent upon the occurrence of appropriate alpha events.

Procedure

All data from each Subject were collected in a single morning session. No special instructions were given to the Subject except that he should relax and remain awake. The Subject was also requested to keep his eyes closed in order to facilitate alpha activity in the EEG record. All EEG data were collected during four successive 30 minute (approximately; the time required for each trial depended on the abundance of alpha activity) recording sessions each separated by a 10-minute rest period. The relatively long recording sessions were used in preference to several shorter trials in order to allow the EEG and behavioral states of the Subject to vary spontaneously, thereby providing a more representative sample of the resting state against which the relationships between EEG and EP processes could be evaluated.

Data Analysis

The various types of EEG and EEG plus EP samples were obtained from the magnetic tape records and sorted into the classes presented in

Table 1. Successive observations of each of these signal types were then averaged in groups of N = 16 samples and written out through the X-Y plotter for subsequent measurement. Three general classes of analyses were then performed. The purposes of these analyses were, first, to describe the averaged ±EEG waveforms, second, to examine the differences within and between the ±EEG and ±EP averages which were due to the phase triggering conditions, and, finally, to detect and quantify possible systematic changes in the relationships among the ±EEG and ±EP signals. Unless otherwise stated, all measurements and analyses were based upon within Subject data.

±EEG Descriptive Statistics

The first block of analyses was performed on the ±EEG averages only. The purposes of these analyses were to derive parameters describing the frequency, phase, and symmetry characteristics of the Cz and C3 alpha rhythm averages, and to define sample points for use in later comparisons of the differences between the ±EP and ±EEG signals.

EEG frequency and phase analyses. These data were used as a means of detecting differences between the fundamental waveform characteristics of the alpha activity recorded from the Cz and C3 electrode placements. The derivation of the frequency and phase parameters is illustrated in figure 3. Frequency (Hz) estimates were derived from the periods of the positive triggered Cz and C3 alpha rhythm averages (Hz = 1/period). The period was defined as the time in milliseconds between the first and second positive peaks (C-A in Figure 3) of each ±EEG waveform. Several pairs of Cz and C3 frequency observations were thereby obtained within and between the data collection trials. The overall

significance of the differences between the paired Cz and C3 frequency estimates was then evaluated by using two-tailed t-tests for correlated data. The presence of phase shifts between the Cz and C3 EEG averages was similarly evaluated by comparing the latencies of the first positive peaks (A, Figure 3) of the successive pairs of Cz and C3, +EEG averages. Whenever significant phase shifts were observed between Cz and C3, the analysis was repeated using the latencies of the first negative peaks of the -EEG averages (A', Figure 3).

EEG symmetry analyses. The presence of symmetry between 180 degree out of phase alpha rhythm averages has important implications for one method of recovering the EP waveform from this type of EEG background activity. Tukey (1968) proposed that the EP could be recovered by adding together the EP plus EEG samples obtained under each of these trigger conditions thereby cancelling the equal but opposite i.e., symmetrical, EEG components. In the present investigation it was assumed that if the added ±EEG averages failed to cancel then the background EEG components of the ±EP signals might also fail to cancel following the addition of the +EP and -EP averages. Under these circumstances any estimate of the EP waveform obtained by ±EP addition would be in error due to the presence of incompletely cancelled alpha activity. Incomplete EEG cancellation following the addition of the +EEG and -EEG averages could occur as a result of adding symmetrical waveforms which were not 180 degrees out of phase or from the addition of asymmetrical waveforms.

A preliminary analyses of the ±EEG averages indicated that these waveforms were occasionally ineffectively cancelled when added together in the memory of the 1062 Instrument Computer. In those instances

Figure 3. Derivation of averaged EEG frequency, phase and symmetry parameters. Frequency of the averaged alpha rhythm was derived from the period (C-A) of the +EEG average. Phase shifts between the Cz and C3 alpha rhythm averages were determined by comparing the latencies to the first positive peaks. Waveform symmetry was evaluated by comparing the times required for the first peak to peak transitions of the +EEG and -EEG averages.

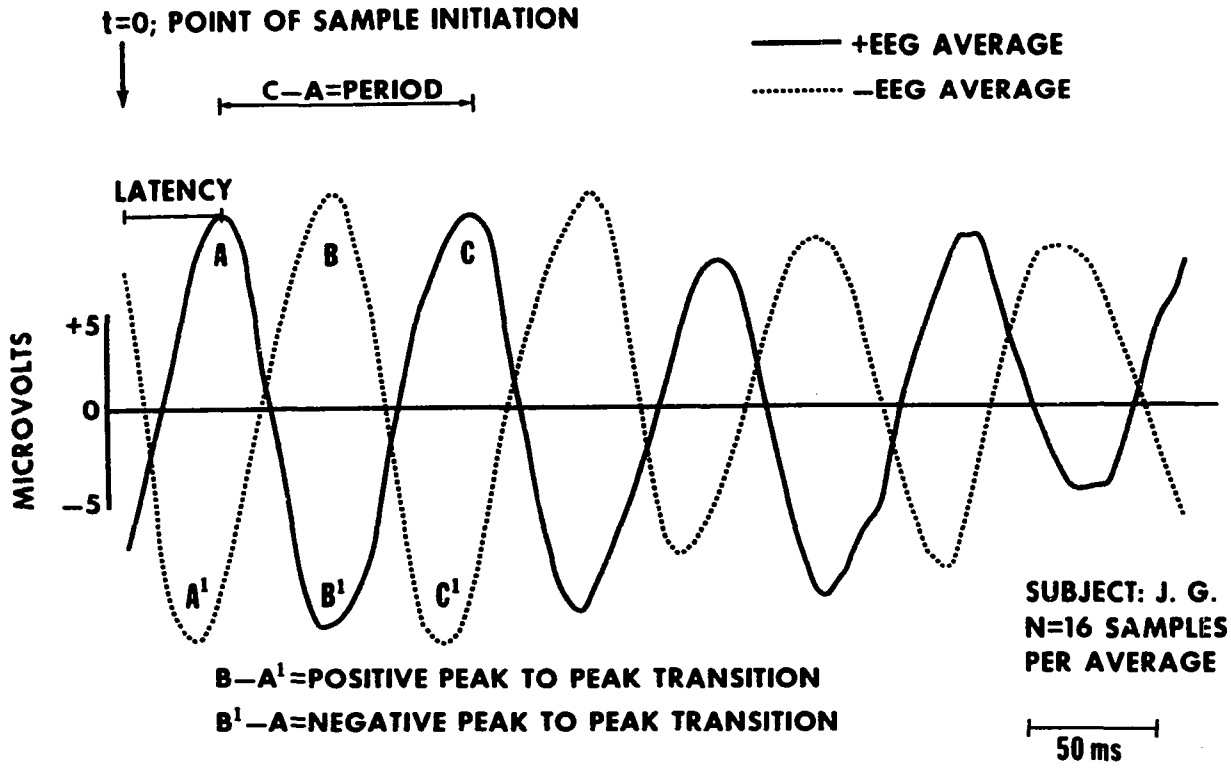

Figure 3. Derivation of averaged EEG frequency, phase and symmetry parameters.

where the residual error was large, a close inspection of the individual +EEG and -EEG averages also revealed that these waveforms were noticably asymmetrical in appearance. Such asymmetries were evaluated by comparing the time required for the first negative peak to peak transition of the +EEG averages with the time required for the first positive peak to peak transitions of the -EEG averages. For the pair of waveforms illustrated in Figure 3 these quantities are indicated by the expressions B' - A and B - A' respectively. The transition time derived from the first +EEG average was compared with that obtained from the first -EEG average and so forth for successive pairs of phase triggered EEG averages obtained within and between the four trials. The significance of the differences between these paired data was evaluated by using t-tests for correlated data.

The averages presented in Figure 3 were selected to illustrate the presence of distortions in the +EEG and -EEG averages. Note that the time required for the A' to B transition is greater than that required for the A to B' peak to peak transition. For this Subject, the positive peak to peak transition times were generally longer than those of the negative excursions for averages obtained under both trigger conditions. The incomplete EEG cancellation which was observed following the addition of the asymmetrical ±EEG averages could not be compensated for by changing the phase at which the samples were initiated. For example, if peaks A and A' of Figure 3 were to be aligned then the latency discrepancy between peaks B and B' would increase.

EEG sample point location. The method of locating sample points corresponding to the peak latencies of the non-stimulus averaged alpha

rhythm is illustrated in Figure 4. The five waveforms in the upper half of this figure are successive averages of alpha activity obtained under the positive trigger conditions (so that the first peak is positive in value). The lower five waveforms are averages of the negatively triggered samples of alpha rhythm. The successive peaks of all alpha rhythm averages were alphabetically identified and the latency from the point of sample initiation to each peak was then measured. The mean latency of each successive peak was then derived by combining all of the data (within and between trials) obtained from the positive and negative trigger conditions. This procedure was used to derive a unique set of sample points for the Cz and C3 recording sites of each Subject. All subsequent amplitude measurements were then made at these time points.

EEG Difference Analyses

The derivation of various "difference" measurements is explained in reference to Figure 5 where the several phase triggered averages obtained under non-stimulus and stimulus conditions are illustrated. The pair of waveforms in the upper portion of the figure are the non-stimulus, positive and negative triggered Cz alpha rhythm averages. The pair of signals in the lower half of the illustration are the corresponding averages of the phase triggered samples obtained under stimulus conditions. Each of these averages was the first in the series of averages obtained during the first of four consecutive trials. However, the locations of the sample points in the illustration were based on the mean latencies of all averaged alpha activity according to the previously outlined procedure.

The first difference analysis was performed upon the non-stimulus

Figure 4. Peak latencies of the averaged alpha rhythm and the location of sample points. The latencies of successive peaks of the averaged non-stimulus alpha rhythm waveforms were measured from the point of sample initiation, t = 0. Sample point A was defined by the mean of the peak A latencies of the combined averages of the positive and negative triggered EEG samples. Similarly, sample points B through F represent the mean peak latencies of the successively later peaks of these waveforms. EEG waveform measurements were not routinely performed beyond point F, i.e., after three complete cycles of the averaged alpha rhythm. The sample points which were derived from the non-stimulus EEG averages were also used to locate the measurement points for the averaged EEG plus EP waveforms.

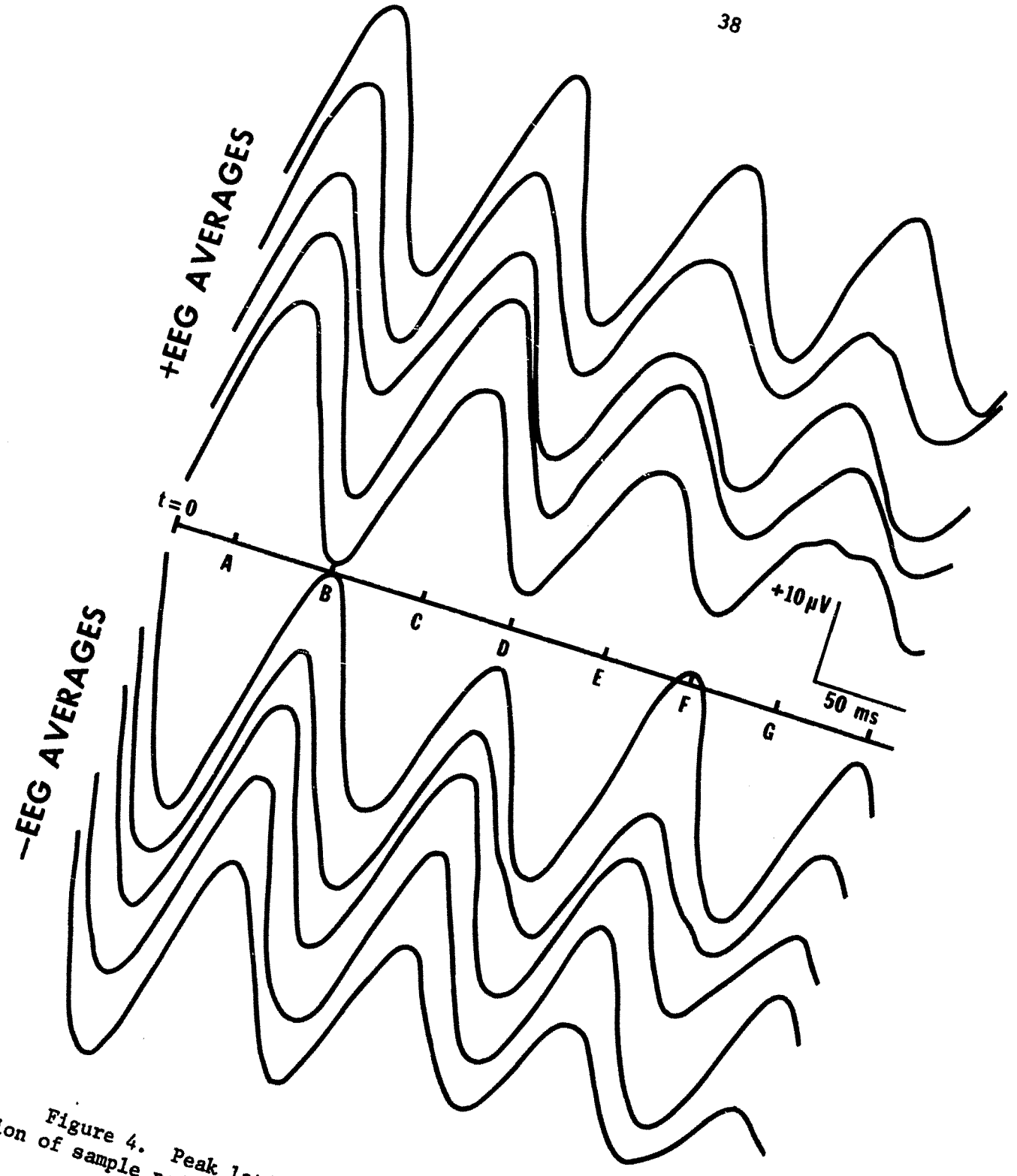

Figure 4. Peak latencies of the averaged alpha rhythm and the location of sample points.

alpha rhythm averages to determine whether or not the phase related differences in the two waveforms were significant at progressively later sample points. This analysis was necessary to confirm the predicted non-random nature of the averaged alpha activity at the points of interest. The analysis was accomplished at each sample point by measuring the amplitudes of the positive and negative triggered waveforms relative to an arbitrary base line value (the estimated line of zero potential). These amplitude values were obtained for each successive pair of averaged signals within and between trials yielding a total of approximately 16 pairs of observations for each sample point. A statistical analysis was then performed upon the paired data to determine whether or not the mean amplitudes of the positive triggered averages were significantly different from the means of the negative triggered averages. Statistical significance for within Subjects data was based on the outcome of t-tests for correlated data. Using the same procedure, a second difference analysis was then performed upon the averaged data obtained under the stimulus conditions in order to obtain comparative data.

By comparing the amplitude differences between +EEG and -EEG signals with the corresponding differences between the +EP and -EP waveforms, it was possible to detect stimulus modifications of the background alpha activity. For example, in Figure 5 the amplitude difference between the upper pair (non-stimulus, +EEG and -EEG) of signals was compared to the difference between the lower pair of signals (stimulus, +EP and -EP) at each of the indicated sample points. By combining data obtained within and between the four trials approximately 16 pairs of difference scores were obtained for each successive sample point.

Figure 5. The derivation of difference measurements from pairs of averaged phase triggered EEG and EEG plus EP samples. Within each stimulus condition, difference and correlation analyses were performed upon the paired "peak-to-base line" amplitudes of the ±EEG and ±EP averages. For example, the pair of scores obtained from the ±EEG averages at point A consisted of the amplitudes of the +EEG and -EEG waveforms measured with respect to the indicated base line value. Between stimulus condition effects were evaluated by comparing the differences ("peak-to-peak") between the ±EEG averages with the corresponding amplitude differences between the ±EP averages. Within and between trial changes in the ±EEG and ±EP amplitude difference scores, and in the estimated amplitude of the evoked potential were also investigated. The amplitude of the evoked potential component was estimated at point E by deriving the mean (X) of the +EP and -EP averages. By comparing the mean and relative difference scores obtained from the ±EP averages it was also possible to relate the extent of stimulus modification of the background EEG to changes in the amplitude of the EP component.

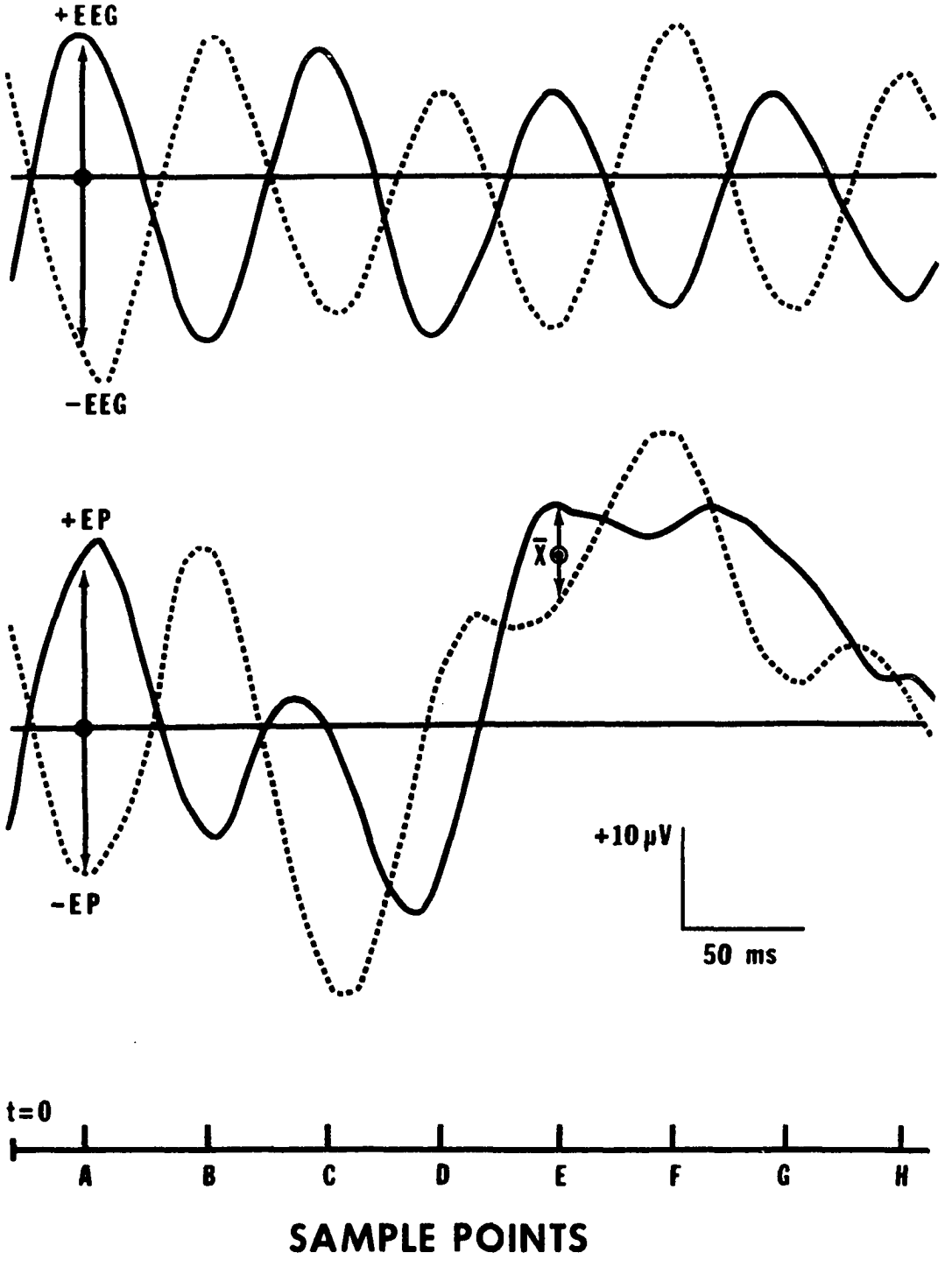

Figure 5. The derivation of difference measurements from pairs of averaged phase triggered EEG and EEG plus EP samples.

For any particular sample point the distributions of ±EEG and ±EP difference scores should have been nearly identical if the phase triggered alpha activity was unaffected by stimulus processes. Significant reductions in the differences between the +EP and -EP averages relative to the differences between the +EEG and -EEG averages could indicate overall amplitude attenuation of the averaged background alpha rhythm, phase shifts, or some combination of these effects. A comparison of the ±EEG and ±EP averages illustrated in Figure 5 clearly reveals a relative reduction in the amplitude difference between the ±EP averages at points E and F.

Systematic EEG and EP Changes

Correlative data. Pearson correlations and regression parameters were also derived from the paired data which were obtained from each of the above difference analyses. Separate correlation analyses were performed for each Subject, EEG recording site, and sample point. The sampling procedures and resulting data (paired observations) for each of the correlation analyses were identical with those used in the difference analyses. There were, therefore, correlation analyses in which the "peak-to-base line" amplitudes (measured as illustrated in Figure 5, point A) of the +EEG and -EEG were compared and a second group of analyses comparing the +EP and -EP amplitudes. The data for a third group of correlation analyses consisted of the "peak-to-peak" amplitudes of the ±EEG and ±EP averages. These three types of correlation analyses correspond respectively to the previously discussed ±EEG, ±EP and ±EEG vs. ±EP difference analyses.

Certain changes in the relationship between the various phase triggered EEG averages should have predictable effects upon the behavior of the correlation coefficients, assuming that these changes could be observed in isolation. Suppose that the overall amplitude of the alpha rhythm changes within and between trials and that these changes also appear in the averaged phase triggered samples of alpha activity (other factors affecting the amplitude of the averaged alpha rhythm are discussed in Chapter 4). If in addition, the amplitude changes were symmetrical, then an increase in the peak to base line amplitude of any peak of the +EEG average should be accompanied by an approximately equal but opposite change in the amplitude of the corresponding peak of the -EEG average. For example, if the first positive peak of the +EEG average were to increase 5 microvolts then the first negative peak of the -EEG average might be expected to decrease 5 microvolts. For this simplified situation an increase in one variable is always accompanied by a comparable decrease in the other variable. Consequently, the correlation coefficient obtained by comparing the +EEG and -EEG amplitudes would be negative. The magnitude of the correlation coefficient would of course depend upon the strength of the linear relationship between the amplitude changes in the phase triggered averages.

The interpretation of correlation coefficients derived from the ±EP averages is more complex. Here, systematic changes in EEG and/or the EP might affect the outcome of the correlation analyses. Furthermore, systematic variation in the background EEG might be due to changes in the ongoing EEG and/or changes unique to the EP sample period (alpha blocking for example). The possibility for interactions among these

three sources of EEG and EP amplitude variation might also be considered. Nevertheless, certain simple relationships among these sources of variability should produce characteristic effects on the correlation coefficient.

Assume for the present that there were no EP amplitude changes but that symmetrical EEG amplitude changes were still present. Under these conditions the correlation coefficients derived from the ±EP average would still be negative. At this point, however, it would not be possible to attribute the underlying systematic variance either to gross changes in the ongoing EEG or to EEG changes unique to the EP sample period. However, if changes in the ±EP difference scores were highly correlated with the corresponding differences between the ±EEG signals, then it might be reasonable to assume that gross changes in the ongoing EEG were influencing both pairs of averages. If the ±EP and ±EEG differences were uncorrelated, the presence of high negative correlations between the +EP and -EP averages might be interpreted as indicating changes in the extent of stimulus modification of the background EEG activity (for example habituation of alpha blocking).

Finally, suppose that there were no changes in the background EEG but that the EP amplitude varied systematically during the data collection trials. Under these conditions the difference between the +EP and -EP averages would remain constant, since these differences are presumably due to the presence of the out of phase EEG components. As the amplitude of the EP component varies, the pair of ±EP signals will be variably displaced above or below the base line. The correlation coefficient obtained by comparing the +EP and -EP amplitudes would be

positive for this set of EEG and EP conditions.

Within and between trial analyses. Additional analyses were performed in order to examine in greater detail the within and between trial variation in the ±EEG and ±EP difference scores and in the amplitude of the evoked potential component of the EEG plus EP averages. It was assumed that systematic variation in the ±EEG difference scores was related to overall changes in the properties of the ongoing EEG. On the other hand, changes in the ±EP difference scores might reflect characteristics of the ongoing EEG and in addition the extent of stimulus modification of the background EEG during the EP sample period. The algebraic means of the combined ±EP averages were used to monitor within and between trial variation in evoked potential amplitude. It is important to emphasize that the means of the EP averages were only estimates of the EP amplitudes since the background EEG activity may have been incompletely cancelled when combining the +EP and -EP averages.

For purposes of the within and between trial analyses the ±EP and ±EEG difference data and EP amplitude estimates were derived only at sample point E. This point was selected on the basis of a preliminary examination of the data which indicated that well developed stimulus modifications of the background alpha activity were likely to be present during this sample period. In addition, there also appeared to be systematic changes in the overall displacements of the +EP and -EP waveforms at this point. This variance was thought to be due to amplitude changes of the constituent large amplitude EP component.

For the within trial analyses the following sampling procedure was used in order to obtain the averaged signals necessary for deriving

the ±EEG difference data and the means and differences of the ±EP averages. First, each trial was subdivided into four to five successive blocks where each block consisted of the samples required to form the averages (N = 16 samples per average) of the ±EEG and ±EP signals. Data obtained from the successive trial blocks were then combined across trials. As a result, for each Subject and recording site the combined +EEG, -EEG, +EP and -EP averages from each successive trial block consisted of N = 64 (4 x 16) samples apiece. These averages were used to derive the ±EP and ±EEG difference scores and to obtain the EP amplitude estimates. The within trial data obtained from the Cz and C3 recording sites of each Subject were then combined across Subjects. For the between trial analyses only data obtained from the first trial block of each trial were used. The Cz and C3 ±EEG and ±EP difference scores and EP amplitude estimates derived from the four successive trials of each Subject were also combined across Subjects. All within and between trial group data were then subjected to analyses of variance and summarized in graphical form.

On the basis of the previously reviewed literature, it was predicted that the extent of the stimulus modification (as evident from the percent change scores) of the phase triggered background alpha activity should become less during the course of data collection. In addition, since the C3 recording site was over the receiving area it was also predicted that there would be more extensive stimulus modification of the background alpha activity recorded from this source as compared to that recorded from Cz. Further, it was predicted that the disparity between the extent of the stimulus modification of Cz and C3 alpha

activity should become greater if these modifications do in fact become localized to the receiving area with successive stimulus presentations. In other words, with repeated stimulation the magnitude of the differences between the ±EP and ±EEG waveforms should diminish more rapidly at the Cz recording site.

CHAPTER III

RESULTS

Because some Subjects became drowsy during one or more of the four experimental trials it was sometimes necessary to terminate these trials prior to obtaining the desired number of averages. Trials were terminated early only when alpha activity was no longer abundant enough to trigger EEG and EP samples. However, it was usually possible to add sufficient samples to one or more subsequent trials in order to obtain at least sixteen averages of N = 16 samples per Subject. These additional samples were acquired in those instances where drowsiness was not evident in the EEG records and where alpha activity was sufficiently abundant to avoid unusually long inter-stimulus intervals. Data collection was limited to three full trials for Subjects C.G. and S.G. due to instrumentation difficulties.

In general, the fixed pre-sample EEG pattern recognition criteria proved satisfactory for obtaining alpha rhythm averages of opposite phase (i.e., the +EEG and -EEG samples) which persisted for several hundred milliseconds. In addition, averages of N = 16 samples were found to produce stable averaged waveform for both the EEG, and EEG plus EP signals for all Subjects. Consequently most of the analyses were based upon data obtained from averages of this size. Examples of the various phase triggered average recorded from the Cz and C3

EEG recording sites of the first two Subjects are illustrated in Figures 6 and 7. These data, which are typical of those obtained from the remaining Subjects, are useful in illustrating certain common characteristics of the various waveforms. In each of these Figures the two phase triggered averages obtained under the stimulus (±EP) and non-stimulus (±EEG) conditions have been superimposed to facilitate comparison of the waveforms. The two pairs of averages on the left-hand column of each figure were recorded from Cz and those in the right-hand columns were recorded from C3. The pairs of waveforms in the upper half of each Figure are the non-stimulus alpha rhythm averages and those in the lower halves are the ±EP waveforms obtained during stimulus trials.

Two characteristics of the non-stimulus alpha rhythm averages are evident in the data presented in Figures 6 and 7. The first of these is the overall damping of the averaged alpha activity from the point of sample initiation to the end of the sample period. The extent of the damping varied considerably between and occasionally within Subjects. The interpretation of this feature of the averaged alpha activity is discussed in Chapter IV. A second characteristic is the amplitude discrepancy between the alpha rhythm averages recorded from the Cz and C3 recording sites. These amplitude differences were sometimes observed in the absence of any appreciable differences between the Cz and C3 EEG amplitude as determined by visual inspection of the EEG ink recordings. It was generally found that the peak to peak amplitudes of the alpha rhythm averages were greater for the EEG source which provided data to the pattern recognition system, irrespective of

Figure 6. Comparisons of the averaged phase triggered EEG samples recorded under non-stimulus and stimulus conditions from the Cz and C3 recording sites of Subject C.G. Cz and C3 data are presented in the left and right columns respectively. Samples which were initiated under the positive trigger condition (solid line) have been superimposed upon those obtained under the negative trigger conditions (broken line). The upper pairs of waveforms are the non-stimulus alpha rhythm averages while the lower pairs were obtained under stimulus condition and therefore contain both EP and EEG components. Subject C.G. had moderately abundant alpha activity and exceptionally large amplitude late EP components which could frequently be detected by visual inspection of the EEG ink records. Pronounced alpha blocking is apparent as a relative reduction in the magnitude of the differences between the upper (EEG) and lower (EEG plus EP) pairs of waveforms.

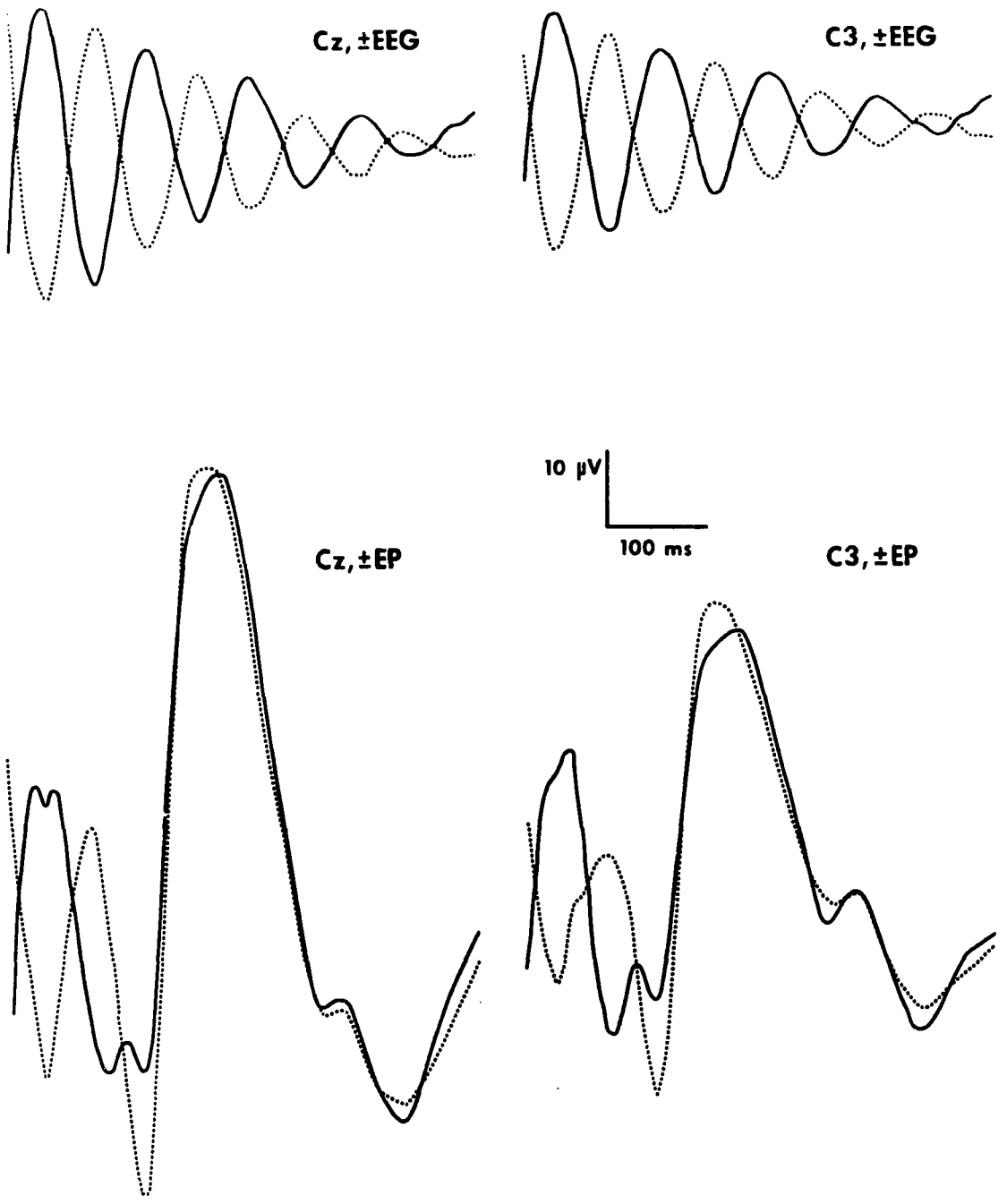

Figure 6. Averaged phase triggered EEG samples recorded under non-stimulus and stimulus conditions from the Cz and C3 recording sites of Subject C.G.

Figure 7. Comparisons of the averaged phase triggered EEG samples recorded under non-stimulus and stimulus conditions from the Cz and C3 recording sites of Subject S.G. The definition and location of the averaged waveforms are the same as those of Subject C.G., Figure 6. S.G. displayed abundant high amplitude alpha activity and less alpha blocking in comparison to C.G. It may be noted that the phase related difference between the Cz averages obtained under stimulus conditions is greater than that between the corresponding non-stimulus EEG averages early in the sample period (i.e., corresponding to point A). A similar slight enhancement of the phase related differences between the ±EP and ±EEG samples was observed in the Cz and C3 data of several Subjects.

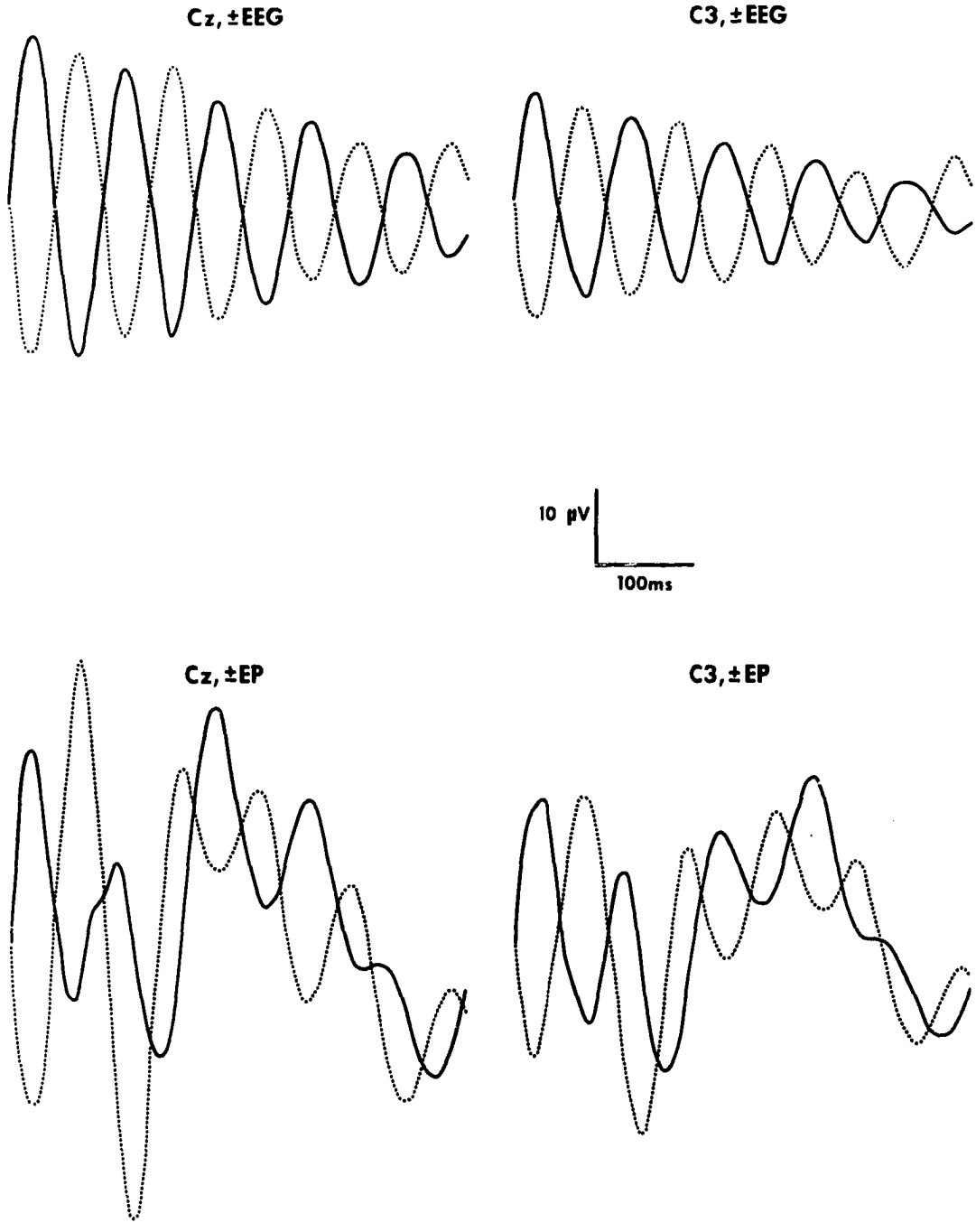

Figure 7. Averaged phase triggered EEG samples recorded under non-stimulus and stimulus conditions from the Cz and C3 recording sites of Subject S.G.

the recording site from which the EEG data were obtained. It is also evident from these illustrations that the non-random alpha activity is a persistent feature of the samples obtained under stimulus (±EP) conditions even though the overall configuration of these waveforms is distorted due to the presence of the evoked potential waveforms. The magnitude of the phase related amplitude difference for late sample points of the phase triggered evoked potential samples is less than the corresponding differences between the averages of the non-stimulus alpha rhythm samples. This relative diminution in the differences between the phase triggered stimulus samples corresponds to the blocking of the background alpha activity during the evoked potential sample period.

Properties of the Averaged Alpha Rhythm

Frequency phase and symmetry parameters were derived from the alpha rhythm averages according to the procedures outlined in Chapter II. These data were found to be useful in examining within and between trial changes in alpha characteristics and in comparing alpha activity derived from the two recording sites. Data presented in Table 2 show that the frequencies (1/C-A, figure 3, Chapter II) of the alpha averages obtained from the two recording sites were similar although there was a tendency for the frequency of the C3 averaged alpha to be slightly faster than than recorded from Cz for Subjects F.G. and O.H. It was also found that the Cz alpha averages tended to lead those recorded from C3, i.e., Cz alpha events (peaks, for example) occurred earlier than those of the C3 alpha averages. These latency differences were occasionally significant within subjects and appeared as a significant between subjects effect as well (Sign test, $p = .035$).

TABLE 2

FREQUENCY AND PHASE PARAMETERS DERIVED FROM THE CZ AND C3 ALPHA RHYTHM AVERAGES

Parameter & Source	Subjects C.G.	S.G.	J.J.	F.G.	B.G.	B.E.	J.G.	O.H.
Frequency (Hz)								
Cz	9.35	10.29	9.26	9.94	11.28	9.90	10.52	11.05
C3	9.40	10.17	9.27	10.31	10.93	10.11	10.44	11.54
Cz-C3	-.05	0.12	-.01	-.37	-.35	-.21	.08	-.49
P	*	*	*	.025	*	*	*	.005
Latency to First Positive Peak (ms)								
Cz	38.23	25.59	31.66	36.43	44.45	29.05	38.05	27.68
C3	40.24	28.74	34.26	39.50	43.76	29.72	28.26	28.45
Cz-C3	-2.01	-3.15	-2.63	-3.07	0.69	-0.67	-0.21	-0.77
P	*.10[a]	.05	.005[a]	.01[a]	*	*	*	*

*statistically non-significant, P > .05

[a]latency comparisons of negative peak were not significant

Inspection of data similar to those presented in Figure 3 suggested that the time required for a transition from a positive to a negative peak of the averaged alpha rhythm was not the same as that required for a negative to positive transition. This waveform asymmetry is responsible for the slight sawtooth appearance of the alpha averages in Figure 3. Such asymmetries were quantified by comparing the first peak to peak transition times of the +EEG averages with the corresponding peak to peak transition times of the -EEG averages. The results are summarized in Table 3 for each recording site. Here it is evident that the waveform distortions due to differential peak to peak transition times are significant or approach significance for several Subjects. These asymmetries do not simply represent phase shifts attributable to different trigger conditions during the averaging process but represent actual distortions of the alpha waves. Given the presence of this type of waveform asymmetries the positive and negative phase triggered alpha rhythm averages would not cancel if added together i.e., there is no phase triggering condition which will yield "equal but opposite" alpha rhythm averages under these conditions. Tukey's (1968) proposed method of extracting the evoked potential by adding (presumably cancelling the EEG) averages obtained against backgrounds of 180 degree out of phase alpha activity, must be considered inaccurate whenever asymmetries in the waveform of the background alpha rhythm are suspected. Nevertheless, Tukey's procedure does provide a convenient means of estimating the EP waveform and was used for this purpose elsewhere in the present investigation.

TABLE 3

WAVEFORM SYMMETRY CHARACTERISTICS OF THE
CZ AND C3 ALPHA RHYTHM AVERAGES

Parameter and Source	Subjects							
	C.G.	S.G.	J.J.	F.G.	B.G.	B.E.	J.G.	O.H.
Cz Symmetry								
+ transition (ms)	52.30	47.43	53.16	46.64	43.22	50.82	51.96	44.23
- transition (ms)	54.61	49.71	52.54	50.99	44.71	49.54	42.88	45.89
Difference (ms)	-2.31	-2.28	0.62	-4.35	-1.49	1.28	9.08	-1.66
P	*.10	.05	*	.01	*	*	.005	*
C3 Symmetry								
+ Transition (ms)	51.75	46.85	53.79	46.81	40.02	50.30	55.30	43.12
- Transition (ms)	54.33	51.16	50.82	48.61	45.54	48.64	41.16	42.95
Difference (ms)	-2.58	-4.31	2.97	-1.80	-5.52	1.66	14.14	0.17
P	.05	.05	*.10	*	*.10	*.10	.005	*

*statistically non-significant, P > .05

Difference Analyses

Sample Points

Sample points were located according to the procedures outlined in Chapter II (Figure 4). Each sample point corresponded approximately to the points of maximum difference between the opposite peaks of the superimposed +EEG and -EEG waveforms. The location of sample points was determined separately for each Subject and recording site. The resulting Cz and C3 sample point latencies in milliseconds are presented in Table 4. Because the location of sample points was dependent upon the frequency of the averaged alpha rhythm, the location of these points differed considerably between Subjects. Therefore, where analyses required the combination of between Subject data the results should be interpreted in terms of the behavior of the averaged waveforms within some time interval rather than at a specific point. Most of the results, however, are based on within Subject analyses.

It may be recalled that there were three difference analyses performed at each successive sample point. The first of these dealt with those within stimulus condition differences between waveforms which occurred as a result of the positive and negative phase triggering conditions. For these analyses the differences between the ±EEG and ±EP averages were separately evaluated. This analysis was necessary for the ±EEG waveforms in order to establish that there were in fact significant differences between the +EEG and -EEG signals as a result of the pre-programmed sampling conditions. A second difference analysis performed upon the ±EP waveforms was done primarily to obtain comparative data.

TABLE 4

CZ AND C3 SAMPLE POINT LATENCIES
IN MILLISECONDS

Subject	EEG Source	Sample Points A	B	C	D	E	F
C.G.	Cz	40.50	93.97	147.17	198.78	251.53	307.22
	C3	42.91	95.28	149.35	200.79	255.30	310.05
S.G.	Cz	27.53	76.18	124.34	173.05	221.35	269.20
	C3	31.60	80.62	128.78	175.36	224.94	270.64
J.J.	Cz	31.33	84.21	138.83	199.00	249.23	299.87
	C3	32.74	85.04	139.24	196.71	247.91	298.14
F.G.	Cz	40.12	88.97	142.93	193.54	246.43	294.56
	C3	42.22	89.83	142.96	192.95	239.94	290.97
B.G.	Cz	45.01	88.58	133.95	193.74	241.14	289.89
	C3	44.88	87.07	135.89	189.54	239.08	288.69
B.E.	Cz	28.56	78.76	128.47	177.60	221.35	264.09
	C3	28.66	78.14	125.95	177.12	218.83	259.54
J.G.	Cz	34.32	81.73	130.47	182.12	231.11	278.79
	C3	33.01	81.27	129.43	179.74	226.69	274.58
O.H.	Cz	28.53	73.58	118.23	162.32	206.06	248.77
	C3	28.52	71.58	114.12	156.69	197.16	238.94

The third difference analysis was concerned with the between stimulus condition differences. The purpose of this analysis was to determine whether or not differences between the non-stimulus ±EEG waveforms were maintained under stimulus conditions. For this analysis the differences between the ±EEG signals were compared with the corresponding differences between the ±EP signals at each sample point.

All statistical computations for the various difference analyses were performed upon data which were measured in arbitrary units. The results of these analyses were then converted to microvolt units through multiplication by appropriate constants. For this reason some slight discrepancies (due to rounding errors) may arise when comparing data contained in the various Tables.

±EEG and ±EP Differences

The summary data from the within stimulus condition difference analyses are presented in Tables 5 through 8. Tables 5 and 6 contain the difference data from the comparisons of the averaged non-stimulus samples of alpha activity recorded from Cz and C3 respectively. The phase related differences between the averaged ±EP samples are presented in Table 7 for the Cz data and in Table 8 for the C3 data. In each of these four tables the first column identifies the Subject from which the data were obtained. The second column labeled "sample size" provides information concerning the total number of difference observations and the number of samples (usually N = 16) used to compute each averaged waveform. For example, the sample size for Subject J.J. is 17N = 16. This indicates that a total of seventeen observations were derived from

TABLE 5

MEAN AMPLITUDE DIFFERENCES IN MICROVOLTS BETWEEN THE POSITIVE AND NEGATIVE TRIGGERED ALPHA RHYTHM AVERAGES RECORDED FROM CZ

Subject	Sample Size	Sample Points A Mean ±S.E.	B Mean ±S.E.	C Mean ±S.E.	D Mean ±S.E.	E Mean ±S.E.	F Mean ±S.E.
C.G.	6N=32	30.35±0.92	-26.90±1.18	20.22±1.29	-14.87±1.68	12.03±1.10	- 6.56±1.34
S.G.	6N=32	33.91±0.90	-33.58±0.97	28.53±0.95	-28.62±0.64	23.23±0.98	-22.53±0.79
J.J.	17N=16	26.37±0.71	-22.14±0.58	15.07±0.72	-10.95±0.76	7.20±0.56	- 5.48±0.51
F.G.	21N=16	19.60±0.79	-18.48±0.67	14.78±0.92	-12.94±0.83	9.78±0.69	- 9.24±1.89
B.G.	16N=16	7.04±0.44	- 6.91±0.83	3.37±0.69	- 4.14±0.62	3.95±0.51	- 3.57±0.73
B.E.	19N=16	22.29±0.66	-19.89±0.91	13.34±0.83	- 7.92±0.91	6.22±1.34	- 3.38±0.96
J.G.	16N=16	9.78±0.74	-12.73±0.70	10.91±0.74	-11.01±0.72	7.76±0.83	- 9.38±0.67
O.H.	20N=16	25.08±0.60	-27.39±0.79	19.82±1.11	-16.13±0.75	11.65±1.20	- 6.60±0.95

All mean differences are significant, $P \leq .05$

TABLE 6

MEAN AMPLITUDE DIFFERENCES IN MICROVOLTS BETWEEN
THE POSITIVE AND NEGATIVE TRIGGERED ALPHA
RHYTHM AVERAGES RECORDED FROM C3

Subject	Sample Size	Sample Points A Mean ±S.E.	B Mean ±S.E.	C Mean ±S.E.	D Mean ±S.E.	E Mean ±S.E.	F Mean ±S.E.
C.G.	6N=32	24.08±0.84	-20.70±1.61	19.03±1.39	-13.76±0.69	10.87±1.39	- 6.53±1.77
S.G.	6N=32	23.65±0.42	-24.12±0.65	18.51±0.76	-19.09±0.82	14.30±0.72	-15.82±1.65
J.J.	17N=16	17.14±0.61	-14.99±0.57	8.76±0.52	- 6.08±0.62	3.54±0.53	- 2.75±0.54
F.G.	21N=16	13.81±0.64	-13.93±0.67	10.25±0.77	- 8.41±0.77	6.29±0.63	- 6.33±0.68
B.G.	16N=16	4.50±0.27	- 4.25±0.57	2.04±0.41	- 1.81±0.37	3.08±0.38	- 1.78±0.45
B.E.	19N=16	26.33±0.71	-23.33±0.65	14.44±0.92	- 8.90±0.91	6.66±1.37	- 3.92±1.02
J.G.	16N=16	18.35±0.83	-24.10±0.74	18.40±0.74	-19.38±0.58	15.48±1.25	-16.98±0.76
O.H.	20N=16	22.36±0.72	-24.64±1.68	18.45±1.03	-13.99±0.73	9.44±0.96	- 5.46±0.79

All mean differences are significant, $P \leq .05$

the various averaged waveforms for statistical analyses and that each averaged waveform represented the mean of 16 successive single EEG samples.

The mean values presented in each table are the average differences between the positive and negative phase triggered averages obtained from all pairs of observations within and between trials. Since the values of the negative triggered signals were always subtracted from those of the positive triggered signals, the signs of the difference observations in each table should be alternately positive and negative as the positive triggered signal takes on values which are greater or less than those of the negative triggered counterpart. This fixed subtraction rule was used in order to detect phase shifts of sufficient magnitude to produce a change in sign between the ±EEG and ±EP difference scores. Phase shifts producing polarity reversals can be seen in the data of Tables 7 and 8 for the Cz sample points E and F and C3 point E of Subject J.G., and in Table 7, point E of Subject B.E. For each of these polarity reversals stimulus modifications of the background EEG were present and of sufficient stability to appear in the combined data obtained from all trials. The nature of these phase shifts will be discussed in greater detail later.

The mean differences reported in Tables 5 through 8 were generally quite significant, particularly for the first few sample points. Within Subject significance levels based on t-tests for correlated data were much less than .05 with few exceptions. These data clearly demonstrate the efficiency of the EEG phase triggering system and the persistence of the non-random characteristics of the background alpha

TABLE 7

MEAN AMPLITUDE DIFFERENCES IN MICROVOLTS
BETWEEN THE +EP AND -EP AVERAGES
RECORDED FROM CZ

Subject	Sample Size	Sample Points					
		A	B	C	D	E	F
		Mean ±S.E.	Mean ±S.E.	Mean ±S.E.	Mean ±S.E.	Mean ±S.E.	Mean ±S.E.
C.G.	6N=32	28.92±1.62	-22.95±2.18	11.12±0.99	- 6.37±1.63	3.43±1.45*	- 1.71±1.26*
S.G.	6N=32	35.52±1.12	-34.57±1.07	32.26±2.51	-23.57±0.80	17.23±1.21	-12.42±1.73
J.J.	17N=16	26.15±0.69	-20.76±0.79	12.04±0.95	- 6.81±1.35	5.82±0.93	- 6.11±0.94
F.G.	21N=16	18.91±0.68	-17.78±0.89	12.57±0.85	- 9.20±0.79	9.39±0.76	- 8.28±0.91
B.G.	16N=16	7.74±0.69	- 7.43±0.65	3.13±0.95	- 3.21±0.88	3.36±0.65	- 3.84±0.77
B.E.	19N=16	24.15±1.13	-20.06±1.27	15.19±0.69	- 9.36±1.34	2.05±1.21*	0.32±1.11*[a]
J.G.	16N=16	7.28±0.83	-11.71±0.96	10.60±1.21	-11.16±1.23	- 2.21±1.20*[a]	0.34±0.97*[a]
O.H.	20N=16	26.34±0.73	-26.35±0.99	20.23±0.83	-19.75±0.84	14.47±1.55	- 8.28±1.02

* Statistically non-significant, P > .05

[a] Polarity reversals relative to non-stimulus EEG differences

TABLE 8

MEAN AMPLITUDE DIFFERENCES IN MICROVOLTS
BETWEEN THE +EP AND -EP AVERAGES
RECORDED FROM C3

Subject	Sample Size	Sample Point A Mean ±S.E.	B Mean ±S.E.	C Mean ±S.E.	D Mean ±S.E.	E Mean ±S.E.	F Mean ±S.E.
C.G.	6N=32	23.81±1.40	-19.89±1.81	9.56±1.28	- 5.16±1.45	3.78±1.34	-2.68±0.75
S.G.	6N=32	26.34±1.45	-25.29±1.24	22.43±2.46	-13.05±3.94	13.37±1.44	-7.94±0.46
J.J.	17N=16	17.94±0.65	-13.60±0.78	6.69±0.92	- 3.18±1.10	2.54±1.02	-3.33±0.81
F.G.	21N=16	13.36±0.64	-13.18±0.74	9.85±0.68	- 7.32±0.58	7.01±0.63	-6.81±0.82
B.G.	16N=16	4.53±0.44	- 4.62±0.50	1.62±0.29	- 0.69±0.80*	2.29±0.44	-2.31±0.56
B.E.	19N=16	27.92±0.96	-23.78±1.10	15.84±0.91	-10.40±1.52	2.40±0.87	-0.18±0.91*
J.G.	16N=16	17.60±0.82	-19.93±0.97	12.80±1.06	- 7.83±0.88	- 0.06±0.81*[a]	-0.86±0.67*
O.H.	20N=16	28.23±0.66	-26.18±0.96	17.88±0.66	-12.65±0.86	9.67±1.16	-4.28±0.83

* Statistically non-significant, P > .05

[a] Polarity reversals relative to non-stimulus EEG differences

activity under stimulus conditions. However, the magnitudes of the ±EP differences relative to the corresponding ±EEG differences also suggested substantial stimulus modifications of the background alpha rhythm. Most frequently there were relative reductions in the differences between the ±EP averages for the late sample points. Occasionally the ±EP differences were greater than those of the ±EEG averages early in the EP sample period.

The overall amplitudes of the Cz ±EEG averages were greater than the corresponding C3 amplitude for the first five Subjects (C.G. through B.G.) while the C3 amplitudes were generally greater for the remaining Subjects (B.E. through O.H.). It may be recalled that for the first five Subjects the Cz recording site also provided EEG data to the pattern recognition system while C3 EEG was used to trigger EEG samples for the last three Subjects. The amplitude differences between the averages obtained from trigger and non-trigger recording sites are largely explained by the statistical properties of the simultaneously recorded signals from each source. Some of the mechanisms governing the amplitude characteristics of the phase triggered EEG averages both within and between recording sites are summarized in more detail in Chapter IV.

±EEG vs. ±EP Differences

The significance of changes in the difference scores obtained from the ±EP stimulus samples relative to the non-stimulus ±EEG averages was evaluated by a procedure similar to that employed for determining the significance of within signal differences. For each sample point the ±EEG difference was subtracted from the ±EP difference this procedure being repeated for the several pairs of ±EEG and ±EP

averages within and between 30-minute trials. Using this procedure, a reduction in ±EP difference relative to ±EEG differences was indicated by a negative score while a relative increase produced a positive score. The resulting distribution of difference scores was tested against the hypothesis that there were no modifications in the phase differences obtained from the ±EEG and ±EP averages, i.e., that the magnitude of the phase-related differences observed in the EEG data was uninfluenced by the presence of EP processes. The resulting mean differences (of differences), t-values (two-tailed t-test for correlated data), and significance levels are presented in Tables 9 and 10 for data recorded from Cz and C3 respectively.

The earliest significant modifications were observed at sample point A and appeared as a relative increase in the ±EP difference data obtained from the C3 lead of Subjects B.E. (latency, 28.5 ms) and O.H. (latency, 28.5 ms). A similar increase in the ±EP amplitude difference approached significance ($P < .10$) in the C3 data of Subject S.G. (31ms) and in the Cz data recorded from B.E. (28ms.). However, the most common features of these data were the significant reductions in the ±EP difference scores late in the EP sample period. Although the relative decreases in ±EP amplitude sometimes failed to reach required significance levels such changes were evident within the first 200 milliseconds in the Cz and C3 data of nearly all of the Subjects. Decrements in the ±EP amplitude difference scores became statistically significant at 81 milliseconds (sample point B) in the C3 data of Subject J.G. and at 90 milliseconds (sample point Cz, Point B) for Subject F.G. However, there was a relative decrease in the difference scores between

TABLE 9

MICROVOLT CHANGES (C) IN THE MEAN ±EP DIFFERENCES
RELATIVE TO THE ±EEG DIFFERENCES
RECORDED FROM Cz

Subject	Sample Size	Sample Points A c	A t	B c	B t	C c	C t	D c	D t	E c	E t	F c	F t
C.G.	6N=32	-1.40	-0.82	-3.95	-1.84	-9.10	-5.52	-8.49	-3.66	-8.59	-4.90	- 4.85	-3.00
S.G.	6N=32	1.61	0.97	0.99	0.95	3.82	1.79	-5.05	-5.35	-5.99	-6.11	-10.10	-5.09
J.J.	17N=16	-0.22	-0.28	-1.38	-1.43	-3.01	-3.21	-4.14	-3.10	-1.37	-1.56	0.62	1.04
F.G.	21N=16	-0.69	-1.10	-0.70	-5.56	-2.29	-1.90[a]	-3.74	-4.26	-0.39	-0.38	- 0.95	-0.67
B.G.	16N=16	0.70	1.23	0.52	0.51	-0.23	-0.20	-0.91	-0.98	-0.59	-0.76	0.27	0.37
B.E.	19N=16	1.86	1.82[a]	0.17	0.10	1.87	1.52	0.95	0.70	-4.17	-2.79	- 3.70	-3.27
J.G.	16N=16	-2.50	-1.74	-1.02	-1.04	-0.31	-0.23	0.15	0.49	-9.98	-6.96	- 9.73	-8.49
O.H.	20N=16	1.26	1.50	-1.04	-0.82	0.40	0.31	3.61	3.07	2.81	1.67	1.67	1.21

Underlined entries are significant; two tailed t-test for correlated data, $P \leq .05$. Note a, $P = .10$

TABLE 10

MICROVOLT CHANGES (C) IN THE MEAN ±EP DIFFERENCES
RELATIVE TO THE ±EEG DIFFERENCES
RECORDED FROM C3

Subject	Sample Size	Sample Points A c	A t	B c	B t	C c	C t	D c	D t	E c	E t	F c	F t
C.G.	6N=32	-0.27	-0.17	-0.81	-1.26	-9.47	-4.59	- 8.60	- 3.44	- 7.09	- 5.38	- 3.85	- 2.22[a]
S.G.	6N=32	2.69	2.04[a]	1.17	1.15	3.92	1.61	- 6.04	- 3.37	- 0.93	- 0.99	- 7.88	- 4.01
J.J.	17N=16	0.80	1.11	-1.39	-1.61	-2.08	-2.29	- 3.11	- 2.69	- 2.17	- 1.06	1.41	0.76
F.G.	21N=16	-0.45	-0.65	-0.75	-0.75	-0.40	-0.42	- 1.52	- 1.80[a]	0.71	0.82	0.34	0.31
B.G.	16N=16	0.00	0.00	0.37	0.54	-0.34	-0.69	- 1.11	- 1.23	- 0.78	- 1.23	0.52	3.94
B.E.	19N=16	1.59	2.34	0.45	0.35	1.39	1.06	1.38	0.88	- 4.26	- 2.77	- 3.73	- 4.98
J.G.	16N=16	-0.75	-0.70	-4.17	-4.13	-5.60	-4.66	-11.55	-10.66	-15.21	-13.06	-16.09	-15.42
O.H.	20N=16	1.87	2.60	- .04	-0.05	-0.57	-0.50	1.71	1.69	0.23	0.17	- 1.18	- 1.08

Underlined entries are significant; two tailed t-test for correlated data, $P \leq .05$. Note a, $P = .10$

sample points A and B for the Cz and C3 data of seven of the eight Subjects. These data suggest that the decrement in the ±EP differences between sample points A and B is occurring more rapidly for the ±EP averages than for the non-stimulus EEG averages. The data also suggest that the onset of processes which produce the relative greater attenuation in the phase related differences of the ±EP averages probably occurs somewhere between (or prior to) sample points A and B, i.e., somewhere between 35 and 83 milliseconds based on the group mean sample point latencies. Significant modifications may have occurred prior to the first sample point but the sample point selection procedures did not provide sufficient resolution for a precise estimate of the point of onset of these processes.

Because the overall amplitudes of the averaged signals differed among Subjects and between recording sites within any particular Subject, the relative changes in ±EP averages of Tables 9 and 10 were also expressed as percent change scores relative to the corresponding ±EEG values. These data are presented in Figure 8. The percentage conversions provide a convenient means of visualizing the relative changes as a function of the sample point and recording site of each Subject.

In Figure 8 the graphs of Subjects B.E. and J.G. indicate relative reductions in background alpha below minus 100 percent. These points correspond to reversals in the relationships between the positive and negative phase trigger samples of the ±EP averages relative to those between the corresponding ±EEG waveforms. For example, at point E the alpha averages have completed two complete cycles so that the initially

Figure 8. Percent change in ±EP difference scores relative to ±EEG difference scores as a function of sample point location. Percent change data were computed by expressing the relative change between the ±EP and ±EEG difference as a percentage deviation from the ±EEG difference scores. Relative increases in the amplitude differences between the ±EP averages are indicated by points above the abscissa. Points below the abscissa indicate relative decreases in the ±EP difference scores. Percent change data were obtained separately for each Subject, sample point, and recording site. For each Subject the solid line graphs represent the Cz data and the broken lines C3 data. The arrows located at various points along the graphs identify the points at which the discrepancies between the ±EEG and ±EP difference scores were significant. The time axis of each graph begins at the point of sample initiation. The full duration of the illustrated time scales is 300 milliseconds. Successive sample points are identified above the abscissa of each graph (opposite the +25% point along the ordinate). Although individual differences are great, the tendency toward a relative reduction in the ±EP difference scores produced a significant ($P \leq .05$ Wilcoxon signed-ranks test) between Subjects effect at sample point E for both the Cz and C3 averages.

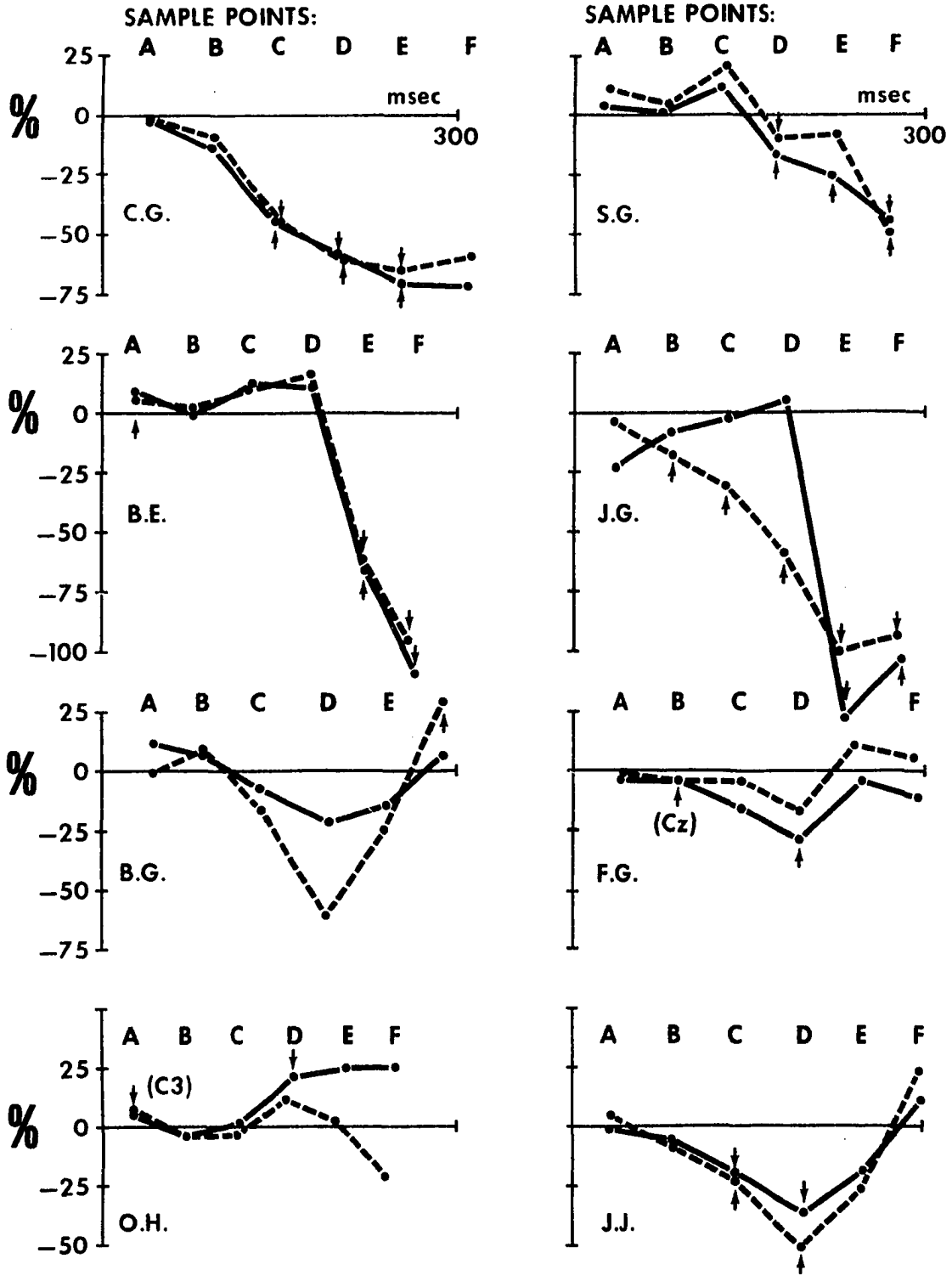

Figure 8. Percent change in ±EP differences relative to ±EEG differences as a function of sample point location.

positive alpha rhythm waveform is again positive in value while that of the initially negative average again becomes negative in value. By using the subtraction procedure in which the value of the initially negative (-EEG) average is subtracted from the value of the initially positive (+EEG) signal, the resulting difference score at point E will be a positive number.

Phase shifts between the ±EEG and ±EP averages would also affect the percent change amplitude scores. Suppose that the peaks of the background alpha activity of the ±EP averages at point E were shifted more than 90 but less than 270 degrees relative to the corresponding peaks of the non-stimulus EEG averages. Under these conditions it would be found that the relative location of the initially positive and negative waveforms has been reversed i.e., the EP waveform would be above rather than below the EP waveform. By using the subtraction rule in which the value of the negative phase triggered signal is always subtracted from that of the positive triggered signal it would be found that the difference between the ±EP averages would now be a negative number at point E while the difference between the ±EEG averages would be a positive number. During the computation of the percent decrement scores of Figure 8, such discrepancies in sign between the ±EEG and ±EP difference measurements yielded a numerator of greater absolute value than the denominator since the difference scores were in effect added in the subtraction process. In these instances the indicated percent change was below minus 100.

The results presented in Figure 8 show that the adopted scoring technique was sensitive to polarity reversals between difference scores

due to phase shifts and that such reversals occurred with sufficient regularity to permit their detection in the grand means of the averaged waveforms of certain Subjects. This sampling procedure is however misleading since phase shifts would not necessarily indicate the presence of reductions in the overall amplitude of the background alpha activity.

The presence or absence of stable phase shifts between the ±EEG and ±EP averages was further examined by removing the EP waveform from the ±EP signals and comparing the residual EEG activity directly with the ±EEG averages obtained under the non-stimulus conditions (Figure 9 and 10). This was accomplished by subtracting the entire negative triggered waveform from the positively triggered counterpart. The +EEG and -EEG activity which was nearly 180 degrees out of phase was in effect added during this subtraction process and the evoked potential waveforms which were presumably in phase under each trigger condition were cancelled. The waveforms which were obtained from the ±EEG and ±EP averages through this procedure are essentially a continuous representation of the discrete sample point data presented earlier. If there were no interactions between the EP and EEG processes the resulting difference waveforms should be nearly identical. It must be pointed out that any discrepancies between the difference waveforms could be attributed to EEG and/or EP modifications. In spite of this ambiguity the procedure proved useful in comparing the differences between the ±EEG and ±EP averages.

The difference waveforms which are illustrated in Figures 9 and 10 were derived from the ±EEG and ±EP averages which were previously illustrated in Figures 6 and 7. The location of the successive positive

Figure 9. Difference waveforms derived by subtraction of the phase triggered averages obtained from Subject C.G. The pairs of waveforms presented to the left and right represent Cz and C3 data respectively. The solid line curves were derived by subtracting the entire -EEG averages from the corresponding +EEG averages. The dotted waveforms were obtained by subtracting the -EP averages from the +EP averages (presumably cancelling the in-phase EP activity). Relative reductions in the amplitudes of the ±EP difference waveforms are accompanied by phase shifts late in the sample period.

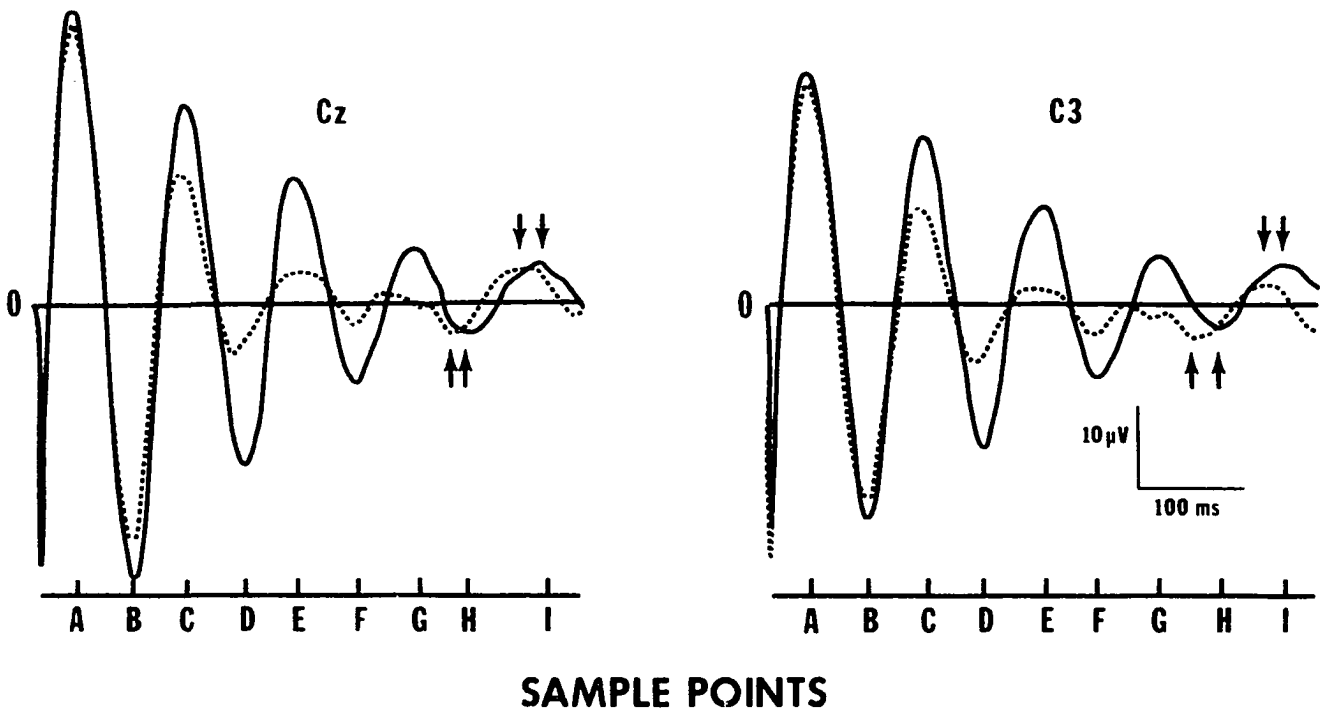

Figure 9. Difference waveforms derived by subtraction of the phase triggered averages obtained from Subject C.G.

Figure 10. Difference waveforms derived by subtraction of the phase triggered averages obtained from Subject S.G. The derivation of the various difference waveforms was explained in Figure 9. The data obtained from S.G. reveals a relative increase in the amplitudes of the ±EP difference waveforms early in the sample period. Where present, relative reductions in the amplitude differences between the ±EP averages are less pronounced than those of Subject C.G. The phase shifts which were evident in Figure 9 are less pronounced or absent in the S.G. data.

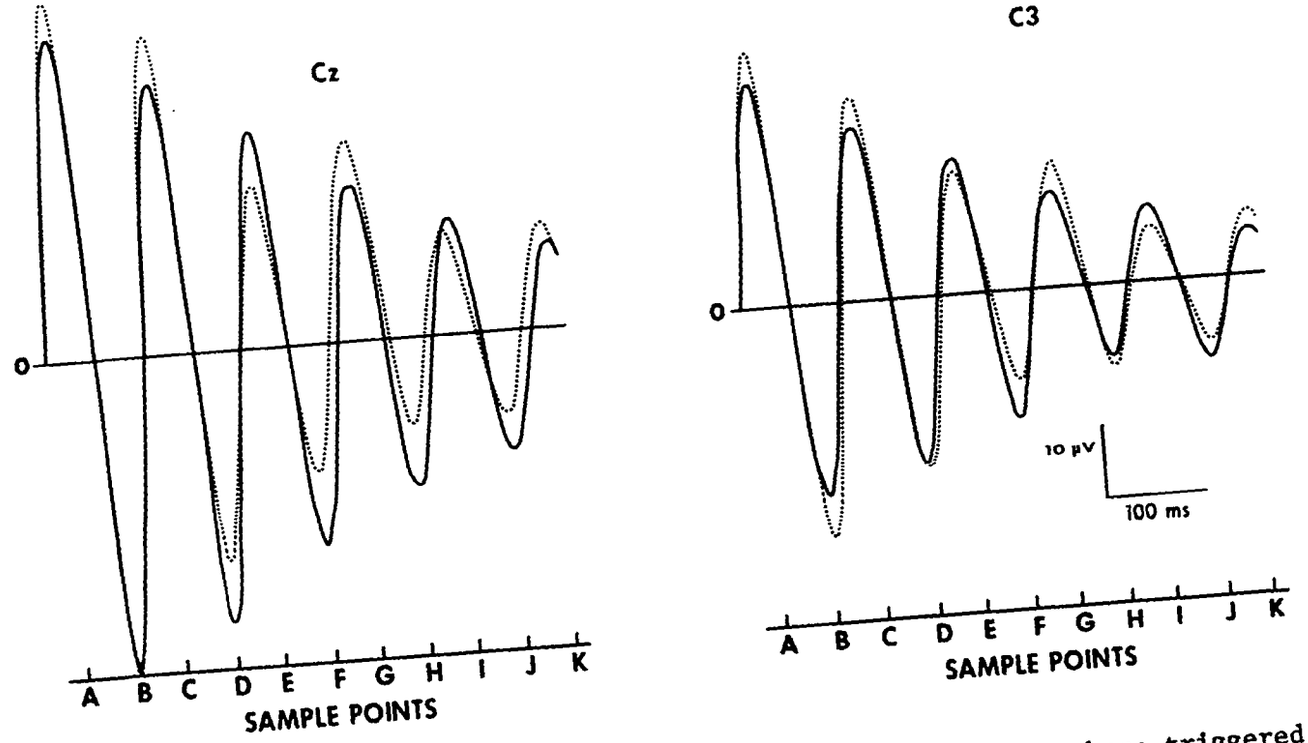

Figure 10. Difference waveforms derived by subtraction of the phase triggered averages obtained from Subject S.G.

and negative peaks of the solid curves (derived from the ± EEG averages) correspond approximately to the sample points which were defined in Table 4. Similarly the percent change in the amplitude of the dotted (± EP difference curve) tracing relative to the amplitude of the ± EEG difference waveform should approximate the values presented in Tables 9 and 10. There may be discrepancies between estimates derived from these difference curves and the tabulated data since the averages from which Figures 9 and 10 were derived represent only part of the total data for each Subject.

Phase shifts (or perhaps changes in the frequency of the background alpha activity) are evident in Figure 9 as discrepancies between the peak latencies of the ±EEG and ±EP difference curves. For example at sample points H and I of the Cz and C3 difference curve, the points of maximum difference between the ±EP averages occurred 13-26 milliseconds earlier than those of the ±EEG averages. These phase discrepancies are indicated by the arrows above and below the difference curve. In Figure 9, a recovery in the amplitudes of the ±EP difference curves relative to those of the ±EEG averages is also evident toward the end of the 512 millisecond sample period. At sample point G, for example, the amplitude of the Cz ±EP difference curve is very nearly zero, or at least, considerably reduced relative to the amplitude of the ±EEG difference waveforms. In contrast the amplitude of the ±EP difference curve at sample points H and I is almost equal to the amplitude of the ±EEG curve, although there is a phase shift between two waveforms. Very little amplitude attenuation or phase difference is apparent in the Cz and C3 difference curves of S.G. in Figure 10. The relative enhancement in the

amplitude of the ±EP difference curve is consistent with the S.G. data presented in Tables 9 and 10 and in Figure 8.

An examination of the difference curve data obtained from each of the Subjects revealed that phase shifts may occur earlier than 100 milliseconds following stimulation but that such shifts became most evident during the 100 to 300 milliseconds interval corresponding to the period of maximum alpha blocking. The available data also suggested that some stabilization of the ±EP, ±EEG phase difference developed as a gradual recovery in amplitude of the ±EP difference waveforms occurred although the recovering background alpha activity appeared to remain slightly out of phase with respect to that of the non-stimulus EEG averages. By the end of a 512 ms sampling period the peaks of the ±EP difference curves occurred earlier than those of the ±EEG waveforms in five out of eight Subjects. Within Subjects, phase shifts in the Cz data were accompanied by similar (in direction) phase shifts between the C3 waveforms. The direction of phase shifts seemed to be stable within Subjects although there was no consistent pattern between Subjects. However, generally inadequate data and the use of relatively short sample periods prohibited a detailed statistical analysis of the phase-amplitude recovery of alpha activity following the period of maximum alpha blocking. It should also be pointed out that amplitude changes or phase shifts between the ±EP and ±EEG difference waveforms might be due to changes in the background alpha activity affecting both trigger conditions or to differential changes between the background activities associated with each of the trigger conditions.

Systematic ±EEG and ±EP Variation

Correlative Data

Three Pearson correlation analyses were performed upon the ±EEG and ±EP data. Each of the three analyses was carried out separately for each Subject, recording site and sample point. The data for the first analysis consisted of the peak to base line amplitudes of successive pairs of +EEG and -EEG averages. Each pair of averages (and each pair of observations) was obtained during the same time period of a given trial. There were generally four to five pairs of ±EEG averages obtained during each of four data collection trials. For example, the +EEG and -EEG amplitudes measured at point A in Figure 5 represent the first pair of C3 observations for this sample point from trial 1. There were three additional pairs of point A, ±EEG observations for the C3 recording site obtained from data collected during the remainder of the first trial. These data, combined with similar pairs of C3 ±EEG amplitude measures obtained during the last three trials, provided the numerical data for one of the ±EEG correlation analyses. This within and between trial sampling procedure was then repeated for successively later sample points within the C3 ±EEG averages and separately for the Cz ±EEG averages of the same Subject. By this procedure, ±EEG amplitude data were obtained from all Subjects. A second correlation analysis was then performed upon the +EP and -EP averages under sampling conditions identical to those of the first analyses. Finally, a third correlation analysis was carried out by comparing the pairs ±EEG and ±EP peak-to-peak difference scores derived from successive time blocks within and between the data collection

trials. The paired data used for each of the correlation analyses were therefore the same as those employed for the difference analyses.

The results of the correlation analyses performed upon the ±EEG data are summarized in Table 11. These data suggest that there was little if any linear relationship between changes in the positive and negative triggered ±EEG averages from the beginning of the first to the end of the last recorded trial. This could mean that there was little systematic variance in the overall amplitudes of the ±EEG averages or that the relationships which were present were more complex in form. There is also the possibility that the low variance, i.e., restricted range in the EEG averages contributed to the low correlation values. The negative signs frequently associated with the ±EEG coefficients probably imply that those EEG amplitude changes which were present produced opposite effects on the positive and negative peaks of the alpha averages at the selected sample points. For example, an overall increase in the amplitude of the EEG average was probably due to an increase in the positive component and a corresponding decrease (increased negativity) in the amplitude of the negative peak. In contrast to the ±EEG correlation coefficients, those derived from ±EP averages (Table 12) were often moderately high and nearly always positive beyond the first sample point. The observation that the ±EP correlation coefficients attained their maximum values during late sample points is consistent with the suggestion (Chapter II) that the underlying systematic variance may be related to changes in the amplitude of the EP. The data presented in Table 13 show that there was a moderate linear relationship between the changes in the difference scores

TABLE 11

CORRELATIONS BETWEEN THE PEAK TO BASELINE AMPLITUDES
OF THE +EEG AND -EEG AVERAGES
RECORDED FROM
CZ AND C3

Subject, (Sample Size)	EEG Source	Sample Points A	B	C	D	E	F
C.G.	Cz	.214	-.420	-.295	-.405	-.180	-.456
(6N=32)	C3	-.037	.481	-.300	-.031	-.106	-.098
S.G.	Cz	.150	-.350	.331	-.360	-.010	-.724
(6N=32)	C3	-.632	.481	.356	-.031	-.600	-.158
J.J.	Cz	-.068	-.404	.060	-.000	<u>-.489</u>	<u>-.584</u>
(17N=16)	C3	-.340	.124	<u>-.495</u>	-.186	<u>-.506</u>	<u>-.491</u>
F.G.	Cz	-.043	-.131	-.044	-.120	-.111	-.120
(21N=16)	C3	-.045	-.020	.043	.401	-.093	-.142
B.G.	Cz	.492	-.081	.114	.267	-.302	.103
(16N=16)	C3	.488	.024	.225	-.173	-.160	.288
B.E.	Cz	<u>-.624</u>	-.010	-.254	-.271	-.057	-.000
(19N=16)	C3	-.130	.000	-.180	-.173	-.094	.043
J.G.	Cz	-.228	.375	.234	.248	-.117	-.328
(16N=16)	C3	.347	-.056	.414	-.167	.257	-.292
O.H.	Cz	<u>.467</u>	-.398	.017	-.180	.040	-.045
(20N=16)	C3	.208	.037	.000	-.064	.020	-.138

Underlined entries are statistically significant, $P \leq .05$

TABLE 12

CORRELATIONS BETWEEN THE PEAK TO BASELINE AMPLITUDES
OF THE +EP AND -EP AVERAGES
RECORDED FROM
CZ AND C3

Subject, (Sample Size)	EEG Source	Sample Points A	B	C	D	E	F
C.G.	Cz	.034	.062	.878	.901	.922	.486
(6N=32)	C3	-.088	.251	.673	.805	.790	.850
S.G.	Cz	-.406	.493	.676	.740	.801	.872
(6N=32)	C3	.203	.127	-.038	-.317	.621	.968
J.J.	Cz	-.066	.448	.267	.413	.381	.161
(17N=16)	C3	-.060	.433	.041	.381	.219	.223
F.G.	Cz	-.022	.520	.590	.242	.245	.501
(21N=16)	C3	.202	.617	.607	-.130	.566	.422
B.G.	Cz	-.050	.494	.453	.608	.830	.505
16N=16)	C3	-.046	.733	.883	.654	.820	.712
B.E.	Cz	-.148	.121	.501	.676	.017	.401
(19N=16)	C3	.028	-.088	.081	.534	-.358	.539
J.G.	Cz	-.487	.010	.647	.590	.616	.458
(16N=16)	C3	-.363	.114	.222	.630	.671	.488
O.H.	Cz	-.222	.460	.362	.698	.328	.649
(20N=16)	C3	.150	.083	.211	.604	.123	.632

Underlined entries are statistically significant, $P \leq .05$

TABLE 13

CORRELATIONS BETWEEN THE ±EEG AND ±EP DIFFERENCE SCORES DERIVED FROM THE CZ AND C3 AVERAGES

Subject, (Sample Size)	EEG Source	Sample Points A	B	C	D	E	F
C.G.	Cz	+.187	+.453	-.338	+.470	+.207	+.692
(6N=32)	C3	+.185	+.568	-.426	+.238	+.596	+.476
S.G.	Cz	+.354	+.613	-.521	-.421	-.626	+.330
(6N=32)	C3	+.458	+.579	-.304	<u>+.879</u>	-.718	-.038
J.J.	Cz	+.475	+.362	+.404	+.309	<u>+.559</u>	<u>+.810</u>
(17N=16)	C3	<u>+.600</u>	+.221	<u>+.511</u>	+.318	<u>+.555</u>	<u>+.565</u>
F.G.	Cz	<u>+.679</u>	+.417	+.101	<u>+.504</u>	-.120	+.146
(21N=16)	C3	<u>+.469</u>	<u>+.497</u>	+.141	+.328	+.170	-.061
B.G.	Cz	+.244	+.211	-.063	+.265	+.338	<u>+.520</u>
(16N=16)	C3	-.186	+.214	-.040	+.058	+.022	+.253
B.E.	Cz	<u>+.692</u>	-.033	+.034	+.380	+.366	+.415
(19N=16)	C3	<u>+.724</u>	+.166	+.174	+.319	+.403	<u>+.710</u>
J.G.	Cz	+.429	-.354	+.111	-.202	-.116	+.299
(16N=16)	C3	<u>+.499</u>	+.149	+.154	-.083	-.041	+.317
O.H.	Cz	+.180	+.337	+.155	-.056	-.282	-.079
(20N=16)	C3	<u>+.470</u>	+.230	+.163	-.231	-.224	+.223

Underlined entries are statistically significant, $P \leq .05$

obtained from the stimulus and non-stimulus averages, in particular for the first sample point. The frequent positive correlations suggest that there are changes in the characteristics of the ongoing EEG which are common to the averaged ±EEG and ±EP data.

Within vs. Between Trial Variation

Further analyses were performed on the ±EEG and ±EP averages at sample point E in order to more closely examine the systematic changes underlying the significant correlations of Tables 11, 12 and 13. This sample point was selected since it had already been established that significant alpha blocking was present during this period and because inspection of the ±EP averages revealed the presence of systematic changes in EP amplitudes as well. For purposes of the present analyses Subjects C.G. and S.G. were excluded due to insufficient data. In addition, sample point F of Subject O.H. was substituted for point E in order to produce a narrower range of sample point latencies between Subjects. The resulting sample interval for the entire group of Subjects was approximately 30 milliseconds in width with a latency range of approximately 220 to 250 milliseconds. Both within and between trial analyses of variance were performed upon the combined data of all Subjects. Unless otherwise specified, a conservative test (Winer, 1962) was used in determining the critical values for the F ratios.

Within trial changes in the difference scores derived from the ±EEG and ±EP averages are illustrated in Figure 11. These graphical data suggest that the peak to peak amplitude of the alpha averages measured at point E tended to decrease with successive samples within the trials and that the general time course of this EEG amplitude change

Figure 11. Within trial changes in the ±EEG and ±EP difference scores. The derivation of data points is explained in the text. Note that there are overall reductions in the amplitude differences between the ±EEG averages and between the ±EP averages. These data suggest that there were within-trial changes in EEG characteristics which had a common affect upon the ±EP and the ±EEG averages.

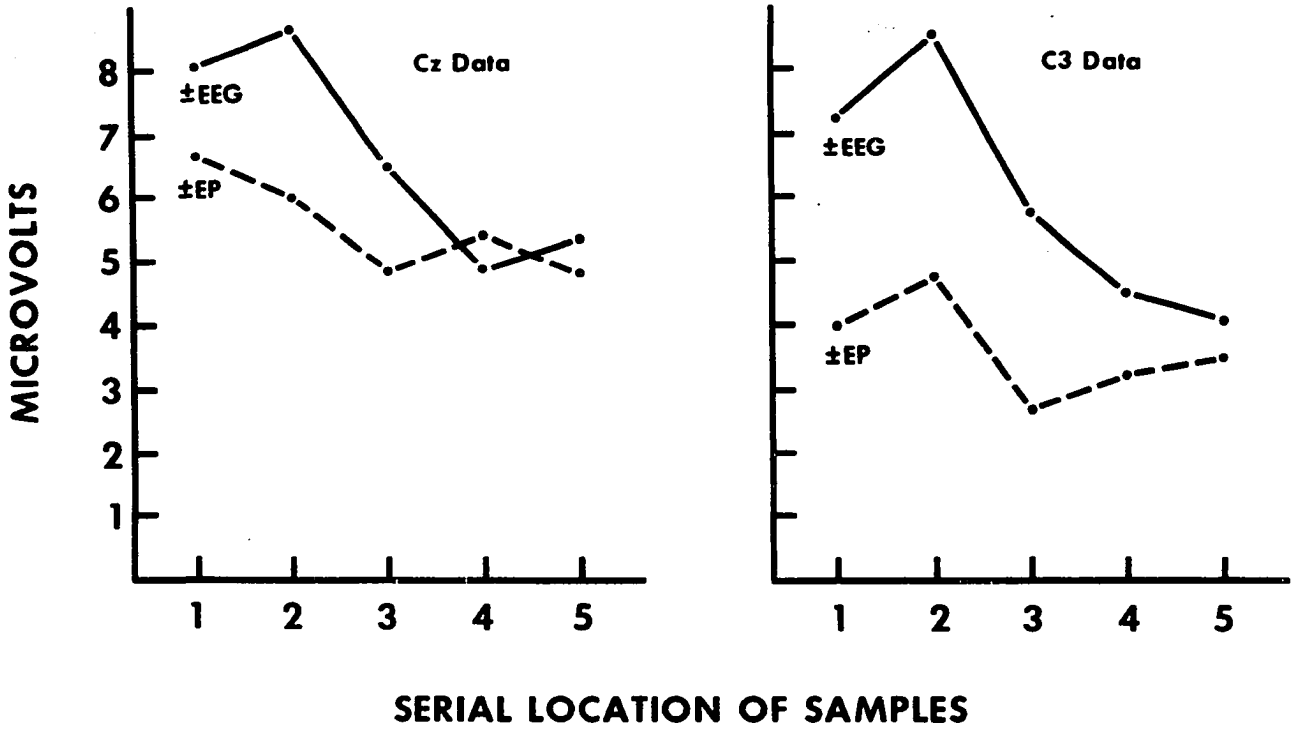

Figure 11. Within trial changes in the ±EEG and ±EP difference scores.

was similar for the Cz and C3 recording sites. The corresponding amplitude differences between the ±EP averages were less than those obtained from the non-stimulus ±EEG averages due to the presence of alpha blocking during stimulus trials. The amplitude differences between the ±EP averages also became less during the course of the trials, but at a slower rate. The net effect was a slight convergence between the ±EEG and ±EP difference scores which suggests that alpha blocking was less pronounced at the end of the trial. These changes in alpha blocking are more easily visualized by expressing the differences between the ±EP signals as a percentage of the corresponding ±EEG differences. The resulting percent data are illustrated in Figure 12 where the lessening effect of alpha blocking is evident. Note in addition that alpha blocking was much more pronounced in samples obtained from the C3 recording site, thus supporting the proposition that differential blocking may occur between recording sites. In this instance the extent of background EEG modification may be greater near the presumed source of the EP, i.e., the sensory receiving area which for Median Nerve stimulation is directly beneath the C3 recording position.

Analyses of variance performed upon the data presented in Figure 11 and 12 indicated that the Cz and C3, ±EEG amplitude difference data were not significantly different. The overall decrease in the amplitude difference between the ±EEG averages (from both recording sites) approached significance ($F = 2.69$, $P < .25$, usual test, $P < .10$) suggesting the presence of gross changes in the ongoing EEG within the data collection trials. There was a significant difference between the Cz and C3, ±EP difference scores (C3 < Cz; $F = 22.5$, $P < .01$). Since no significant

Figure 12. Within trial changes in the relationship between the ±EP and ±EEG difference scores. A comparison of the Cz and C3 graphs suggests that the extent of stimulus modification of the background EEG is greater for averages recorded from C3. The within-trial increase in the percentage scores suggests that the discrepancy between the ±EP and ±EEG difference scores is reduced for samples taken late in the data collection trials.

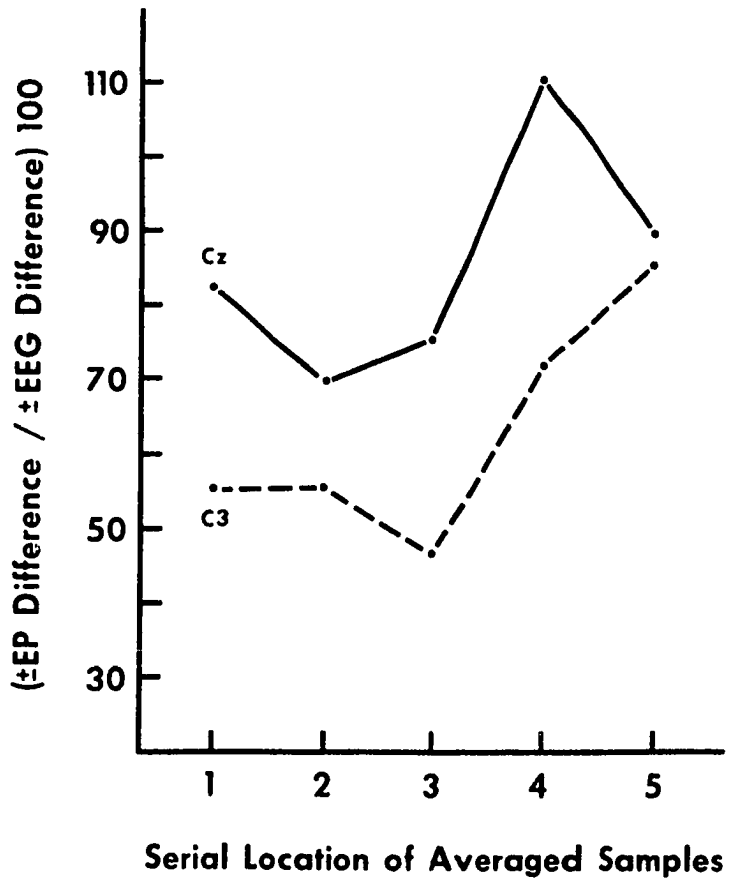

Figure 12. Within trial changes in the relationship between the ±EP and ±EEG difference scores.

differences were found between the Cz and C3 ±EEG difference scores, the relatively smaller C3 ±EP differences probably represent more extensive modification of the background alpha rhythm near the somatosensory receiving area (C3). The overall difference between the combined Cz and C3 ±EEG vs. ±EP data was not significant ($F = 2.16$, $P < .25$) but the presence of an interaction between successive samples and the extent of the differences between the ±EEG and ±EP difference data was suggested ($F = 2.77$, $P < .10$). This interaction appears in Figure 11 as a convergence between the Cz and C3 EEG and EP difference curves, and in Figure 12, as an upward trend in the Cz and C3 percent change graphs. However, there was no convincing evidence for differential rates of recovery in the amplitude of the background EEG between the Cz and C3 recording sites.

Systematic changes in the ±EEG and ±EP averages also occurred from the beginning of one trial to the next as illustrated in Figure 13. The between trials increase in the amplitudes of the non-stimulus alpha rhythm averages paralleled a similar amplitude increase observed in the paper recordings of the EEG. Such changes in alpha abundance seemed to be relatively common as the Subjects became accustomed to the experimental routine. The graphical data of Figure 13 also suggest a close parallel between changes in the ±EEG and ±EP difference scores obtained from Cz. By contrast, the extent of alpha blocking in the ±EP samples recorded from C3 remained nearly constant from the beginning of one trial to the next, even though the overall amplitude of the non-stimulus C3 alpha rhythm averages increased. The between trial ±EP differences expressed as a percent of the corresponding ±EEG differences are presented in

Figure 13. Between trial changes in the ±EEG and ±EP difference scores. Each data point represents the group mean of the amplitude differences between the first set of averages obtained during each trial. A between-trial increase in the amplitude difference between the ±EEG averages is evident.

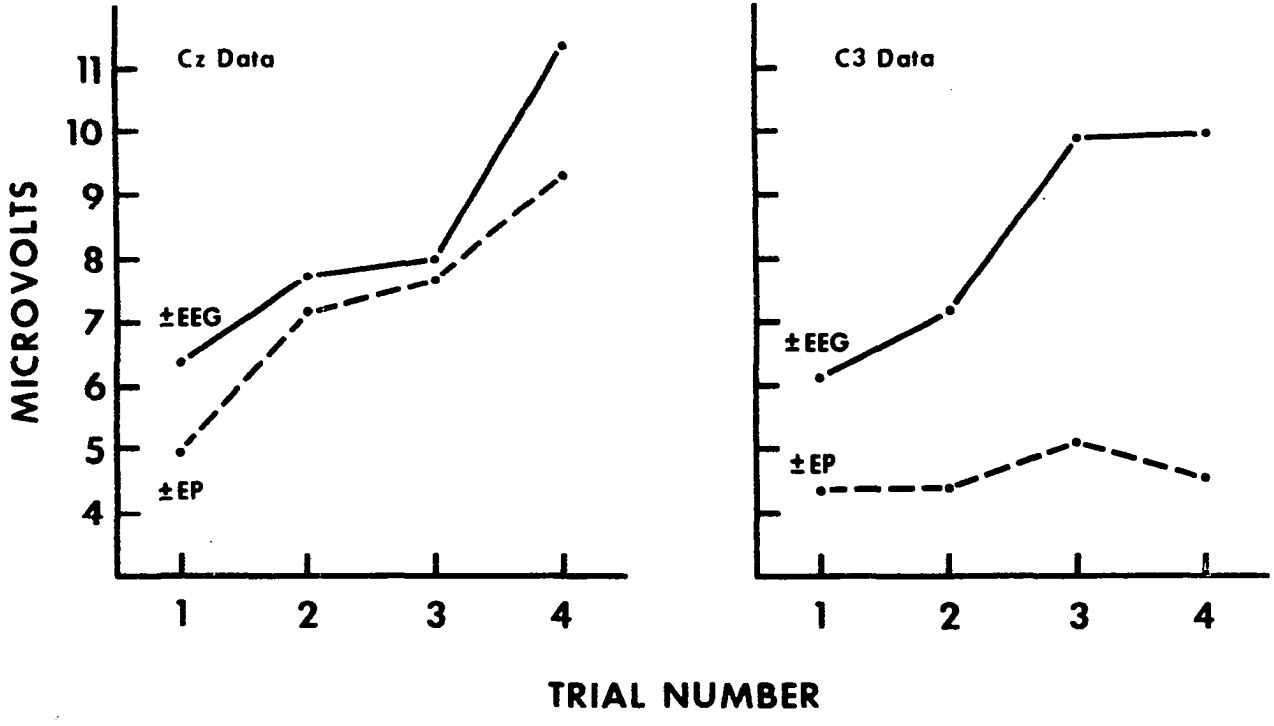

Figure 13. Between trial changes in the ±EEG and ±EP difference scores.

Figure 14. Again the overall differences between the Cz and C3 difference scores are evident. Note, however, that the 20 percent decrement in the C3 curve reflects the divergence between the ±EEG and EP difference data presented in Figure 13 rather than an actual decrease in the EP difference score alone.

The analyses of variance performed upon the data presented in Figure 13 showed that there was no significant difference between the Cz and C3 ±EEG curves although there was a tendency for a EEG trigger site by recording site interaction ($F = 4.65$, $P < .10$). The latter might have been anticipated on the basis of earlier results which showed that the amplitudes of the ±EEG averages were typically larger for data recorded from the source of the trigger EEG independent of recording site. There was a significant difference between the Cz and C3 ±EP difference data ($F = 23.7$, $P < .01$) as well as a possible trials by recording site interaction ($F = 3.55$, $P < .25$, usual test, $P < .05$). The main effect of signal type, i.e., ±EEG vs. ±EP over trials and recording site, approached significance ($F = 5.27$, $P < .10$) but the main effects of trials ($F = 2.14$, $P < .25$) and recording site ($F = 2.3$, $P < .25$) were not significant.

Each of the above within and between trial analyses was concerned with changes in the amplitude of the EEG. The within and between trial changes in the amplitude of the EP were also examined. For this purpose the amplitude of the EP component of the ±EP averages (the same averages used for the within and between trial difference analyses) was estimated by taking the algebraic mean of the appropriate +EP and -EP averages at point E. As earlier results have shown, there

Figure 14. Between trial changes in the relationship between the ±EP and ±EEG difference scores. The relationship between the Cz ±EP and ±EEG difference scores remains more or less constant even though there may be overall between-trial changes in EEG characteristics. The general downward trend in the C3 graph reflects the apparent interaction between the ±EEG and ±EP graphs presented in Figure 13.

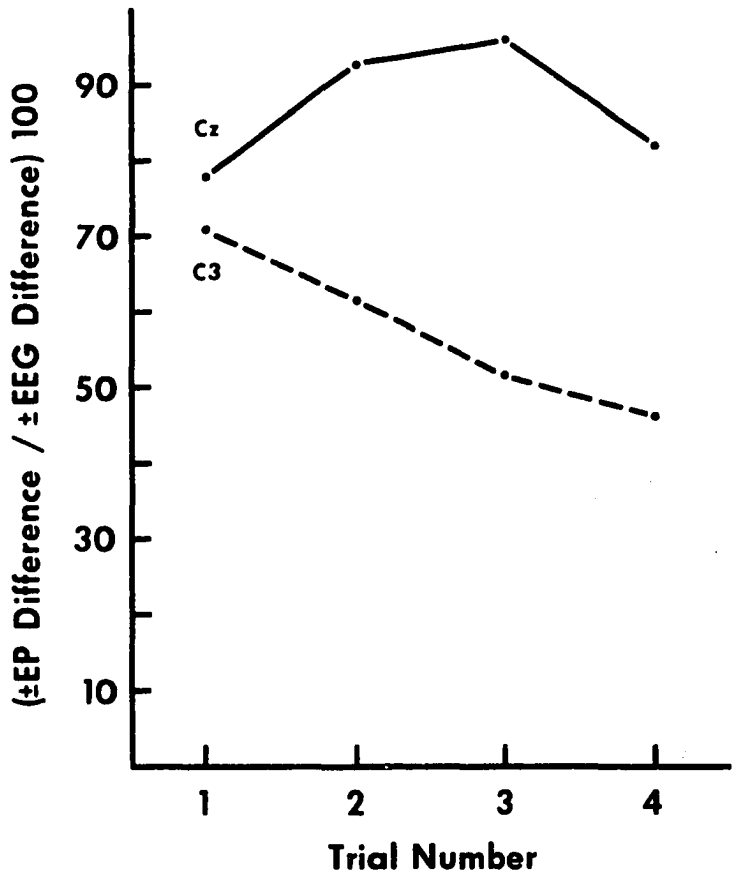

Figure 14. Between trial changes in the relationship between the ±EP and ±EEG difference scores.

is likely to be an error associated with this method of extracting the EP from non-random EEG background activity due to waveform asymmetries or to differential phase shifts between the two alpha components (i.e., even though symmetrical, the background alpha components may no longer be 180° out of phase due to the alpha blocking). Nevertheless, it was felt that changes in the means of the ±EP averages were probably faithful representations of amplitude changes in the EP waveforms. There were two reasons for believing the EP amplitude estimates were reliable. First, the amplitude differences between the ±EP averages attributable to the phase triggering were typically small at this sample point due to the presence of alpha blocking. Under these circumstances it seemed reasonable to suppose that the magnitude of the error resulting from cancellation of the background EEG was also small. A second consideration was that the changes in the amplitude of the estimated EP component were quite large relative to the amplitude differences between the ±EP averages.

Within and between trial changes in EP amplitude expressed as a percent of the initial value are illustrated in Figures 15 and 16. The within and between trial amplitude decrements were quite extensive for both Cz and C3 recording sites. The effects of these systematic EP amplitude changes upon the ±EP waveforms probably accounts for the significant correlations presented in Table 12. Separate analyses of variance were performed on the within and between trial EP amplitude data and upon the percent change data presented in Figures 15 and 16.

The analysis of variance upon the within trial EP amplitude data revealed that the amplitude of the Cz EP was significantly ($F = 26.2$,

Figure 15. Within trial changes in evoked potential amplitude. Estimates of the amplitudes of the Cz and C3 evoked potentials were derived at sample point E by averaging together the +EP and -EP averages obtained from the same time period. Five successive EP amplitude estimates were thus obtained from each trial. This procedure was repeated for each trial, recording site and Subject. The resulting EP amplitude data were then combined across trials and Subjects yielding five successive group mean amplitude estimates for the Cz and C3 evoked potentials. Finally each of these mean amplitudes were expressed as percentages of the group mean EP amplitude obtained from the first time period. These initial EP amplitude estimates were 26.35 μV for Cz and 12.56 μV for C3.

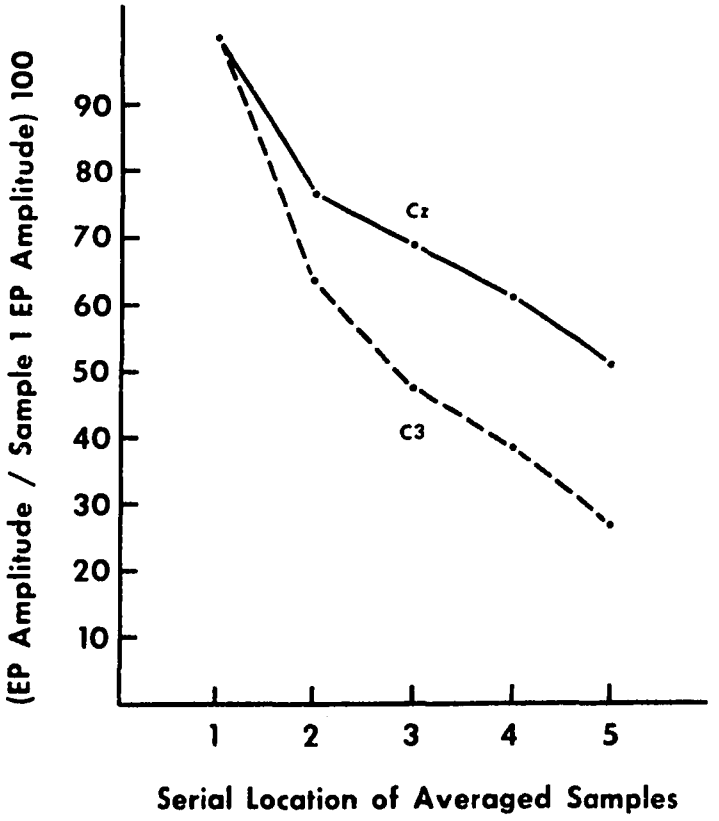

Figure 15. Within trial changes in evoked potential amplitude.

Figure 16. Between-trial changes in evoked potential amplitude. EP amplitudes were estimated at sample point E by deriving to mean of the first pairs of EP averages obtained from each trial. A separate estimate was obtained for each trial, recording site, and Subject. Data obtained from each trial and recording site were then averaged across Subjects. The group mean amplitudes for trials two, three and four were then expressed as percentages of the first trial EP amplitudes. The group mean amplitudes for trial one were 36.33 μV for Cz and 19.50 μV for C3.

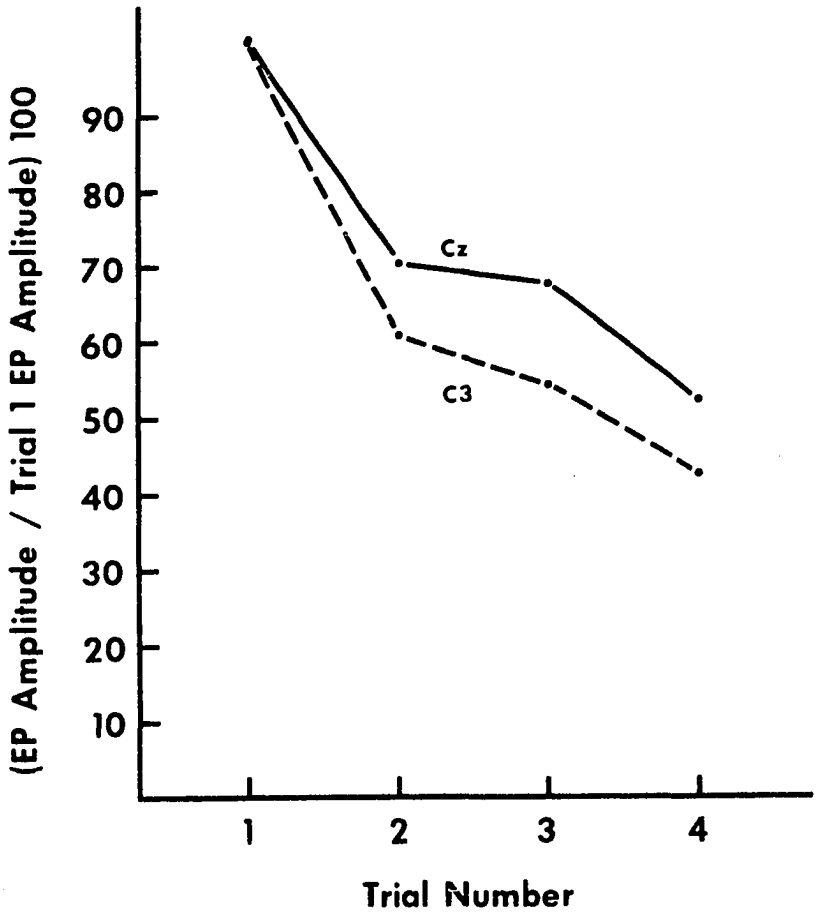

Figure 16. Between-trial changes in evoked potential amplitude.

$P < .01$) greater than that of the C3 EP during the selected 220-250 millisecond sample period. There was also a significant ($F = 14.5$, $P < .05$, usual test, $P < .01$) reduction in EP amplitude common to both recording sites. A second analysis of variance performed upon the data presented in Figure 15 re-confirmed the reduction in EP amplitude ($F = 20.3$, $P < .01$) and in addition suggested the presence of a recording site by samples interaction ($F = 2.74$, $P < .25$, usual test, $P < .10$), as well as an overall difference between the Cz and C3 percent change data ($F = 4.53$, $P < .10$). The latter results imply that there is not only a proportionately greater reduction in EP amplitude at C3, but that this amplitude reduction may be occurring at a greater rate within the specified sample interval. A significant EP amplitude difference between Cz and C3 was also found in the between trial data (Cz > C3; $F = 56.4$, $P < .01$). There was also a possible between trial reduction in EP amplitude common to both recording sites ($F = 4.58$, $P < .10$, usual test, $P < .05$) but no significant interaction between recording site and trials ($F = 2.39$, $P < .25$).

The effects of decreasing EP amplitude and increasing background EEG amplitude may be generalized to the situation in which EPs are elicited at random with respect to background EEG (in particular where alpha activity is abundant). It is particularly important to emphasize that the relative changes between the overall amplitude of the EP and the background EEG may work together to adversely affect the signal to noise properties of the averaged EP. Consequently, unless the nature of these EP, EEG interactions can be specified, the random variance associated with the averaged EP cannot be conclusively assigned to either EP or background

EEG processes. The nature of these interactions will be discussed in greater detail in Chapter IV.

CHAPTER IV

DISCUSSION

The results of this experiment and the literature which will be discussed are most relevant to studies in which human scalp recorded sensory evoked potentials are to be extracted from EEG background activity containing a significant amount of alpha activity. However, this is not an uncommon situation in that a great number of evoked potential studies are carried out under conditions which are designed to relax the Subject, remove him from extraneous stimulation, and so forth. Therefore, Subjects may frequently find themselves in electrically shielded, sound proof, dimly lit rooms under conditions which are most conducive to the production of alpha activity. Although stimulus modifications of predominantly non-alpha EEG may also be important to evoked potential averaging, there has been no attempt to generalize beyond the possible effects which are attributable to the presence of alpha activity.

In order of occurrence, the somatosensory stimulus modifications of the background C3 and Cz EEG consisted of the following: an apparent enhancement of phase related alpha differences for some Subjects; a relative attenuation in the background alpha activity with preservation of synchrony; continued EEG attenuation accompanied by phase shifts which

were of an inconsistent nature across Subjects; and finally, recovery of the amplitude differences between the ±EP averages relative to the differences between the ±EEG averages. Some of these modifications were not observed in all Subjects, and there was considerable variability in magnitudes of the effects between Subjects. There seems little doubt, however, that stimulus modifications of the background EEG may occur as early as 30 milliseconds following stimulation (perhaps even coincident with the earliest EP components) and become well developed 200 milliseconds into the EP sample period. The results of the present investigation suggested that these effects were in general greater and more persistent when recorded from C3. Further, the magnitudes of the stimulus modifications of the background alpha rhythm were observed to vary systematically within and between data collection trials. In addition to the EEG, EP interactions the amplitude characteristics of the EP were also observed to vary systematically.

The appearance of stimulus modifications of the background EEG and in the amplitude of the EP during the EP sample period raises certain questions concerning the use of the averaging technique as a means of obtaining EP data. For example, to what extent are EP, EEG interactions likely to affect the basic sampling requirements and to what extent should such interactions govern the interpretation of EP variance estimates? Of what significance are the differential EEG, EP interactions between recording sites? And finally, how do EEG, EP interactions influence the EP variance reduction schemes outlined in Chapter 1?

Background EEG Modifications and EP Variance

The first problem to be considered is that of determining the

appropriate sample size to permit the reliable extraction of the EP from the ongoing EEG. In Chapter I it was suggested that the pre-stimulus, i.e., non-stimulus, EEG could be sampled in order to obtain EEG amplitude distribution parameters. These parameters in turn could be used to estimate the sample size required in order to reduce the confidence limits for the residual EEG below the anticipated amplitude of the smallest EP components. The presence of amplitude reductions in the background EEG during the EP sample period would result in an increase in the signal (EP) to noise (EEG) ratio particularly during the time in which the late EP components were observed since this is also the period of maximum alpha blocking. Therefore sample size estimates based on non-stimulus EEG amplitude characteristics are likely to be conservative, i.e., the actual background EEG variance during the EP sample period may be overestimated by this procedure. The possibility of a marked reduction in the amplitude of certain EP components and a relative increase in the amplitude of the background EEG activity should also be considered when determining appropriate sample sizes. Similarly, different signal to noise ratios between recording sites should be considered.

The effects of EEG, EP interactions upon the interpretation of the trial to trial random variance of EP samples must also be considered. Even though random EP variance per se might appear to be attractive as a physiological parameter, it must be realized that the variance of the scalp recorded EP cannot be independently observed due to the presence of the background EEG activity. If the EP and EEG processes were independent it might be possible to formulate an additive model in which

the EEG variance could be estimated from non-stimulus EEG samples and then subtracted from the variance of the EP plus EEG samples in order to obtain an estimate of the EP variance. However, since EEG variance characteristics may change considerably during the EP sample period, the problem of formulating appropriate analyses of variance models might prove extremely tedious. For practical purposes it must be concluded that EP variance cannot be observed as a separate entity. This is not to say that variance data obtained during the averaging process is not useful, but rather that these variance measurements cannot be clearly attributed to EP or EEG processes alone.

Two methods of reducing the EP averaging requirements by controlling properties of the background EEG were introduced in Chapter I. Walter and Gardiner (1970) proposed that the EP averaging requirement could be reduced by subtracting the predicted background EEG function from the EP sample during each trial. Since the EEG activity is subtracted (within the accuracy of prediction) from each sample the averaging requirement would be substantially reduced. Ideally the EP could be observed without averaging if the background EEG could be predicted exactly and then subtracted on each trial. A problem arises when the predicted background EEG functions are based on data obtained from non-stimulus EEG epochs. Where the background EEG is modified during the EP sample period the subtracted (predicted) EEG function will be in error. However, where EPs are elicited at random with respect to the ongoing EEG, the errors due to the discrepancies between predicted and obtained background EEG functions might be cancelled during averaging. The relative efficiency of this procedure therefore depends on the extent of the stimulus

modification of the EEG. Where there is little EP, EEG interaction, a substantial reduction in the number of samples per averaged EP might be obtained. Where stimulus modifications of the background EEG are extensive the error resulting from subtraction of the predicted EEG might actually be greater than the obtained background EEG. Under these conditions the use of the EEG subtraction procedure could adversely affect the averaging requirement during those portions of the EP sample period corresponding to the points of maximum EEG modification.

Another method of reducing EEG variance for the purpose of EP averaging is to elicit EPs against a background of selected non-random EEG activity. This technique was utilized in the present investigation. Specifically somatosensory stimuli were triggered according to predetermined conditions of the alpha rhythm. It was suggested that the averaged EP could then be obtained by subtracting the non-random EEG average obtained under non-stimulus conditions, from the averaged non-random EEG plus EP samples. This procedure is subject to the same limitations as the autoregressive prediction method. Specifically, data which were presented in the present investigation showed that any such subtraction of non-stimulus from stimulus EEG samples is likely to include a residual error. This error reflects the difference between the predicted vs. obtained background EEG activity attributable to stimulus modifications of the EEG during the EP sample period.

The observed differential alpha blocking between the Cz and C3 recording sites is of some importance in the use of the above variance reduction schemes. Since there was less alpha rhythm blocking at Cz, the discrepancy between the predicted and observed background EEG activity

would also be less. Thus, Cz might be selected in preference to C3 (under conditions of the present experiment) if it were desirable to obtain a reliable averaged EP with minimum averaging. There are, no doubt, combinations of experimental conditions under which there is relatively greater independence between the background EEG and stimulus conditions than were observed in the present study. To the extent that such independence exists, the use of the above methods for controlling background EEG characteristics might prove to be an extremely efficient means of reducing the EP averaging requirement.

Tukey (1968) proposed that the EP could be derived by adding together averaged samples consisting of EPs obtained against backgrounds of EEG which were 180 degrees out of phase. In principle this procedure should cancel the out of phase EEG activity while summating the in phase EPs. Data obtained in the present investigation revealed the presence of waveform asymmetries which would prohibit complete cancellation of background EEG activity. There is also a possibility that differential alpha blocking (discussed below) could also prevent complete cancellation under certain sampling conditions. In spite of these difficulties it was felt that Tukey's proposed method provided a convenient means of estimating the EP component of the combined EP plus EEG averages for purposes of the present study.

In spite of the difficulties encountered in interpretating certain characteristics of evoked potentials elicited against backgrounds of non-random EEG, it is concluded that this type of averaged signal could prove to be a useful asset to future EP investigations. There seem to be several distinct advantages to using

this type of signal. For example, information concerning systematic changes in the ongoing EEG is to some extent preserved in the averaged EP plus EEG waveform. Possible interactions between the EEG and EP, which are unique to the EP sample period, may be studied in the averaged waveform. Because EEG and EP characteristics may vary independently, the combined signal may be particularly responsive to certain treatment variables. The trial to trial variability of the EP plus non-random EEG samples may be much lower than EP samples obtained against backgrounds of random EEG. This decreased variance in turn reduces the number of samples required for averaging and consequently increases the practical utility of averaged signal. There are, of course, disadvantages to the use of this type of averaged EP plus EEG signal. The sampling method which was used in the present investigation resulted in the selection of a very limited range of EEG "states". These EEG states might have influenced EP characteristics and to some extent may have determined the nature of the observed EP, EEG interactions.

Alpha Blocking and After-Activity

The stimulus modifications of the background alpha rhythm which were found to occur during the first 100 milliseconds of the evoked potential sample period are probably least bothersome to the interpretation of the averaged EP. Even though overall changes in the amplitude of the background EEG may occur, these are typically small and apparently do not disturb EEG phase and frequency characteristics. Under conditions in which the EP is elicited at random with respect to EEG the preservation of the random phase and frequency characteristics

of the ongoing EEG is one of the conditions necessary for a cancellation of the background EEG during the averaging process. In general, the problem of obtaining averaged EP components during the first 100 milliseconds of the EP sampling period becomes one of estimating appropriate signal-to-noise ratios. There are data suggesting that stimulus effects upon the background EEG may become more troublesome to the interpretation of later portions of the averaged EP waveform.

Magnus and Ponsen (1965) found that rhythmic activity or "ringing" developed 200-250 milliseconds following visual stimulation and that this activity was only slightly affected by the phase of alpha during sample initiation. Peacock (1970) proposed that pre-stimulus alpha activity blocks following visual stimulation and is then followed by a period of alpha regeneration which may be time locked to stimulus. This stimulus synchronized alpha activity may appear late in the averaged waveform as a brief burst of alpha activity. Barlow and Estrin (1971) have shown that photically induced after-activity is not simply a return of the alpha rhythm which was blocked by the stimulus, but that the induced alpha activity is characteristically more synchronous than that of the phase triggered non-stimulus alpha samples. These investigators also found little phase correspondence between alpha rhythm averages obtained under stimulus and non-stimulus conditions although the frequencies of the two types of averages were the same On the other hand, Goldstein (1970) found no phase shifts between pre- and post-block alpha rhythm when stimuli were delivered against random phases of pre-stimulus alpha activity and concluded that alpha rhythm pacemaker information is preserved during the period of alpha blocking.

Remond and Lesevre (1967) presented data which suggested that the extent of photic alpha blocking and the characteristics of EEG activity during recovery of alpha activity were both a function of the phase of the alpha rhythm during stimulus delivery. Stimuli which were perceived coincident with maximum negativity (i.e. triggered on previous maximum positivity) of the alpha wave produced alpha blocking and inhibition of the late rhythmic after discharge while stimuli perceived during positive alpha peaks (triggered on negative peaks) produced minimal or no blocking. Stimuli which were delivered at zero crosses of the positive and negative going components of alpha waves produced phase shifts 100 milliseconds following stimulation. An acceleration of the after-discharge was observed in response to stimuli delivered during the positive going phase of the alpha wave. Remond and Lesevre also found that the averaged after-activity was of similar frequency but of greater amplitude when compared to the averaged non-stimulus alpha rhythm. This relative increase in the amplitude of the averaged after-discharge is consistent with an increase in EEG synchrony relative to the point of sample initiation. Horstfehr's (1967) data also suggested the apparent absence of alpha blocking in response to photic stimuli delivered during the negative phase (stimulus information processed during positive phase) of a summated EEG signal derived from a nine electrode array. Stimuli delivered during the positive phase of the alpha rhythm produced well-defined blocking.

Many of the factors which are known to influence after-activity in the visual system have been summarized by Peacock (1970) and Barlow and Estrin (1971). After-discharge or ringing occurs only when alpha is

present, it is enhanced on eye closure and by repetitive photic stimulation, but it is not significantly altered by long flash durations. Ringing disappears with arousal or attention, with natural or barbituate-induced sleep, when the eyes are opened, and in conjunction with certain types of cerebral disease. In addition, Walter (1964) showed that after-activity was influenced by attitudes of Subjects. It is possible that many of these factors influence the properties of the after-discharge occurring in response to non-visual stimuli as well.

Beyond 100 milliseconds into the EP sample period, there is a possibility that the waveform of the averaged evoked potential could be influenced by EEG activity. First, suppose Remond and Lesevre (1967) were correct in their proposal that alpha blocking may occur for stimuli delivered during maximum positivity but not in response to stimuli delivered during the maximally negative phase of the alpha rhythm. Then, there is also the possibility that background alpha activity associated with negative phase stimulation could be incompletely cancelled in the averaged EP since the comparable alpha activity for stimuli occurring during the negative alpha phase may fail to occur due to the blocking of the background alpha rhythm. Further, because the extent of blocking may itself be a function of experimental variables, the resulting error may become systematic with respect to treatment conditions. It remains to be determined to what extent differential alpha blocking could influence averaged EPs obtained in response to stimuli presented at random with respect to alpha activity. The after-discharge, or ringing, which may occur following the alpha block may also appear with the averaged EP waveform. This EEG event may coincide

in time with certain late EP components which have been observed to vary in response to a great variety of experimental treatments. The properties of the after-discharge may also vary with treatment conditions. There could, therefore, be some difficulty in determining whether systematic changes in the late components of the averaged EP waveform were due to EP processes or to changes in whatever EEG processes were responsible for the after-discharge. Through the use of appropriate experimental controls, the confounding effects of after-discharge might be reduced or eliminated.

Non-stimulus Alpha Rhythm Averages

Certain properties of the alpha rhythm averages obtained in the present investigation should also be discussed. The amplitude characteristics of alpha EEG averages obtained in the absence of stimulation are known to be affected by a number of variables. One of the more conspicuous features of the averaged alpha rhythm is the progressive diminution in amplitude over the sample period. Remond, et al. (1969), proposed that this reduction in amplitude was primarily due to the random phase and frequency characteristics of the trains of alpha activity which were averaged. Joseph, et al. (1969), further elaborated this hypothesis and proposed a mathematical model from which an equation describing the damping of the alpha average could be derived. The latter is based upon the frequency instability of the alpha rhythm and assumes that the distribution of alpha frequencies is approximately normally distributed about the mean alpha frequency.

The Joseph, et al., explanation of the damping effect appearing in the averaged alpha activity is fairly straightforward. Since the

phase triggering conditions require that samples be initiated under constant phase conditions, the initial half waves of alpha activity will have characteristically low phase-frequency variance over this period. This variance is ideally zero at the point at which the sample was initiated. However, in moving further into the sample period, the spontaneous phase and frequency variation of the individual alpha averages begins to develop. The net effect is that, at successively later sample points, there is a corresponding increase in the variance of the individual samples. This variance reaches some maximum value as the amplitude properties of the individual alpha waves become random with respect to one another. Through the process of averaging, the initially coherent sample points will combine to produce the greatest amplitude of the average, while truly random portions of the waveform should average to zero. This behavior of the alpha rhythm averages is similar to the damping of the autocorrelation functions which are used to evaluate coherence within EEG epochs. The relationship between EEG averaging and autocorrelation and cross-correlation procedures has been discussed by Remond, et al. (1969), Joseph, et al. (1969, Barlow and Estrin (1971), and others.

Random phase and frequency characteristics of the alpha rhythm have also been used to explain the overall differences between the amplitudes of averaged alpha activity recorded from more than one scalp location where only one site provided the EEG data used in initiating the samples. In this situation, it is obvious that phase and frequency conditions appropriate to initiating the sample at the trigger site may be inappropriate with respect to activity recorded from adjacent sites.

There may therefore be additional random variation due to uncertainty with respect to the conditions of the EEG at the non-trigger source. The extent of the amplitude attenuation between the trigger and non-trigger EEG averages reflects in part the coherence between these sites in much the same way as the more rigorously derived cross-correlation function. The results of the present study showed that there were indeed order relationships between the amplitude of the alpha rhythm averages obtained from the Cz and C3 recording sites and that these order relationships were as predicted by the above random EEG variance model.

Data obtained from autocorrelation studies of the alpha rhythm may be used to estimate the approximate maximum duration of distinguishable alpha activity in averaged alpha rhythm samples. Goldstein (1970) cited data which indicated that the alpha rhythm may remain non-random with respect to initial sampling conditions for as long as 1.6 seconds, although he concluded that one second of alpha coherence is a more realistic limit. It is not apparent from such autocorrelation data how many signals must be averaged to clearly demonstrate the presence of coherent alpha for data points located late in the sample period. The results of the present investigation indicated that averages of as few as sixteen samples were sufficient to produce reliable alpha rhythm averages of several hundred millisecond duration. This was found even when EEG was recorded from central placements of Subjects with only moderate amounts of alpha activity.

There is also a possibility that the amplitude behavior of the individual bursts of alpha activity could contribute to the damped

appearance of the averaged alpha rhythm. However, Remond, et al. (1969), argued that this probably was not the case under the conditions of their experiments since the amplitude trigger criterion was only one-tenth the amplitude of most alpha bursts. Consequently, the probability of samples being consistently initiated at the beginning of alpha bursts was quite low. On the contrary, their method of sampling randomized the amplitude characteristics of the alpha bursts with respect to the final averaged signal (Joseph, et al., 1969). A problem arises, however, where alpha bursts are of a duration comparable to the sample lengths and where such bursts are separated by predominantly non-alpha EEG. Under these circumstances, the earliest a sample may be initiated with respect to alpha rhythm bursts is determined by the onset of the alpha activity and the time required to fulfill sampling criteria. For practical purposes, such samples may be considered to occur at the beginning of the burst. Samples could also be initiated at any time during the burst depending on when the alpha activity occurred with respect to programmed inter-sample intervals. Under these conditions, the sampling of the bursts of alpha rhythm might not be entirely random with respect to alpha amplitude events. A sample initiated at the end of the burst might be followed by low amplitude non-alpha activity, while samples initiated early in the alpha burst would of necessity include the subsequent alpha activity. But there is no equivalent sample which begins before the burst of alpha rhythm, and ends in alpha activity. If the alpha rhythm bursts are short the averaged sample of alpha activity might reflect the actual amplitude damping of the trailing portion of the alpha bursts. The length of the EEG sample with respect to the average

duration of the alpha rhythm bursts may therefore be an important variable determining the relative contribution of alpha activity modulation effects to the resulting averaged waveform.

The average time between the recurrence of bursts of alpha with respect to the sampling rate may also influence the averaged alpha rhythm waveform. If alpha events occur infrequently, the triggering system is likely to be enabled during periods of non-alpha activity and therefore consistently initiate samples at the beginning of the subsequent epochs of alpha activity. Such a situation would provide another example of possible non-random sampling with respect to the amplitude modulation characteristics of bursts of alpha rhythm. The results of this investigation suggested that selective sampling of the alpha rhythm may have occurred. It is not proposed that the EEG sampling procedure can reliably account for the damped appearance of the alpha averages, but it is concluded that the influence of sample criteria and the general characteristics of the alpha with respect to overall EEG characteristics should be seriously considered when interpreting parameters derived from alpha rhythm averages.

Certain relationships within and between the alpha rhythm samples recorded from the Cz and C3 sites were of sufficient stability to be preserved in the averaged data. The slight but sometimes significant phase shifts between the Cz and C3 alpha averages which were found in the investigation are consistent with similar findings in other studies. The frequency differences between Cz and C3 are not as easily explained, although it is known the frequency composition of the gross EEG may vary for activity simultaneously recorded from different scalp

locations. The finding that there were significant asymmetries in the positive and negative going components of the averaged alpha rhythm was unexpected since these EEG averages were illustrated as nearly symmetrical waveforms in the available literature. The observed asymmetries may have been the result of the specific trigger conditions detecting and averaging alpha rhythm characteristics which are peculiar to the particular Subjects. There is also the interesting possibility that these and other alpha amplitude-polarity asymmetries are frequently present but lost in the process of averaging an excessive number of alpha samples in an attempt to obtain "representative" averages. The problems associated with averaging large numbers of evoked potential samples in order to obtain suitable signal-to-noise ratios have already been discussed in Chapter I. It seems reasonable to assume that the same limitations could apply to the averaging of non-random EEG samples. Most investigators averaged in excess of one hundred alpha samples while data in the present investigation were derived from averages of sixteen or at most thirty-two samples. The smaller sample sizes of the present study proved quite satisfactory, although the low variance may reflect the relative efficiency of the sampling conditions.

It is well known that state changes such as drowsiness have effect on both EEG synchronization and EP amplitude characteristics. With respect to the averaged alpha rhythm, Peacock (1970) found that successive averages of 150 samples of occipital alpha, sampled at the rate of 1/2 sec., "waxed and waned" in amplitude due to short-term systematic changes in alpha frequency characteristics. In the present study EEG changes were frequently observed (in the ink write-out of the

EEG) to occur within and between data collection trials. In spite of this, the overall characteristics of the averaged alpha rhythm changed very little. It may have been that the adopted EEG sampling criteria were such that only relatively homogeneous EEG samples were selected from EEG epochs. Although the definition of EEG sampling criteria was liberal with respect to alpha frequency band limits (7.5 - 13.5 Hz), the programmed amplitude threshold and sequential properties requirements may have been too rigorous. This may have been particularly true of the EEG amplitude criterion which was found to be sufficiently reliable to obtain alpha rhythm averages in the absence of any further EEG sampling criteria. The programmed criteria were apparently biased toward the detection of large amplitude events such as sustained bursts of alpha activity. These sampling conditions minimized the probability of acquiring alpha samples during those states in which low amplitude EEG characteristics were predominate.

CHAPTER V

SUMMARY

The purpose of this study was to investigate the relationship between the somatosensory evoked potential (EP) and coincident non-random background alpha activity. An electronic EEG pattern recognition system was developed which permitted the acquisition of alpha rhythm samples which were 180 degrees out of phase with respect to one another. This system was then used to obtain Cz and C3 alpha rhythm samples of each phase under non-stimulus and stimulus (shock to the Median Nerve) conditions. These EEG and EEG plus EP samples were then sorted and averaged into consecutive blocks of 16 samples within each of four data collection trials. Through this procedure four to five averages of each signal type were obtained during each trial from the Cz and C3 recording sites of each Subject. By comparing the phase related differences between alpha rhythm averages obtained under non-stimulus and stimulus conditions, it was possible to evaluate certain assumptions underlying the process of evoked potential averaging.

The EP averaging model asserts that under ideal conditions the presence of stimulus elicited processes does not substantially alter the background EEG characteristics during the evoked potential sample period. If evoked potentials were elicited at random with respect to EEG processes, and if the resulting samples were then averaged, the random

EEG activity should tend to cancel out leaving the residual non-random EP waveform. Given the condition of EEG, EP independence, it is possible to establish confidence limits for the amplitude variance of the residual EEG activity following the averaging process. Similarly, if the amplitude distribution parameters of the ongoing EEG were known, it should be possible to specify the sample size required in order to detect any particular EP component of specified amplitude.

If there were no interaction between EEG and EP processes, then the phase differences (due to non-random sampling) between alpha rhythm averages should be the same under non-stimulus and stimulus conditions. This should be true even though the addition of the EP waveform would tend to distort the overall configuration of the averaged alpha rhythm samples. However, significant modifications of background alpha activity were detected as early as 30 milliseconds following stimulation and may have occurred earlier, perhaps coincident with the earliest somatosensory EP components. Although relative increases and decreases in amplitude differences were observed early in the EP sample period, the most consistent finding was the later marked reduction in the amplitude (alpha blocking) of the alpha rhythm averages. Significantly greater stimulus modifications of the background EEG were observed to occur at C3 and the extent of alpha blocking was found to diminish with successive samples at both recording sites. Significant between sample reductions in the amplitudes of the C3 and Cz evoked potentials were also observed.

The data suggest that the signal (EP) to noise (EEG) ratio may differ between adjacent recording sites due to differential stimulus

modification of the background EEG. Further, there may be systematic changes in the signal to noise ratio during the period of data collection. Confidence limits for averaged evoked potentials based on characteristics of non-stimulus EEG are likely to be conservative if EEG modifications similar to those of alpha blocking are present. However, due to these EEG, EP interactions, the random variance characteristics of averaged (or unaveraged) evoked potentials cannot be attributed to EP processes alone unless the nature of stimulus modifications of the background EEG can be specified. Stimulus modifications of the background EEG also limit the use of proposed techniques in which non-stimulus EEG functions are subtracted from EP plus EEG samples as a means of reducing signal variance. Nevertheless, such procedures may provide a highly efficient means of reducing the number of samples required for EP averaging.

Finally, it was proposed that there are distinct advantages to preserving EEG information by eliciting EPs against backgrounds of non-random EEG activity. For example, stimulus modifications of the EEG which may be unique to the EP sample period may be studied and the averaging requirement may be reduced due to the lower trial to trial variance of the selected EEG background activity. Among the possible disadvantages: selection of a state represented by the EEG and retrieval of the separate EP and EEG averages from the combined signal average.

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