ON THE AVERAGED EEG DURING A DISCRIMINATION TASK

bу

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Abstract

Ten subjects performed a visual discrimination during which their EEG records were taken. Subjects responded following a click that occurred one second after each visual stimulus. In Condition A there was no informative feedback but in Condition B a tone one second after the click indicated whether the discrimination had been correct.

The length of the average slow potential shift or contingent negative variation (CNV) was evaluated following the click to see if presentation of the tone increased its duration. There were large variations between subjects although an average of individual records showed a negative potential after the click in both conditions. Six of the ten subjects showed larger negative potentials just preceding the feedback tone in Condition B than at the corresponding time in Condition A.

Differences between averages for Condition A and Condition B were found for each subject and the covariances between these difference records were factored in an attempt to lassify types of difference records present. Three factors were necessary to account for 68% of the variance in the records.

Estimates of reliability for CNVs were made and possible cellular sources for the negative surface potential were discussed.

Acknowledgment

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INTRODUCTION

In recent years there has been considerable interest in the relation between electrical measures of brain activity and the behavior of men and animals. Early studies focussed principally on the frequency and waveform of human electroencephalograms and on the electrical responses evoked by sensory stimulation measured at the cerebral cortex in animals. The latter experiments often had to be carried out with the aid of anesthetics, yielding results that were not not easily generalized to normal human behavior, or even to the bahavior of unanesthetized animals. Experiments on humans mainly attempted to examine the conditions under which particular waveforms and frequencies appeared in the ongoing EEG. Some researchers were able to examine cortical and subcortical evoked responses in humans (e.g. Brazier 1964) and others superimposed successive EEG records to get a better view of activity following peripheral stimuli, but only with the advent of automatic signal averaging devices (see Dawson (1954) for the first application to electroencephalography) was it possible for workers to accurately measure the effects of sensory stimulation in the electroencephalogram. By presenting many stimuli of the same type to the subject and averaging the electroencephalograms following the stimuli, experimenters can reduce the effects of ongoin; activity that is uncorrelated with the stimulation and enhance the activity evoked by the stimuli so that it may be studied in detail. Much work has been done relating stimulus intensity and modality with the distribution at the scalp and the form, latency, and amplitude of the evoked potentials. (Landau 1967, Perry and Childers 1969, Donchin and Lindsley 1969).

Investigation of evoked responses in situations requiring subjects to make discriminations has led to reports by many investigators of a late component of the evoked response particularly notable in these situations which different workers have related to attention, predictability of the stimulus, and amount of information transmitted by the stimulus (see Donchin and Smith, 1970).

In addition to these relatively brief evoked responses lasting less than 500 milliseconds, there have been reports of longer-lasting potentials measured at the cortex in animals during sensory stimulation (Gumnit 1960, Gumnit and Grossman 1961) and associated with learning and reinforcement (Mnukhina 1961, McAdam et al. 1962, Roland and Goldstone 1963, Low et al. 1966a, Roland 1967, Chiorini 1969, Irwin and Rebert 1970, Donchin et al. 1971). These studies followed extensive demonstrations of cortical slow-potential changes following electrical stimulation of the thalamus and the cortex (Brookhart et al. 1958, O'Leary and Goldring 1959, O'Leary and Goldring 1964).

Averaging of human electroencephalograms in a reaction-time paradigm resulted in the discovery of a sustained potential shift before the response which Walter and associates (1964) named the contingent negative variation (CNV). In this experiment a brief stimulus (a click or flash of light) called the conditional stimulus was followed one second later by a series of clicks or flashes called the imperative stimulus. The subject was instructed to press a button as soon as possible after the imperative stimulus began, terminating the series of clicks or flashes. The averaged EEG between vertex (top of the head) and mastoid electrodes showed a mean difference in potential of about twenty microvolts, negative at the vertex, during the period between the conditional and imperative stimuli. This potential dropped to zero after the response. The authors described this "contingent negative variation" in their averaged signals as a "sign of sensorimotor association and expectancy".

Many variations of the original CNV paradigm have been examined in attempts to define the conditions necessary for the CNV to appear and to test various explanations of the phenomenon, most of which have been summarized in a recent review by Tecce (1972). Tecce attempts to evaluate the data that has been collected as it applies to descriptions cast in terms of expectancy, attention, motivation, cognition, and conation. Almost all of these explanations attempt to describe the subjects' state or condition during the occurrence of the CNV. Walter et al. (1964) apparently use the term "expectancy" because the amplitude of the CNV in their experiments was related to the probability that the second (imperative) stimulus would occur, or to instructions as to this probability. Other workers have often used "expectancy" in a broader sense, indicating an internal state of the subject. Walter and his co-workers also suggested that the CNV might be a sign of "cortical priming". McAdam (1968) looked at the latency of the somatosensory evoked response during the negative shift to evaluate this possibility, and found some limited support for the notion that the cortex is more excitable during the CNV.

Although most discussion of the CNV does not focus on the subjects' responses, Low et al. (1966a,b) have suggested that the CNV occurs because the subject intends to respond. Crucial to this point of view are the results of Kornhuber and Deeke (1965) and others whose subjects showed negative potentials at the vertex preceding movements which they made at their own discretion. They had subjects press a switch several hundred times, either with

hand or foot, while they recorded the EEG and signals that indicated the timesof the switch closures. Averaging the EEG records so that all switch closures came at the same point in the average, they found preceding the movement an average vertex-to-mastoid potential of -5 to -10 microvolts starting one-half to one second prior to the movement and reaching a maximum at or just after the switch closure. This result was verified by other

workers and premotor potentials were revorded from various points on the scalp to attempt to locate the source of the potential, presumed to be somewhere in the cortex (Gilden et al. 1966, Vaughan et al.1968. Von Becker et al. 1968, Vaughan 1969, Deeke et al. 1969, Kornhuber et al. 1969).

It is not unreasonable to compare the premotor potential or Bereitschaftspotential, as Kornhuber and Deeke called it. with the CNV. Most CNV experiments require a motor response by the subject, sometimes a rather gross response like catching a ball or petalling a bicycle (Walter 1967), but usually a small movement such as Kornhuber and Deeke required. However, it has been shown that subjects not required to make an immediate overt response may still show a CNV following a conditional stimulus. Donchin et al.(1972) approached this problem with four tasks and a threemode factor analysis of the CNVs obtained, concluding that a motor response is not necessary for a CNV to be present. Yet much muscle activity, especially of the face and mouth, has been recorded from subhects doing problems or reading to themselves (McGuigan 1970). It is virtually impossible to demonstrate that the subjects in these CNV experiments did not make small movements accompanied by motor potentials, and none of the above experiments included any systematic attempt to locate muscle activity that might have occurred in the absence of observed responses, nor were the subjects carefully watched for movements. Thus while it is clear that the subhects showed CNVs without making the responses that were measured, it does not necessarily follow that the CNV is independent of motor activity and different from the Bereitschaftspotential.

Studies that were designed to assess the effect of motivating instructions on CNV amplitude have indirectly supported the suggestion that the CNV and the Bereitschaftspotential are closely related. Waszak and Obrist (1969) reported an increase in the amplitude of CNVs when subjects were told to respond as quickly as possible as compared with times when they were told to remain relaxed. Trwin et al. (1966) showed increase, in CNV amplitude with increases in the level of a shock that followed the response. Motor potentials measured by McAdam and Seales (1969) were doubled by instructing subjects that they would be paid ten cents for each correct response. Thus attempts to motivate subjects more highly have increased both CNVs and Bereitschaftspotentials.

Several attempts have been made to localize these slow potentials on the head. By comparing the sizes of averaged signals taken from different pairs of electrodes on the same subject, it is often possible to find regions of maximal activity for a particular averaged potential. There are several reports that motor potentials are larger at scalp locations contral teral to the musculature required for the movement (Vaughan et al. 1968, Kornhuber and Deeke 1965, Vaughan 1969, McAdam and Seales 1969). It has been more difficult, however, to localize the CNV (Vaughan 1969), though most workers find it largest at the vertex. This would suggest that the motor potential and the CNV have different origins except that Deeke et al. (1969) report motor potentials maximal at the vertex and results of transcortical recording (Donchin et al. 1971) indicate that negative shifts during conditioning occur at many loci and depend on the specific task performed.

It is important to remember that although these changes in potential found in averaged EEG records are usually assumed to be caused by neural activity, they may also be caused by eye movements (Hillyard and Galambos 1970, Surwillo 1971). Many workers have reported averaged eye-movement potentials that look very much like CNV records, often of larger magnitude than concurrently recorded CNVs. These are reportedly reduced by fixation of the eyes (Hillyard and Galambos 1970).

Muscle activity is another possible contributor to electroencephalograms, since muscle units have electrochemical characteristics similar to neurons. Various investigators have suggested
that muscle activity may be responsible for certain evoked response
components (Bickford et al. 1964, Davis et al. 1964, Mast 1965,
Kern et al. 1969), though there appear to be no reports of
muscle activity contributing to averaged slow potentials. Kohler
et al. (1952) report slow potential changes with tongue movements
but give no data; the report does not appear to have been
systematically verified.

Another possible generator of slow potentials is the galvanic skin response (GSR). If direct current were applied across the recording electrodes, or if the electrodes had different junction potentials, the GSR could appear in EEG recordings, since the modulation of battery or electrode potential by the skin resistance would be indistinguishable from potential changes due to neural or muscular activity. The use of amplifiers with very high input impedance eliminated this problem.

If electrical activity recorded at the scalp is actually due to brain activity, as is usually assumed (Vaughan 1969), the question arises as to how it is related to the firing of cortical neurons whose properties are not fairly well understood (Hodgkin 1964, Stevens 1966). Although it has long been believed by some

that slow potentials recorded at the surface of the cor'ex are closely related to the probability of the firing of neurons (Gerstein 1961), reports by different investigators indicated different polarities of waves associated with neural firing. Many recent papers, particularly ones reporting computer summations of wire microelectrode data, now point to the conclusion that the slower potential changes in the cortex are a very close measure of the probability of firing for neurons in the region of the electrode (see for example Verzeano 1968), the maximum firing rate occurring at the times the slower evoked activity is vecoming more negative. This suggests that, given a close relation between cortical activity and surface recordings, negative shifts at the scalp occur when many cells near the surface of the cortex are firing. In regard to CNV recordings, if electrodes on the mastoid processes, relatively remote from brain tissue, are not affected by non-neural sources of electrical activity, then an increase in potential that is negative at the vertex indicates an increase in'firing of neurons near the vertex.

This conclusion tends to support the notion of "cortical priming" and the studies of cortical excitability during slow potential changes that were mentioned earlier. It also agrees with conclusions that can be drawn from the comparison of motor potential studies and data on single neural discharges in the motor cortex of monkeys making conditioned arm movements, to be discussed later. However in electroencephalography it is rarely known for certain that recordings are solely the result of neural activity. Any of the possible sources or any combination of them may be responsible for changes in an EEG record, so that it is important not to assume a single source when others have not been explicitly ruled out. Virtually all electroencephalography, including the research described in this paper, must be done with incomplete knowledge of the physiological sources of the potentials recorded.

The present experiment was stimulated by results published by Timset et al. (1970) indicating that of a group of subjects in a hospital, patients classified as psychotic or neurotic were much more likely to show a CNV that lasted $1\frac{1}{2}$ seconds or longer after the response. Ninety-one percent of the psychotics tested and thirty-four percent of the neurotics showed this prolongued CNV, but only nine percent of the normals showed this pattern. Timset et al. suggested that the prolonged CNV could indicate that psychotics and neurotics perceived the experimental task as lasting beyond the time of the response.

If the neurotic and psychotic subjects in this experiment showed prolonged CNVs because they were expecting something more to happen, that is, if the extension of their CNVs indicates the same kind of activity as that occurring before the response, then it seems reasonable that normal subjects might also show prolonged CNVs if experimental conditions could be arranged so as to make them expect some event to follow the response. Such conditions would be set up by presenting a stimulus of some significance to the subjects after each response.

A recent report (Delse et al. 1972) describes the effects on the CNV of having an additional stimulus presented before the response. In a delayed auditory discrimination the averaged EEG was found to be typically negative at the vertex for two seconds, from just after the first stimulus until after the response signal. The findings of this study include a CNV as measured before the second stimulus and also a mean negative potential one second later, at the time of the response signal(imperative stimulus). Rebert(1972) presented information about the subjects' reaction times after they responded, but stopped averaging too soon to be able to tell if this procedure lengthened the duration of the CNVs. It does not appear unreasonable that normal subjects might show prolonged CNVs if they were waiting for stimuli following their responses, but there is no experimental evidence that demonstrates it.

The present experiment was designed so that the form of the first stimulus indicated which of two responses was to be made. The second stimulus, one second later, told the subject to respond, and a third stimulus, another second later, indicated whether the response had been correct. Control trials were run in which no informative feedback was provided. Half of the subjects received a neutral third stimulus on these control trials, and half received no feedback stimulus at all. Control trials were always run first so that the subjects would not have reason to pay special attention to this stimulus(or its absence) until they were given special instructions about it. It was anticipated that the CNV would be extended in the blocks of experimental trials in which information was given to the subject after his response.

METHOD

The ten subjects in this experiment were paid volunteers, recruited in the cafeteria-lounge. The EEG was recorded with Beckman silver-silver chloride pellet electrodes placed just above the naison, at the vertex (Cz) and bilaterally at the frontal and parietal positions designated F3,F4,P3 and P4 according to the international 10-20 system of electrode placement (Cooper et al. 1969). Prior to placement of electrodes, electrode sites were cleaned with isopropyl alcohol. Beckman electrode cream was used to secure electrodes placed over hair; those placed directly on the skin were secured by Beckman adhesive pads and electrical contact was made with Beckman electrode paste. Skin drilling (Geddes 1972) was used to lower electrode resistance to between 1000 and 2000 ohms, referred to electrically connected reference electrodes placed over the mastoid processes. An additional electrode on the forehead served to ground the subject.

Recording was done with the subject in an electrically shielded soundproofed booth. Input from the electrodes was fed through six of the eight balanced preamplifiers of an Elma-Schoenander model 160 Mingograph, the remaining two preamplifiers being used to monitor pulses indicating the occurrence of stimuli and responses. All data were recorded with a Precision Instruments Model PI-6200 FM magnetic tape unit, as well as on the polygraph chart. Time constants for the amplifiers were set at 5 seconds and high-frequency rolloff began at 700 Hz.

Signals from the first four preamplifiers were averaged from the beginning of each trial with a Fabri-Tek model 1050 signal averager. Dwell time was 20 ms. per address, with 256 addresses per record, so that the total averaging epoch was 5.12 seconds per trial. Digital output for each block of 16 trials was punched on paper tape with a Tally paper tape punch for later analysis.

Initial data handling was done with a Hewlett-Packard Model 2116B computer. The mean and standard deviation was computed for each record and points more than 2.5 standard deviations from the mean were adjusted as described later. Differences between conditions were computed for each subject and means over all subjects found. The adjusted data was also transferred to magnetic tape for further analysis on an IBM Model 360 computer.

The discriminative stimuli were provided by mounting strips of tape on glass slides so as to form L-shaped silhouettes. Eighty slides were prepared. Thirty-two had longer vertical than horizontal arms, thirty-two had longer horizontal arms, and sixteen had arms of equal length. These were ordered so that the correct response was known for each of the eighty slides, an arbitrary response designated for figures with arms of equal length. The figures were projected sequentially with a Kokak Carousel projector and transmitted via a closed-circuit TV system to the booth in which the subject was seated. Also in the booth were two loudspeakers, one of which received 100 ms tone bursts at 1000 or 500 Hz which served as feedback to the subject.

Each subject was seated comfortably in a reclining chair and positioned so that the TV monitor was in view when his head was at rest against the back of the chair. If necessary, his head was supported with a pillow. This was done to minimize muscle potential artifacts originating in neck and scalp muscles. The subject was instructed to look at a fixation point located at the center of the TV monitor. He was given a 4" X5" box with two pushbutton switches over which he was asked to place the first two fingers of his right hand. Sample stimuli were presented and the subject was instructed to press the right button if the vertical bar on the screen was longer and the let button if the horizontal bar was longer.

There were two experimental conditions. Condition A, the control condition, did not include informative feedback, although non-significant tones were present for half of the subjects. One second after the signal averager was triggered a discriminative stimulus was presented for 100 ms. on the television screen upon which the subject was fixating his eyes. One second after the onset of this stimulus a click was presented, following which the subject was to respond. For half the subjects there were no further stimuli and for half there was a tone pitched either at 500 or 1000 Hz one second after the click. The pitch of this tone bore no relation to the response of the subject, but changed irregularly from trial to trial.

Condition B, the informative feedback condition, always followed at least sixteen trials under condition A. Events proceeded in exactly the same way, with visual stimulus, click, response, and tone in the same sequence, except that the tone occurred for all subjects. However, for this condition the subject was instructed that the pitch of the tone indicated whether he had discriminated correctly or not, a high-pitched tone indicating a correct choice and a low-pitched tone indicating an error, very late responses or failures to respond being counted as errors. The subject was instructed about this difference and asked to always try to make the correct choice. Sample high and low tones were presented and one or two practice trials run with each subject. At least two blocks of sixteen trials were performed by each subject under this condition. Between blocks of trials the subject was told to relax until the data could be punched out on paper tape, after which he was told that a new series of trials was to begin. Instructions were given over an intercom system.

The experimenter initiated each trial. Following this several events had to be coordinated. The averager had to be triggered, the slide projector, the stimuli had to be presented at the right times, and marker signals had to be sent to the polygraph for recording. The triggering of these functions was accomplished with a set of Grayson-Stadler timers and solid-state logic modules.

Results

Only averaged signals from the vertex will be discussed in this paper, since data from this electrode placement has been most frequently discussed by other investigators. Examination of averages during data collection indicated that frontal-to-mastoid and vertex-to-mastoid averages were highly correlated. Pilot work indicated that the CNV was larger after an initial set of training trials, so a block of unrecorded trials preceded each analysed block. Before analysis each data point which had a value exceeding 2.5 standard deviations from the mean for the record was replaced by the mean value of the preceding and following points. This treatment eliminated all artifactual extreme scores. It also reduced the amplitude of the large, brief evoked responses.

Before plotting, the data was also centered and rescaled. Since the averager introduced a constant DC bias in each record, each sixteentrial average was centered by subtracting the mean value for the first fifty points from all subsequent values. These first fifty points covered the first second of each trial which provided a suitable baseline period because the subject received no external stimulation during this period. After each experimental session, calibration signals of 20 microvolts were passed through the amplifiers and synchronously averaged. A computer routine then used this data to convert the arbitrary values of the CNV averages to a microvolt scale. The centered, rescaled average for each subject under each condition is shown in Appendix A.

The most impressive thing about these averages is their extreme variability. Evoked responses following the stimuli are apparent, but there is much more variation of small amplitude than had been anticipated from reports of CNV experiments. This made effective data analysis rather difficult. The experimenter had anticipated gauging the length of the CNV by visually examining the data and determining when the negative shift dropped to the baseline. In fact, the observed variations in potentials were so great as to make such a judgment rather arbitrary. Four of the subjects

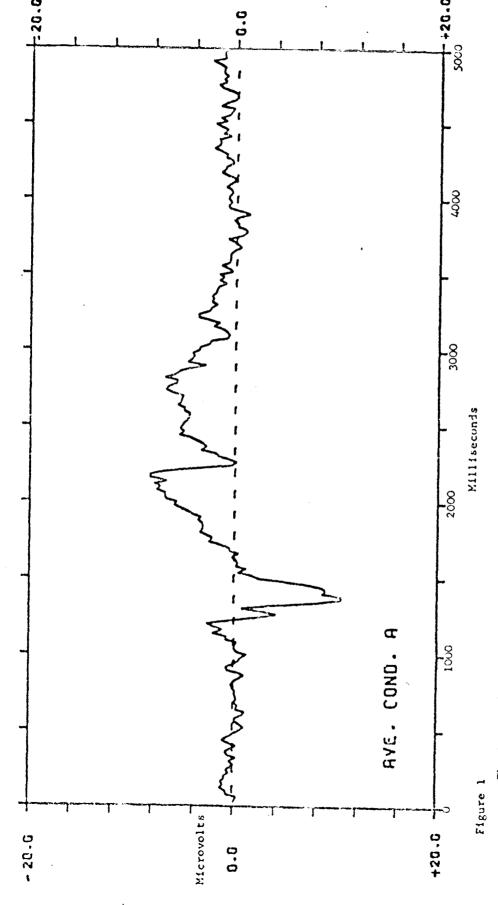
seemed to show the predicted difference and three a difference in the opposite direction, but the variability made these judgments so tenuous that other types of analysis were considered.

A first clarification was gained by computing differences for each subject between averages for Condition A and Condition B, resulting in what one might call a net CNV due to the presence of feedback in Condition B. Plots of these difference records are shown in Appendix B. In these difference records one finds only two of the ten subjects showing the predicted difference in average negative potential before the onset of the feedback tone 3000 ms after the beginning of the record. However there is a negative shift peaking some 200 ms after the onset of the tone in six of the ten difference records. This is much like a CNV but may indicate an altered response to the tone rather than activity preceding the evoked response. This negative peak resulting from the change in conditions also appears both in the average difference over subjects (Figure 3) and in the first factor of the factor analysis of the difference records (Figure 7) to be discussed later.

The difference records in Appendix B are a little smoother than the individual sixteen-trial averages in Appendix A, but not enough to make them readily readable. Examination of the averages over subjects for the two no-feedback conditions, A and A', indicated that there was little difference between them, so they were lumped together to form a composite Condition A record (Figure 1). Condition B records for all subjects were also averaged together to form the composite record shown in Figure 2, and Figure 3 shows the composite of the difference records. Averaging over subjects in this way gave some improvement in the interpretability of the data, though there was some reduction in the amplitude of the evoked responses in comparison with the original records. This is doubtless because of inter-subject variability in the latency of the evoked responses which is commonly observed (Perry and Childers 1969).

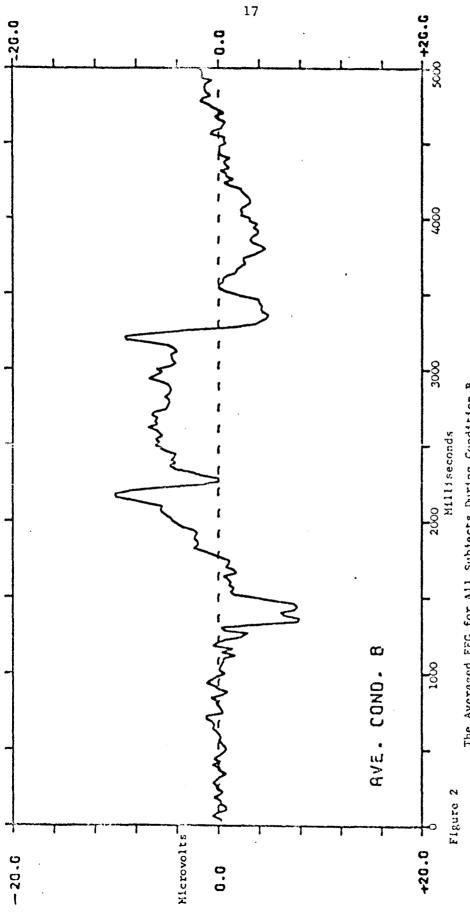
Examination of Figures 1 to 3 shows that the composite CNV remained above baseline after the evoked response to the click (at about 2300 ms) in both the control (Figure 1) and the feedback (Figure 2) conditions, but that there was no consistent difference between conditions until after the time that the tone was presented (3000 ms). Figure 2 also shows a positive potential following the auditory evoked response which has been noted by other authors (e.g. Waszak and Obrist 1969).

The technique of averaging over subjects is commonly used but has some pitfalls, just as averaging over trials has (Brazier 1964, Cooper 1969). It is not easy to know if the records obtained are representative of the whole sample, or if the extreme contributions of a few subjects obscure the modal response. In order to clarify this question in the present case, a principal components analysis was done on the covariance matrix of the differences between conditions for each subject. The difference records for all subjects were taken as the variables, so that the factors after orthogonal rotation defined classes of individuals who had similar records. Plots of the factor scores for the first three factors of this analysis after a varimax rotation are shown in Figure 7 and will be discussed more thoroughly in a later section of this paper. Clearly defined classes of individuals did appear, indicating different response types among the subjects.



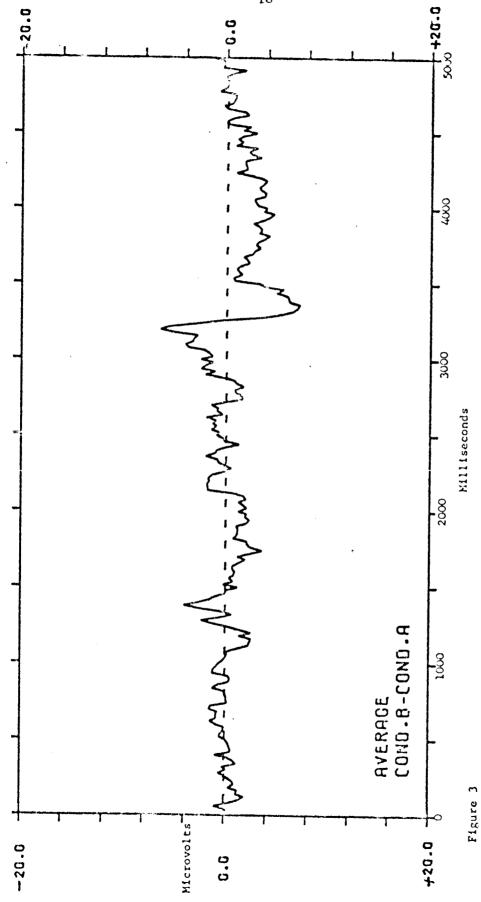
The Averaged EEG for All Subjects During Condition A



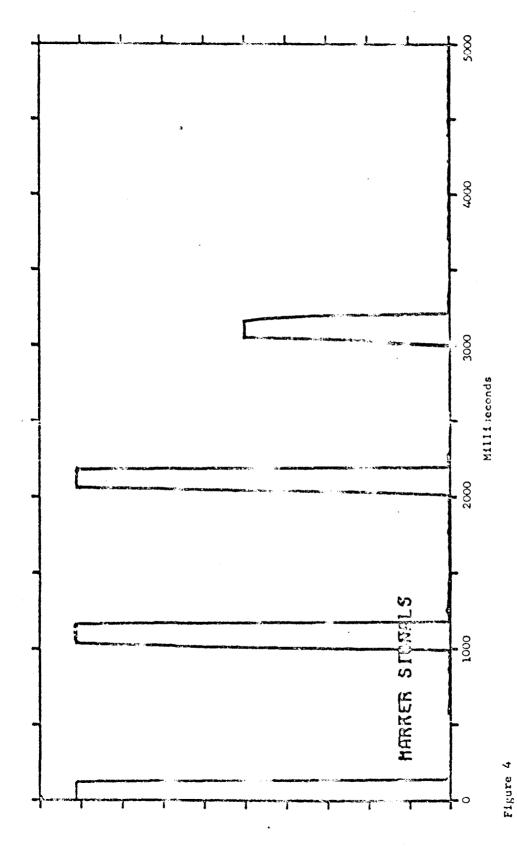


The Averaged EEG for All Subjects During Condition B

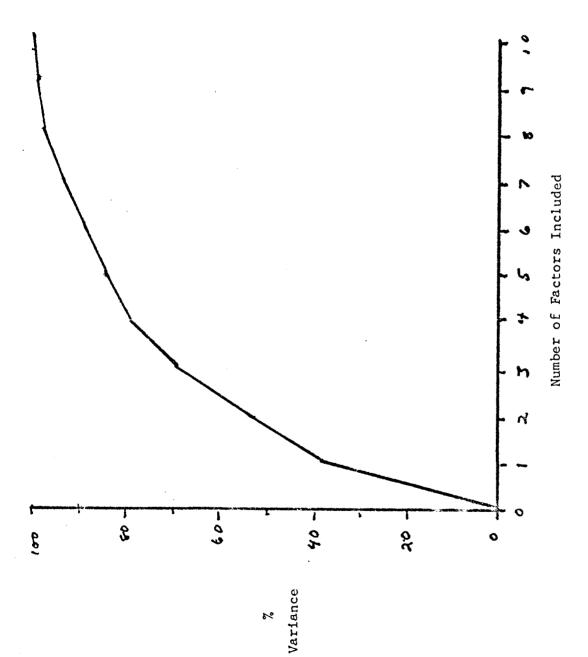




The Average Difference Between Records for Condition A and Condition B.

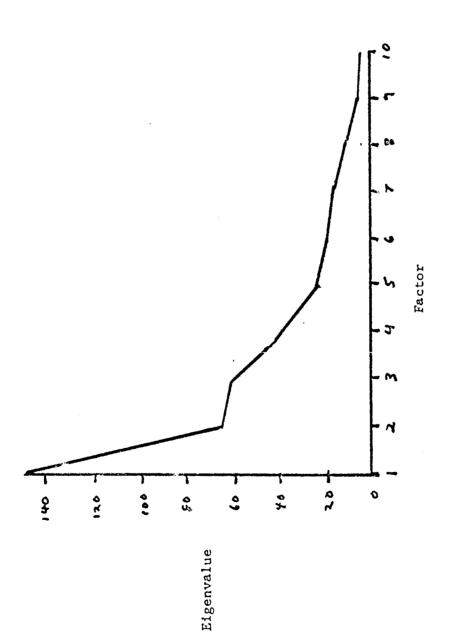


Marker Signals Indicating Beginning of Trial, Onset of Visual Stimulus, Click, and Tone.



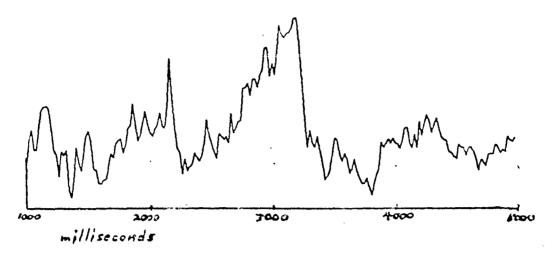
Analysis 3: Variance versus Number of Factors Included

Figure 5



Analysis 3: Eigenvalues of the Principal Components

Figure 6



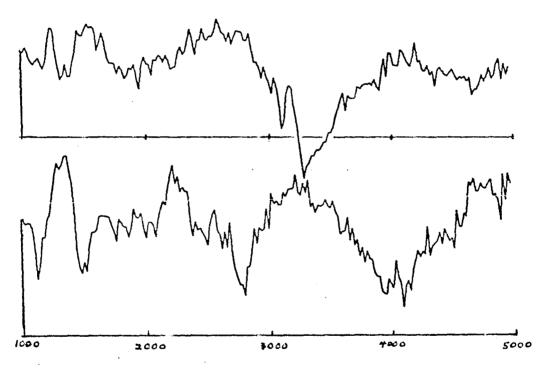


Figure 7

Analysis 3: Factor Score Plots for the First Three Factors After Rotation. The Graphs Begin At The Onset of the Visual Stimulus.

Subject	Factor I	Factor II	Factor III	
в.Т.	X			
N.S.		x		
S.R.			<u>x</u>	
в.К.	x			
T.N.	x			
T.E.	x	x		
м.м.		<u>x</u>		
Z. K.			x	
S.E.	. x		x	
S.A.		x		n

Figure 8

Third analysis: Factor loadings for the three-factor rotation for the factoring of difference records. A small "x" indicates a small loading, an underlined "X", a large loading.

DISCUSSION

Preliminary Observations

Among the results found reliably in all the subjects' records were the responses evoked by the stimuli presented. Those evoked responses appear in the averages over subjects and in the individual records also. Figure 4 may be helpful in analysing these records. It shows the averaged marker signals corresponding with the presentation of all stimuli and with all responses for one subject during sixteen trials. The onset of a marker signal corresponds in each case with the onset of a stimulus, although in the case of the click, the stimulus terminated before the end of the marker. As one can see, the stimuli (visual figure, click, and tone) occurred at points marked 1000, 2000, and 3000 on the graphs, corresponding with intervals of one, two, and three seconds following the triggering of the signal averager. In figures 1 and 2 one can see the response evoked by the visual stimuli with a very prominent positive component occurring some 300 to 400 milliseconds after the onset of the stimulus (at 1000 ms) and lasting 200 to 300 ms. This positive wave is not so noticable in many CNV experiments and is apparently due to the fact that a discrimination was required. Several investigators (John et al. 1967, Kopell et al. 1969. Donchin 1970, Hillyard 1969, Donchin and Smith 1970, Donchin and Cohen 1970) have observed this positive component of the visual evoked response which has variously been named the slow positive component (SPC) and the p3 or P300 component, the latter because of its polarity and approximate latency in milliseconds. The same phenomenon seems to occur in experiments using auditory stimuli (Sutton et al. 1967, Ritter et al. 1968, Sheatz and Chapman 1969, Vaughan et al. 1969). Some investigators have related the phenomenon to stimulus predictability (Ritter et al. 1968), others to information delivery (Sutton et al. 1967) or to stimulus significance (Vaughan et al. 1969).

These studies show the late positive way only when stimuli are detected and when they are not totally predicatable. The stimuli in the present experiment were always detectable but their form was not predictable by the subject, so one would expect to find a prominent late positive wave following the visual stimulus.

The feedback tone was also unpredictable for the subject and easily detected during the feedback condition in this experiment, and Figure 2 illustrates the large late positive component evoked by this stimulus also. This result corresponds with the results of other experimenters (Hernandez-Peon et al. 1956, Davis 1964, Haider et al. 1964). It is very interesting to note that although half the subjects heard the tone in Condition A, there is almost no indication of an evoked response to the tone for that condition (Figure 1, points 3000 to 3500) while there is a very large response in Condition B where the tone had significance for the subjects (Figure 2, 3000 to 3500).

A large diphasic response with a positive peak around 300 ms after the stimulus onset is also found following the click that acted as the signal for the response. This suggests that the importance of the stimulus to the subject rather than its predictability is most crucial for the generation of the slow positive wave, or alternatively, that attention to the stimulus is the critical factor (Donchin and Cohen 1970). The click occurred on each trial and therefore cannot reasonable be called unpredictable.

Earlier components of the evoked response are not clearly seen in the records, partly because of the variability in averages of so few trials as these, partly because of latency differences between subjects' responses which tended to obliterate the response in averages over subjects. Quite as important is the limited resolution possible with this data sampling rate. Each piece of data in these averages represents the integration of 20 ms of EEG, while early components of the evoked response last 50 ms or less. This, combined with limitations on the accuracy of the timers used, would make early evoked response components very difficult to recover.

The negative shift or CNV was not as consistently observed as the evoked responses, although almost every record shows the click-evoked response beginning at a more negative potential than the visual evoked response. The average over subjects shows this clearly. Negative shifts observed in this experiment are not as large as might have been expected from other experiments such as those of Walter et al. (1964), Irwin et al. (1966), Walter et al. (1967) and McAdam (1969). Though the reasons for this are not obvious, one might theorize that subjects found the conditions distracting and thus had smaller CNVs (Tecce and Scheff 1969) or that the subjects were not highly motivated, motivation being related to amplitude of CNV according to some workers (Irwin et al. 1968).

A possible conclusion from the averages over subjects (Figures 1-3) is that the CNV persisted so long in the first condition that differences between conditions were greatly attenuated. However this conclusion does not apply to all cases since not all subjects even showed clear CNVs. The group average may not be representative of most of the subjects' responses; in fact it may cover up two or more response types. This is always a serious problem in evoked response work, where averages for one individual often differ very much from averages for another subject and latency differences may greatly reduce the size of responses averaged over subjects. Variations in latency may be observed in the individual records shown here and this probably accounts for the relatively small amplitudes of evoked responses in the averages over subjects.

John et al.(1964) discuss this problem in some detail. They point out that there are striking similarities in averaged signals which are difficult to demonstrate without special data-analytic techniques such as factor analysis. Further discussion is given in an article by Donchin (1969). Factor analysis of averaged EEG and cortically recorded signals is illustrated by Freeman (1968), Ruchkin et al. (1964), Donchin et al. (1972) and in the paper by John and co-workers mentioned above.

Factoring the Data

Factor analysis is often useful in reducing the rank of a data matrix. The data matrix in this case was composed of the pairs of averaged EEGs for each subject. Factoring was done in an attempt to reduce the number of data sets required to describe the original set of averaged signals. Since the factoring was designed to find a set of typical subjects, it could be called a Q analysis with the averaged EEG records treated analogously to test profiles in a profile analysis (Rulon et al. 1967); Q analysis determines the minimal set of profile types that characterize a data set.

Previous investigators appear to have factored the correlation matrix or the covariance matrix when working with evoked responses (see John et al. 1964, Bures et al. 1967). If the form of a signal regardless of its amplitude is of principal interest, the correlation matrix should be factored. When the amplitudes of the signals are also important, the covariance matrix is more appropriate. In the present experiment, natural units were available because the first second of EEG activity on each trial was recorded and averaged providing a natural origin for subsequent data. When data is in natural units such as microvolts and centered with respect to some invariant zero-point, the product moment of the raw score matrix is most appropriate for factoring (see Horst 1965, p109).

It was felt at first that the variance of the first fifty points in every record might be a reasonable extimate of the error variance of the record. A preliminary principal components analysis was done after rescaling the cross-product matrix by these baseline variances. (Standard deviations of the baseline data are given in Table I for reference). This weighted the variables very unequally in the analysis (see Table II for the cross-products matrix) and made interpretation of the factor loadings very difficult. It was felt that certain

peculiarities in some of the baseline records (for example the extreme values early in the records of subject S.R., Appendix A) made these baseline variances poor estimates of the error variance in the data. For this reason a second analysis was done using the unscaled mean cross-products. The factors were rotated orthogonally according to the varimax criteria. Factor loadings for the varimax rotation of the first six factors are shown in Table 3.

It had been hoped that one or more factors would correspond to the typical vectors for Condition B, the feedback condition, after the varimax rotation was done. So far as this rotation maximizes simple structure (Thurstone 1947) and so far as the data are generally different in conditions A and B, there should be one or more factors that had high loadings on Condition A variables and low loadings on the Condition B variables, or vice versa. Such a loading pattern was not discovered (see Table 3). The data vectors (variables) are entered by subject, first for Condition A, then for Condition B, so that some factors should show uniformly high loadings on alternate variables. No factor loading pattern of this sort emerged. Either the rotation procedure was inappropriate or the data could not be factored into types that differed on the two conditions.

Because of question about the applicability of the rotation procedures, an alternate approach was taken, suggested previously by John et al. (1964) and Ruchkin et al. (1964).

The averaged signal for Condition A was subtracted from the signal for Condition B for each subject as described earlier. These ten difference vectors were cross-multiplied to form a matrix of mean cross-products and factored as described above. Random variability is reduced as effectively by subtracting this way as by adding or averaging signals (Magnusson 1966, p93).

A factoring of this sort could be used to indicate what response types went into the mean difference between conditions shown in Figure 3.

Factor loadings for this analysis are given in Table 4 and Table 5. Table 4 shows the unrotated principal components, Table 5 the varimax rotation of the first three factors. This number of factors was arrived at by looking at the eigenvalues of the factors (Figure 5) and the variances included by different numbers of factors (Figure 6). Looking at the drop in eigenvalues after the third factor and studying the rotated factor score plots, the three-factor solution was chosen. The first two factors account for 54% of the variance, and as will be shown later, demonstrate most of the apparent features in the average over subjects (Figure 3). To retain 95% of the variance, eight factors would have been required, but this is clearly to make a meaningful reduction from the ten original variables.

Factor scores for the three-factor rotation are plotted in Figure 7. This figure shows:

- 1. A first factor that might be construed as a CNV factor, with a rise from 2600 to 3200 ms and a decline to 3500 ms after trial onset, from just before the onset of the tone to about half a second after it;
- 2. A second factor with scores showing a sharp dip between 3100 and 3600 ms, from the end of the evoked response to the tone until approximately 500 milliseconds later, corresponding to the slow positive component discussed earlier;
- 3. A third factor typified by a long slow wave peaking at 4000 ms.

The plot of factor scores for F_{a} ctor 1 in Figure 7 suggests that ther was a type of difference record which showed an increasing negativity during the 750 ms preceding the second tone. This undoubtedly corresponds with the observation made previously that six of the ten subjects showed a greater vertex negative potential just before the tone under Condition B.

The factor score plot for the second factor indicates that a different group of subjects was responsible for the positive shift following the tone in the average difference record (Figure 3 at 3500 ms.) than was responsible for the negative potential preceding the tone in that figure. In other words, the slow positive wave after the tone did not always go with the negative potential preceding it; a different subset of subjects was involved.

The plot for the third factor shows a rather long period of relative positive potential at the end of the task, maximal about 4000 ms after trial onset, appearing in the averages of Figures 2 and 3 as well as in Figure 7. This post-feedback positivity is most typical of yet a third subset of subjects.

Though the groups of subjects contributing to each factor overlapped, each factor tended to be representative of only a few subjects, and was dominated by the record of a single subject. Figure 8 decenstrates the extent to which simple structure was attained with the three rotated factors.

To summarize, then, it was possible to factor the subjects' records into three distinct types exemplifying each of the major features apparent in the average of differences between the experimental conditions. The first factor profile showed a rise in negative potential just before the onset of the feedback tone. This is similar in shape to the response prior to the second tone in the experiment of Delse et al. (1972) and to the "CNV" of McAdam and Rubin (1972) as well as the readiness potential of Kornhuber and Deeke (1965). The other two factors showed respectively a late positive response after the tone and a slow positive potential one second after the tone. This factoring of response types suggests that extensive conclusions should not be based solely on the average over subjects, since different groups of subjects responded differently.

Variability Within Subjects: The Noise Problem

To examine variability apparent in the raw averages, the data were converted into difference records to reduce variability due to individual subjects. Another sizable source of variability was within the records of individual subjects. This variability should not be surprising at all, really; signal averaging was used to reduce it in the first place.

Changes in the EEG due to external stimulation of the organism may be thought of as signals superimposed on the ongoing activity which tends to mask the . To the extent that this activity is stochastically distributed over trials for every interval following the stimulus, the improvement in signal-to-noise ratio obtained by averaging increases with the square root of the number of trials. A similar situation occurs in psychological testing where error in measurement contributes "noise" to the subject's true response (the "signal"). The relation between reliability in psychological testing and the signal-to-noise ratio in signal detection theory has been discussed by Chronbach and Gleser (1964) who demonstrate that the signal-to-noise ratio =r/(1-r), where r is the specific reliability of the test. With this in mind, some estimates were made of the reliability of the records analysed in this experiment.

It will be remembered that the second sixteen trials under each experimental condition were analysed. For one of the subjects there was data available for the first sixteen trials of one condition. The correlation between blocks of trials was .51, corresponding to a signal-to-noise ratio of one-to-one. This is probably a low estimate of reliability because, as mentioned earlier, pilot work had indicated that there was an increase in the size of the CNV over trials. For a better estimate of reliability, the data for three sets of thirty-two trials were retrieved from the original data tape and alternate trials averaged for both odd and even numbered trials.

The pairs of averages obtained in this way correspond to scores on odd and even items on a psychological test, from which split-half reliabilities can be computed. Since each average consisted of sixteen trials, however, it was not necessary to "step up" the coefficients to get a fair estimate of reliability. The three coefficients computed in this way were .78, .64, and .62, giving estimates of signal-to-noise-ratio from 1.6 to 3.8 for the sixteentrial blocks in this experiment. Forty to sixty percent of the variance in the records was reliable.

An estimate of the upper limit on reliability that could have been obtained in this experiment can be found by using the variance of the baseline data as an estimate of error variance and the mean square of the following data as an estimate of true and error variance lumped together. Such reliability estimates were computed for each subject. The mean estimate was .83, with a standard deviation of .15 for the set of estimates. The high mean value may reflect the assumption that unreplicable EEG variability remains about the same throughout each trial. There are indications that this is not true. Elul (1968) shows that the amplitudes of EEG signals are distributed differently during problem-solving and resting periods, and Horvath (1969) finds that an early response evoked in the cortex of cats is more variable near its peak than at other times.

Another way of estimating reliability in the present experiment would be to find the correlations between Condition A and Condition B records for each subject from the end of the baseline period until the onset of the tone. This is defensible because there was in general little difference between responses for the two conditions until after the tone was presented. Reliabilities calculated this way should be good estimates for other CNV experiments except that they should be a little high because the baseline period is not included here. The mean of ten coefficients obtained in this way was .69 and the standard deviation was .31. If the one negative coefficient was ommitted, the mean was .78 and the standard deviation was .14.

Several factors may be important in limiting the relfability of these averages. One obvious factor is signal size. Several other investigators have reported CNVs of the same general size as found in this experiment. Hillyard and Galambos (1970) for example, show CNVs of about 13 microvolts when eye movements were well-controlled or their contributions subtracted from the averages. This value compares with about 8 microvolts prior to the response cue and about 6 microvolts prior to the tone in the present experiment. Some investigators report CNV amplitudes as high as 20 microvolts, but it is not clear that these are mean values for an unselected group of subjects.

Another factor affecting reliability is the number of trials. While 16 trials is not a low number for CNV work, evoked response studies often average 50 to 150 responses, greatly increasing signal-to-noise ratios in the averages. Going from 16 to 128 trials, for example, would increase the signal-to-noise ratio by a factor of 2.8.

Some experimenters show records apparently averaged over very few trials which seem very free of "noise". This is partly due to the scaling of figures in the journals, but also due in certain cases to the use of smoothing procedures used in the data analysis. These procedures are often quite defensible, depending on the frequency range of the signals of interest and the bandpass of the amplifiers.

Smoothing routines have the same effect as analog filtering at the preamplifiers, except that they reduce machine-generated noise as well as unwanted EEG or electrode noise; their effect is to lower the maximum frequency response of the data analysis system. Judicious choice of filter settings has always been important in EEG work (see Perry and Childers 1969). By reducing the range of frequencies passed by the amplifiers, one is able to exclude a large portion of the "noise" spectrum, thus greatly improving the signal-to-noise ratio. In this experiment the EEG could have been filtered

to eliminate the high frequencies, down to perhaps ten cycles per second. Since most of the energy of the waking EEG lies between ten and fifty cycles per second, this would greatly reduce the "noise" energy in the averages. The same thing could be done digitally, as mentioned above. The problem is that such drastic low-pass filtering could decrease the amplitude of the evoked response, increase its latency, and alter its waveform. Possible alterations in signal amplitude, phase, and waveshape are inherent in any filtering system, analog or digital.

Another source of noise in the EEG is muscle potentials. These can be reduced by filtering and also by reducing the extent of chronic muscle tension in the subject. In the present experiment attempts were made to limit the extent of muscle potential artifacts. In addition to continuous monitoring of the EEG, care was taken to see that the subjects were able to see the stimuli without tensing their neck muscles. A pillow was used to support the head and the subject was re-positioned whenever excessive muscle activity appeared in the EEG. It is possible that muscle potentials contributed significantly to the noise level in the EEG, but care was taken to eliminate them.

Finally there is the possibility that AC noise from the power lines got into the records. Much attention was given to shielding the equipment and the subject from stray fields and 60 Hz notch filters were placed between the amplifiers and the signal averager, so this should not have been a problem.

It therefore appears that with the present electrode and amplifier system the signal-to-noise ratio could have been reduced only by an increase in the number of trials per average or by more smoothing or filtering of the signals. Electrical Sources of the CNV

The CNV, when observed, may be the result of one or more of several physiological processes. In any particular case, any one of these may be principally responsible for an observed negative shift at the scalp. An attempt will be made here to enumerate the possible sources and assess their importance in the present experiment.

Hillyard and Galambos (1970) and Surwillo (1971) have discussed the effects of eye movements on CNV records. The eye forms an electrical dipole with the retina negative with respect to the cornea. This means that when a subject looks down, any scalp electrode will have a greater negative potential with respect to mastoid reference electrodes than when the subject looks up. Potential changes due to eye movements are largest at locations nearest to the eyes, with frontal electrodes showing much larger eye-movement artifacts than occipital electrodes when mastoid references are used. Eye movements themselves may be monitored with electrodes placed near the eyes. Peters (1971) has briefly reviewed applications of this technique.

Several methods for controlling eye movement artifacts in CNV experiments have been suggested. One method found quite effective by Hillyard and Galambos (1970) was eye fixation, which was incorporated into the design of the present experiment. A separate pair of eye electrodes or a single sub-orbital electrode referenced to the mastoids would have made independent averaging of eye movements possible. This would have been very interesting because signals from forehead electrodes seemed correlated with vertex signals and because Wasman et al.(1970) have found evidence of interactions between CNVs and electro-oculograms in subjects who were supposed to be fixating their eyes.

It is generally assumed that electrical activity at the scalp is generated in the cerebral cortex. Vaughan (1969) reviews some of the evidence for this assumption. It seems likely that cortical activity can be measured at the scalp, but the more basic question of how neural activity affects gross cortical potentials is still being answered (Landau 1967, Thompson 1970). Theoretical models have usually been based on the notion of a dipole generator imbedded in a uniformly conducting medium (Shaw and Ross 1955). A recent discussion by Humphrey (1968) for example, derives the antidromic evoked response in pyramidal cells by considering an idealized pyramidal cell within a uniformly conducting medium. Recording electrodes would detect the field set up in this conductor from a flow of current from apical dendrites of the cells to their somata. Reduced current flow from the dendrites during inhibitory post-synaptic potentials would appear at the surface as a negative potential with respect to distant reference electrodes.

Another possibility discussed by Creutzfeldt et al. (19662) and Amassian et al. (1964) is that a negative wave could be generated by dendritic spikes travelling toward the surface. Others (O'Leary and Goldring 1964) have discussed the possibility that surface negativity could result from presynaptic inhibition, presumably near the cortical surface.

None of these theoretical suggestions has been totally proven. Earlier studies of the relation between neural activity and gross potentials (Clare and Bishop 1956, Verzeano and Calma 1954) were not entirely conclusive although this and some recent work (Fromm and Bond 1964, 1967) generally found spike activity to be maximal near the positive peaks of surface records (see also Pattan and Amassian 1960). Creutzfeldt et al. (1966a,b) found surface-negative potentials and cellular depolarization closely related to responses evoked by electrical stimulation of the thalamus and to spontaneous surface waves as well.

These rather conflicting results are in contrast with straightforeward findings obtained more recently by averaging signals from metal microelectrodes in the brains of unanesthetized animals. There seems to be a high correlation between local evoked responses recorded by these electrodes and the probability of spike activity recorded by the same electrodes, regardless of their depth or location (Fox and O'Brien 1965, John and Morgades 1969). Most studies find a strong relation between spike probability and the negative slope of the evoked response (Dill et al. 1968, Verzeano et al. 1968, Fox and Norman 1968, Thompson et al. 1969, Weinberger 1969). It therefore looks as if the firing of neurons causes extracellular regions near them to become briefly more negative, as might be expected from the present understanding of the mechanism of neural discharge (Hodgkin 1964, Eccles, 1957). However, it is still impossible to prodict the exact effect of cellular discharge on scalp recordings without some notion of the number of cells firing, their location, and their orientation with respect to the electrode.

It is also becoming increasingly apparent that any complete theory of the genesis of evoked or sustained cortical potentials must include a description of glial cell activity. It was pointed out several years ago (Galambos 1961) that a complete theory of brain function would have to include glial activity. Recent reviews (Kuffler 1967, Lasanski 1971) indicate the extent of current research on the functions of glial cells. Work with tissue cultures (Hild and Tasaki 1962) and amphibians (Kuffler et al. 1966, Orkand et al. 1966) have shown that glial cells are passively responsive to external fluid composition, particularly potassium ion concentration, which recent work indicates may in turn control the rate of glial protein synthesis (Takahashi et al. 1970). Other workers (Henn and Hamburger 1971) find a possible role for glial cells in the control of inhibitory transmitter substance in the brain.

Effects of glial cell activity on gross evoked responses and sustained potential shifts have been analysed by several workers. The glial cells form an electrically interconnected network, as opposed to the neurons which are electrically isolated. The affinity of the glial "compartment" (Adey 1970) for potassium ions has led to the suggestion that uptake of K⁺ by glia may be responsible for the beta-wave of the electronate ogram (Miller and Dowling 1970), the slow positive wave of the cerebellar evoked response (Eccles et al.1969), part of the excitability cycle in seizure activity (Sypert and Ward 1971), and slow potential shifts. Some workers find evidence for glial contribution to evoked responses and sustained potentials (Castelluchi and Goldring 1970, Cohen 1970, Somjen 1970), though others do not find that glial depolarization contributes to slow potential surface records (Grossman et al. 1969).

Another line of evidence is provided by recent studies of pyramidal tract neurons and conditioned movement in animals (Evarts 1966, 1968, Humphrey et al. 1970) which have shown that pyramidal motor unit firing is closely correlated with operant conditioned responses in monkeys. When forceful responses were required of the monkeys, firing continued between responses, but the maximum firing rate always occurred at approximately the same time that the greatest force was emerted (Evarts 1968). The ease with which units may be found whose firing is related to movements (Humphrey et al 1970) suggests that a very large number of cells in the motor cortex are involved in any gross movement. Since an average negative potential occurs on the scalp over human motor cortex prior to voluntary movements (Kornhuber and Decke 1965, Gilden et al. 1966) it seems very likely that the firing of many motor units corresponds with a negative signal in the averaged EEG. One could test this hypothesis indirectly by looking at the motor potential (Bereitschaftspotential) when responses of various strengths are required. The larger the force exerted, the greater the amplitude of the motor potential should be if cellular activity corresponds with EEG activity as suggested here.

It is important to remember, however, that cortical unit activity and eye movements are not the only possible sources of potential changes at the scalp. Other physiological processes, particularly muscle activity, might be involved. Some evoked responses or components of evoked responses are caused by contractions of either cranial or neck muscles according to the reports of Bickford et al. (1964), Davis et al. (1964), Mast (1965), and Kern et al. (1969). Muscle activity can be recorded in the absence of overt movement (Natson 1930, McGuigan 1970), as when subjects are "thinking" or reading to themselves. Although muscle activity has rarely been considered as a possible contributor to slow potential changes, several muscles (for example the temporal muscle, the sternomastoid, masseter, and tongue) lie near or beneath electrode placements often used in electroencephalography. The nature of muscle tissue and the potential changes in muscle units during contraction (Aidley 1971) suggests that slow potential shifts could occur in muscles during contraction. If they do occur, they could be very important to the interpretation of CNV data.

CONCLUSIONS

The results of this experiment do not show the prolongation of the CNV that was predicted. Certain subjects gave the expected results, but their data was not typical. The mean of all subjects' data was helpful in identifying the important features occurring in both conditions, but the Q-analysis of response types indicated quite clearly, as preliminary examination of the data had, that there was no single response type that truly represented the whole group. The fact that there appear to be several types of response, and in fact several types of change in individual pattern following the change in experimental conditions, serves to illustrate the extent of variability in av raged EEG signals that has yet to be explained.

Although there is certainly no support from this data for the conclusion that normal subjects do not show prolonged CNVs, neither does it appear that this experiment effectively manipulated the length of the CNV. It does seem that just prior to the tone the vertex was likely to be more negative than say, half a second later. The previously cited experiment by Delse et al. (1972) also demonstrated the same effect.

Changing the significance of the tone seems to have greatly increased the amplitude of the response evoked by it. This is a very interesting demonstration of the already well-documented fact that instructions can greatly influence the size and form of evoked EEG activity in humans. Just what causes this activity nobody knows; and it will be very interesting to see what future research reveals about it.

Probably the most important conclusions of this experiment are those concerning the variability of CNV records and methods of evaluating averaged EEG data. The reliability estimates that were derived on the basis of within-subject variability were

fairly large, suggesting quite replicable patterns of response for each subject, though the variability across subjects was very great. How people could show such reliable individual patterns that are so different is a puzzle worth studying in some detail. One possibile way of considering the problem is in terms of individual mediating responses. Perhaps the variation among subjects is due to different postures and actions regularly taken by each subject during different portions of the experimental trials. This sort of activity has been observed in animals during delayed discriminations, and may well also be characteristic of people. If it occurs, it would indicate that motor activity, represented at the motor units or at the motor cortex, might be a significant contributor to the variable slow-potential shifts in these data.

SUMMARY

The EEG records of ten subjects were averagd over the trials in discrimination tasks with and without informative feedback. The inclusion of an informative tone did not clearly increase the duration of the vertex negative slow-potential, although six of the ten subjects showed a larger negative potential just following presentation of the feedback tone.

Most subjects showed negative shifts at some time after the presentation of the first stimulus, but the latencies and durations of the shifts were highly variable. Each subject tended to have a fairly reliable response pattern regardless of the experimental condition but the patterns varied greatly across subjects.

The most noticable change resulting from the introduction of feedback was a very large evoked response to the feedback tone. The response when the tone did not carry information was very small by contrast. Evoked responses to the first two stimuli were apparent in all subjects; a late positive component was very apparent in the response to the visual stimulus.

An increase in the number of trials per average or a reduction in the frequency range of the amplifiers would have reduced within-subject variability. Averages over subjects, either equally weighted as in Figures 1-3, or differentially weighted as in Figure 7, appear to make the data more interpretable, though the simple average tended to obscure different response types, as factoring showed.

The negative shift, when it does occur, is probably related to the average motor potential prior to voluntary movements which other workers report. The motor potentials appear to indicate increased neural firing in the motor cortex, although activity in other cortical areas may be involved in the CNV. It is also possible that muscle activity contributes to slow potentials, although no evidence is currently available on this point.

Subject	Standard Deviation	of Baseline Signal
	Condition A	Condition B
В.Т.	2.10	1.81
N.S.	3.16	1.46
S.R.	1.55	3.87
В.К.	2.62	2.46
T.N.	1.79	1.66
T.E.	1.38	1.84
М.М.	. 2.48	2.06
Z.K.	2.63	2.31
S.E.	1.91	2.85
s.A.	1.79	1.57

Table I Standard Deviations of Baseline Signals

Mean Cross-Products

	2	3	4	5	9	7	æ	6	0		2	3	4	5	9	7	8	6	0	
8.06																				Toble
.85	76.9																			11.
2.15	.76	1.92																		
7.28	90*9	3.61	23.14																	
-1.19	60.	11	3.65	16,26																
29	-1.54	28	-2.05	49	1,65															
2.44	2.86	1.15	3,52	71	32	3.26														
3.33	.13	1.18	3.17	-1.60	.01	1.54	3,38													
2.48	1.76	1.39	. 88	-7.67	-1.12	1.85	2.11	9.94												
6.32	2.00	2.60	6.41	-6.71	-1.02	2.45	3,33	8.77	14.10											
	.85 2.15 7.28 -1.1929 2.44 3.33 2.48	.85 2.15 7.28 -1.1929 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76	.85 2.15 7.28 -1.1929 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.611128 1.15 1.18 1.39	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 49 71 -1.60 -7.67	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 32 .01 -1.12 3.26 1.54 1.85	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 32 .01 -1.12 3.26 1.54 1.85 3.38 2.11	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 49 71 -1.60 -7.67 1.65 32 .01 -1.12 3.26 1.54 1.85 3.38 2.11	.85 2.15 7.28 -1.19 29 2.44 3.33 6.94 .76 6.06 .09 -1.54 2.86 .13 1.92 3.61 11 28 1.15 11.18 23.14 3.65 -2.05 3.52 3.17 16.26 49 71 -1.60 1.65 49 71 -1.60 1.65 49 71 -1.60 1.65 32 .01 3.26 1.54 3.38	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 32 .01 -1.12 3.26 1.54 1.85 3.38 2.11	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 32 .01 -1.12 3.26 1.54 1.85 3.38 2.11 2.94	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 32 .01 -1.12 3.26 1.54 1.85 3.38 2.11 2.94	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 49 71 -1.60 -7.67 1.65 49 71 -1.60 -7.67 1.65 49 71 -1.60 -7.67 3.26 1.54 1.85 3.38 2.11 2.94	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 32 .01 -1.12 3.26 1.54 1.85 3.38 2.11 9.94	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 32 .01 -1.12 1.65 32 .01 -1.12 3.26 1.54 1.85 2.94	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 11.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 32 .01 -1.12 1.65 32 .01 -1.12 3.26 1.54 1.85 3.38 2.11 9.94	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 -11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 32 .01 -1.12 3.26 1.54 1.85 3.38 2.11 9.94	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 32 .01 -1.12 3.26 1.54 1.85 3.38 2.11 2.94	.85 2.15 7.28 -1.19 29 2.44 3.33 2.48 6.94 .76 6.06 .09 -1.54 2.86 .13 1.76 1.92 3.61 11 28 1.15 1.18 1.39 23.14 3.65 -2.05 3.52 3.17 .88 16.26 49 71 -1.60 -7.67 1.65 32 .01 -1.12 3.26 1.54 1.85 3.38 2.11 2.94

Table IIa

Analysis 1: Product-Moment Matrix After Rescaling By Baseline Variances.

Mean Cross.Products

20	8.11	5.20	2.89	14.47	.02	-1.56	4.38	4.21	3.74	8.44	5.50	4.71	10.49	14.85	.6.31	6.13	-1.07	59	16.68	17.70	
19	7.48	6.65	2,35	16.79	2.69	-1.52	4.72	2.87	.83	5.73	4.67	.21	12.85	12,17	06.9	6.51	-3.80	-3.96	20.62		
18	2.78	-3.80	1.04	-3.73	-5.37	96*	20	2.29	3.55	4.18	2.59	7.68	-2.10	2.66	35	.73	5.25	8.49			
17	1.02	-1.48	1.01	-3.68	-3.26	.01	.74	1.39	4.29	2.09	2.60	5.19	-2.74	1.17	.59	67	8.27				
16	4.03	.93	.89	67.4	. 80	.19	1.21	1.15	1.87	5.89	2,41	3.11	5.23	7.12	2.47	68.9					
15	3,58	2.14	06.	5.61	.57	66	1.81	1.63	1.66	2.98	2.54	1.43	4.62	5.50	5.04						
14	8.92	2.55	4.00	14.13	.30	-1.53	2.71	4.18	5,39	11.55	8.34	9.37	10.75	20.46							
13	6.05	2.78	2.10	14.20	3.70	95	1.97	1.82	54	4.17	5.23	1.55	13.54								
12	5.68	-3.48	1.87	1.26	-5.27	.47	.34	3.75	5.77	7.72	79.7	13.60									
11	4.29	.05	2.63	6.37	97.	.10	1,30	1.96	2.15	3.97	7.44										
	_	6 1	~	_+	١٥	٠,٠	_	~	_		_									_	

Table IIb

18

Analysis 1: Product-Moment Matrix After Rescaling By Baseline Variances.

				Facto	rs		
		I	II	111	IV	V	VI
	1	3.58	1.54	89	-2.45	2.34	 76
	2	.44	-2.03	2.32 .	.10	2.82	 79
	3	3.25	.98	.67	.18	.81	70
Variables (individual	4	5.59	-2.46	1.33	-1.05	2.27	.26
averages)	5	.73	 96.	1.14	17	50	5.52
	6	71	.35	-4.02	05	 73	.04
	7	1.12	.17	.43	03	3.79	 95
	8	2.21	1.80	71	37	1.85	-1.07
	9	. 48	2.05	2.20	86	.82	-4.15
	10	2.35	1.18	1.32	-2.97	.58	-4.08
	11	2.62	1.28	.06	69	. 42	.18
	12	2.74	4.45	82	-2.18	98	-2.20
	13	6.52	-2.75	 05	-3.95	2.21	1.84
	14	6.75	1.69	2.63	-5.01	.83	-1.04
	15	1.48	.944	1.55	-2.74	3.74	.97
	16	1.25	 39	60	-5. 33	.78	-1.02
	17	 09	4.65	.65	.82	. 49	75
	18	. 1.33	6.36	-2.86	34	-1. 29	-2.91
	19	3.79	-2.57	.68	- 3.58	5.23	.64
	20	3.66	34	1.13	-3.69	3.63	57

Table III

Analysis 2: Rotated Loadings on the First Six Factors Accounting for 86% of the Total Variance.

Unrotated Principal Components

	5.7		×	26	.52	00.	78	02	32	• 38	•04	60.	2.10
	7.0		ΙX	09.	69	.23	.27	2.14	.56	41	59	09	.42
	11.9		VIII	79.	17	59	-2.17	05	1.78	-,42	54	1.55	54
	17.3		VII	2.23	.12	09	2.01	12	74	- .33	21	2.58	.70
	19.2		IA	• 39	34	90	1.44	-1.45	2.63	-1.67	1.39	-1.29	.78
Eigenvalues	23.8	Components	>	1.33	3.91	1.37	.34	.30	1.51	1.31	34	65	16
E18	37.6	8	ΛI	1.20	.24	-1.83	-1.13	1,96	52	1.71	4.93	.03	17
	60.2		111	3.54	-1.68	5.43	-1.80	-1.53	-1.22	-1.90	1.95	71	37
	64.2		11	3.00	-2.82	-1.18	79	-2.23	.58	5.24	-1.94	-2.19	2.00
	148.0		1	-4.58	-2.86	5.67	2.53	.72	4.58	7.60	2.61	5.00	2.37

Table IV

Analysis 3: Unrotated Principal Components

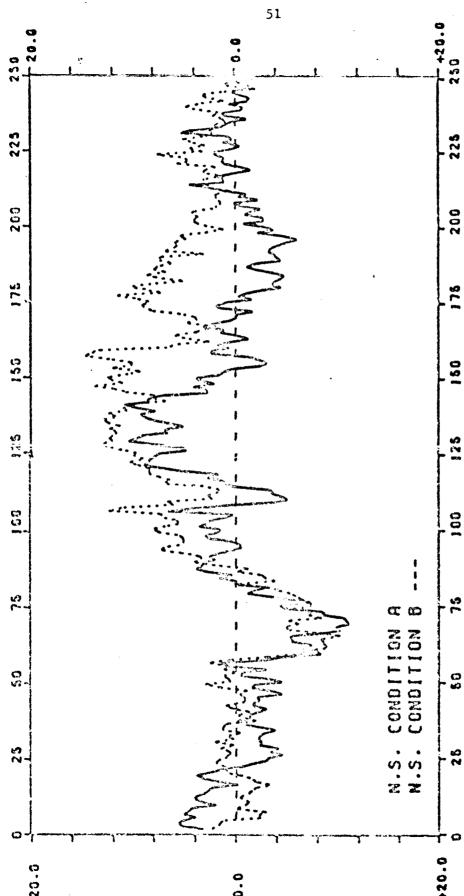
	III	16	-1.92	7.68	.27	30.	1.45	62	3.60	3.01	.37
FACTORS	7 1	78	-3.79	2.00	1.21	-1.16	3.38	7.11	13	1.39	3.06
	ы	-5.41	76.	.25	2.91	2.56	3.05	1.10	1.19	4.39	55.
		П	7	8	4 Sararrays		9	7	8	6	10

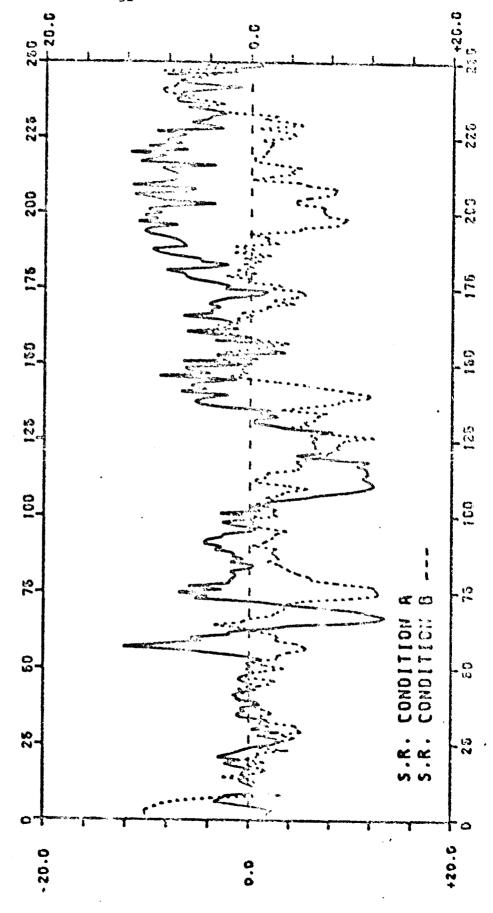
tine	for	
Third analysis: Varimax Rotation of	First Three Factors Accounting	68% of the Total Variance

Table V

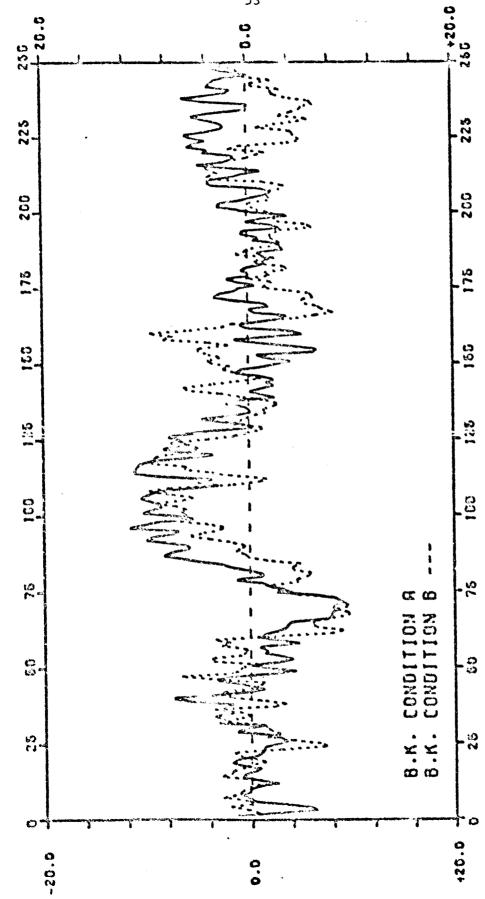
Appendix A

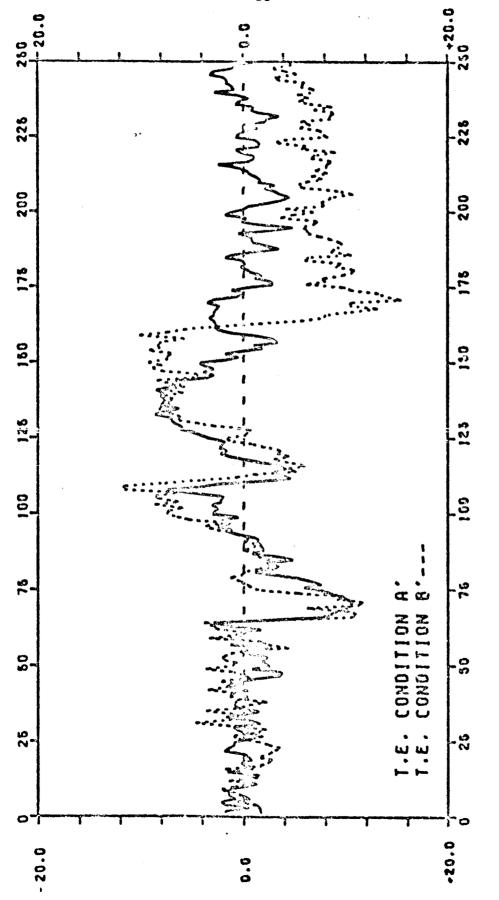
Sixteen-trial averages for each condition performed by each subject. The first five subjects performed under Condition Λ , in which tones were presented after the responses but were not contingent on the response (Pp. 50-54). The second five subjects performed under Condition Λ^{\dagger} , in which tones carrying information followed the responses (Pp. 55-59).

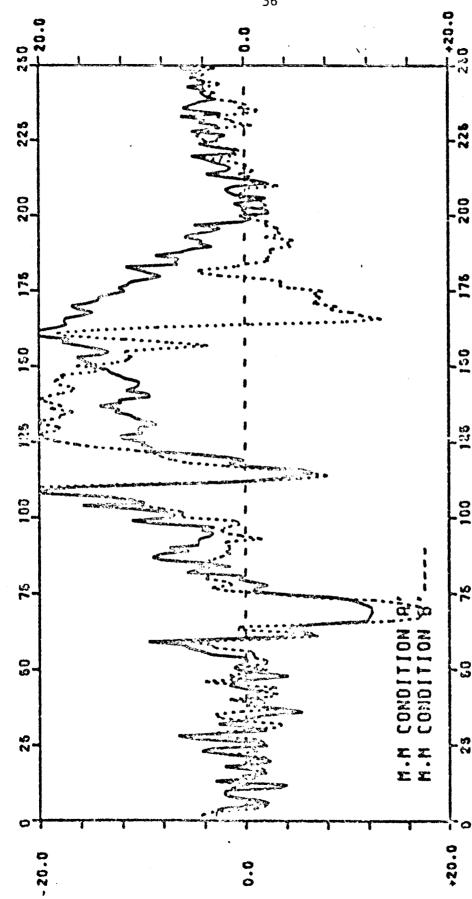


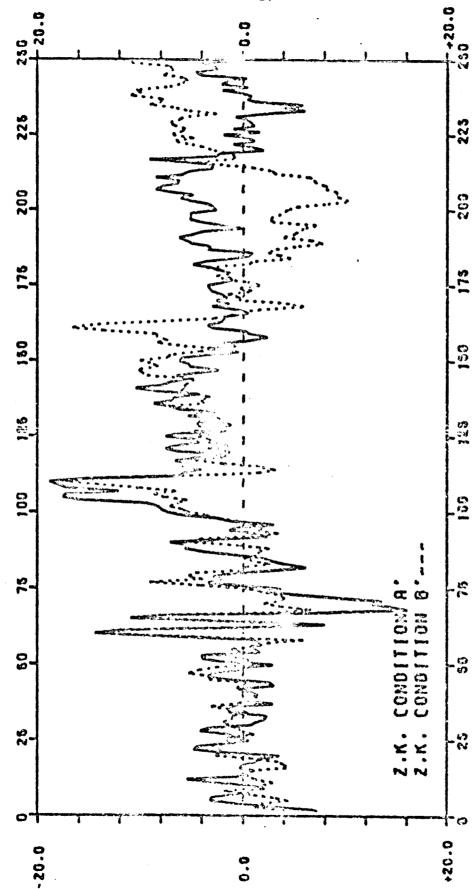


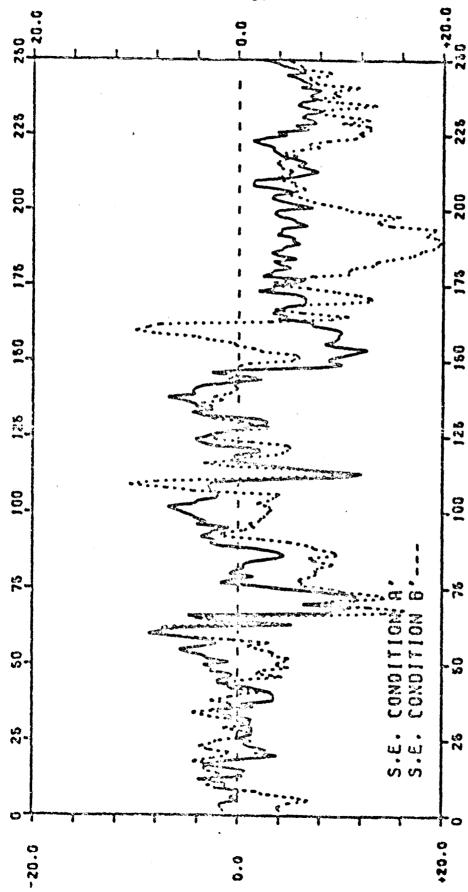


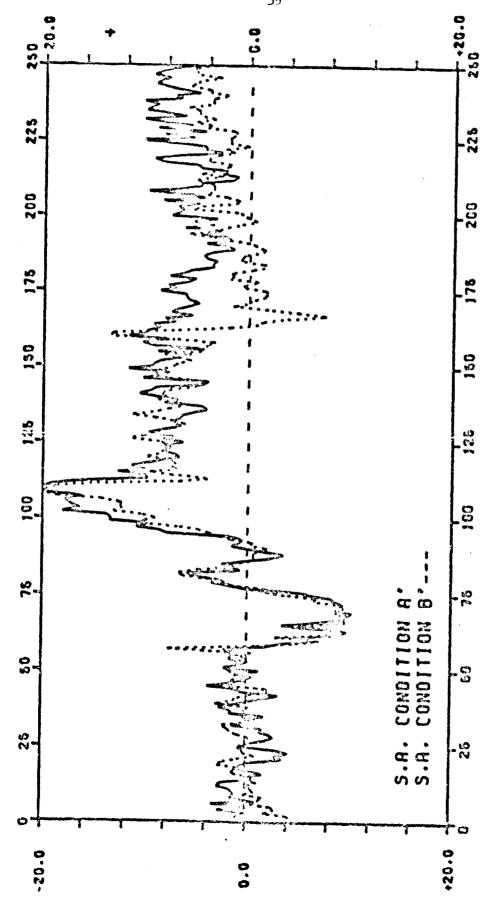






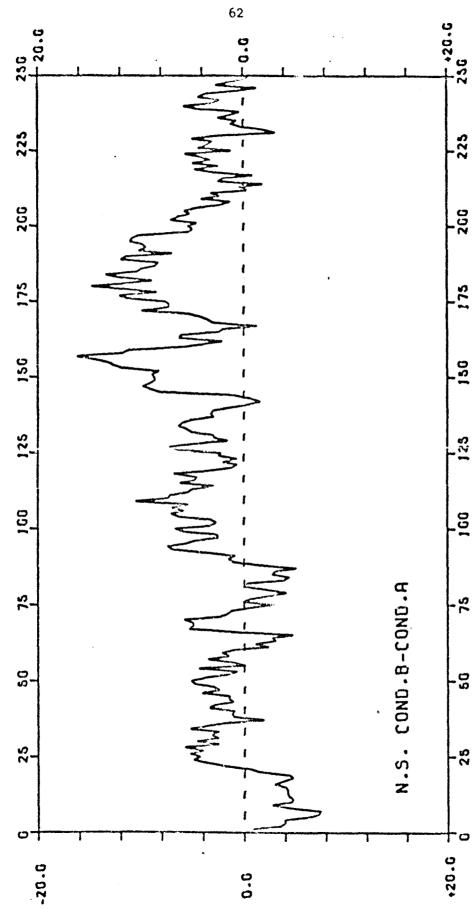


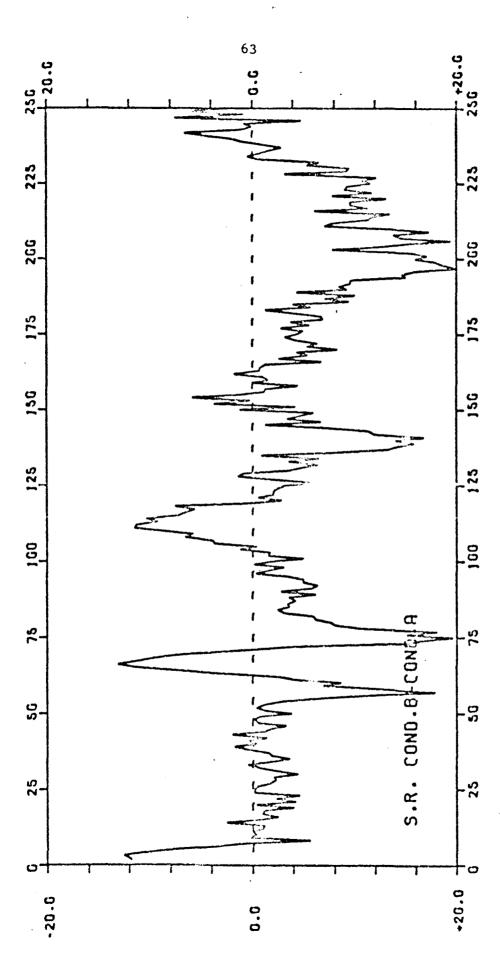


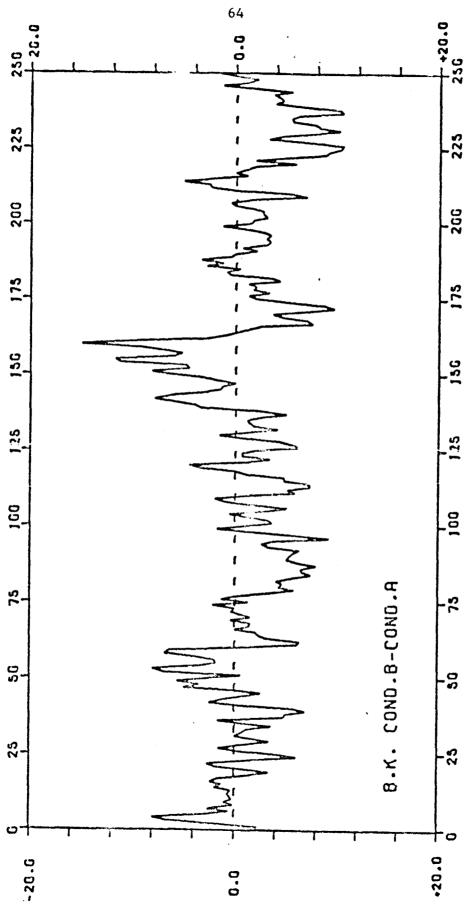


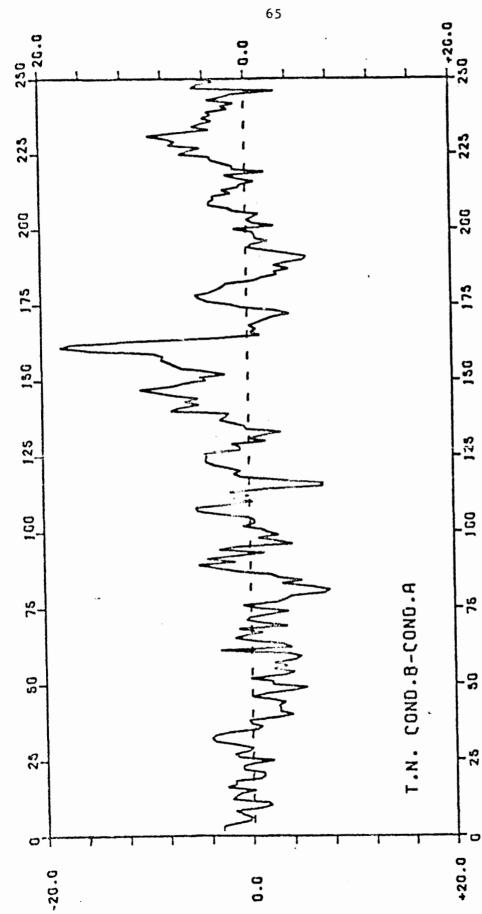
Appendix B

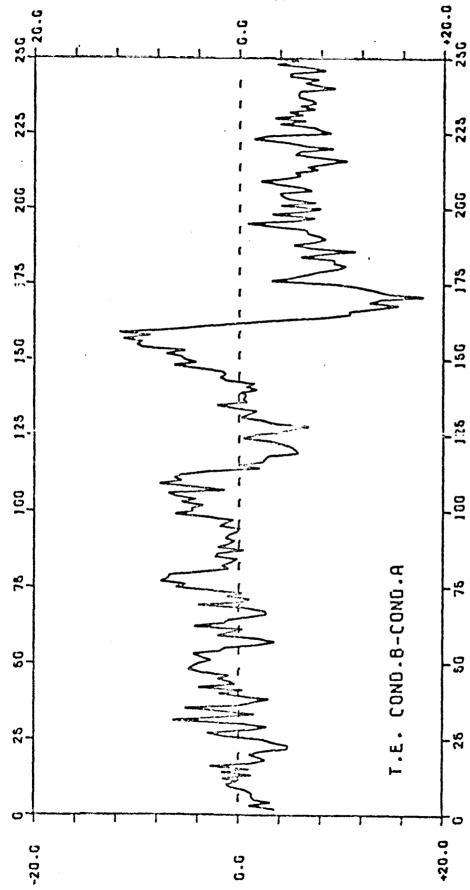
Differences between Condition A (or A^{\dagger}) and Condition B records for each subject.

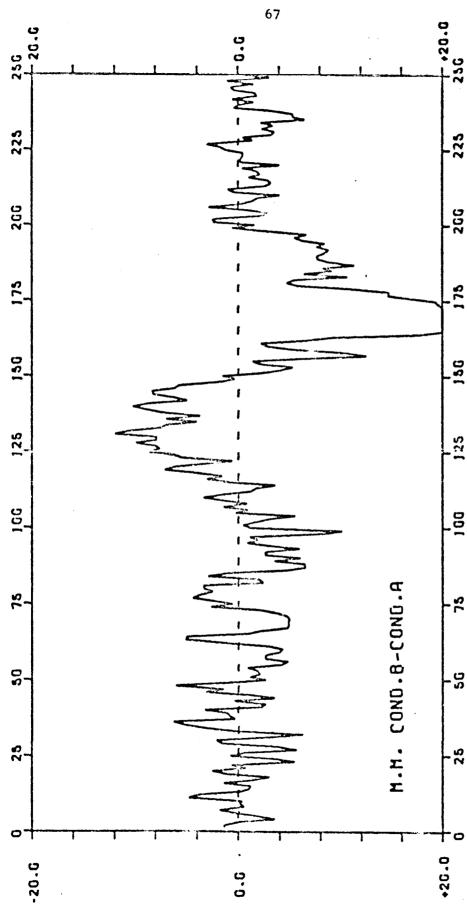


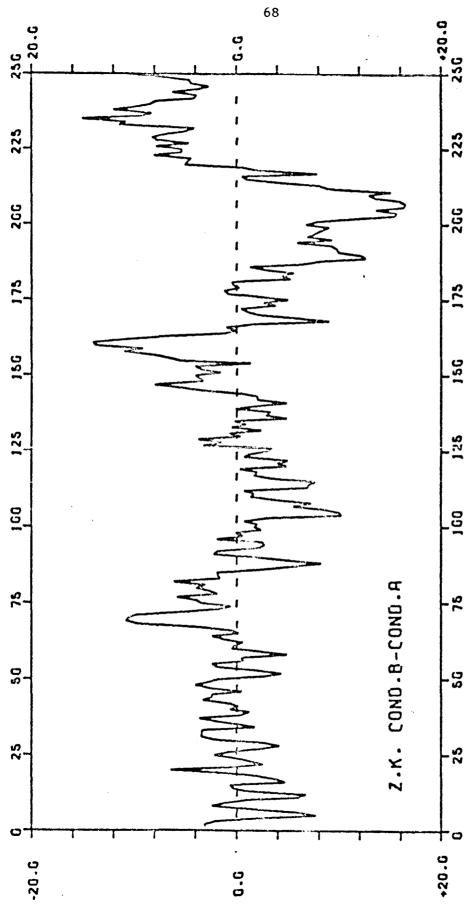


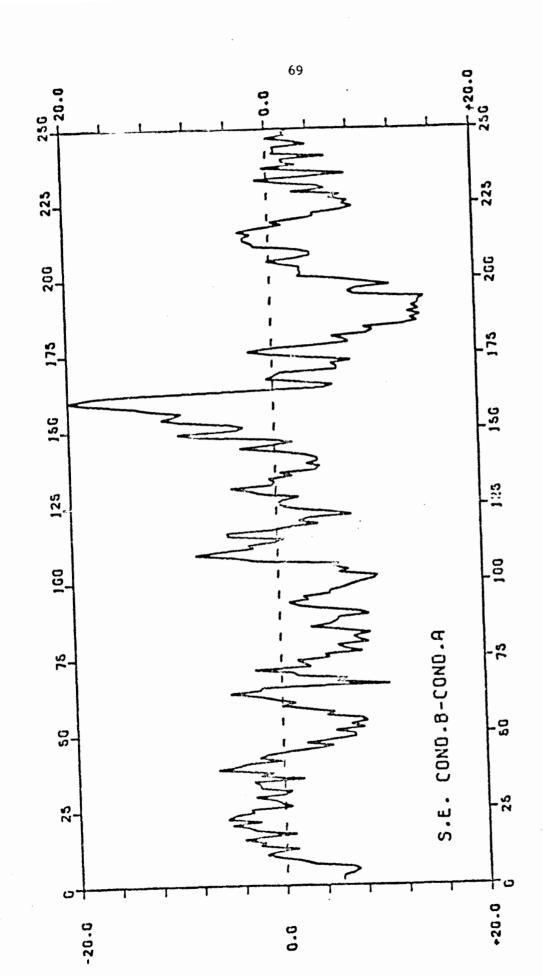


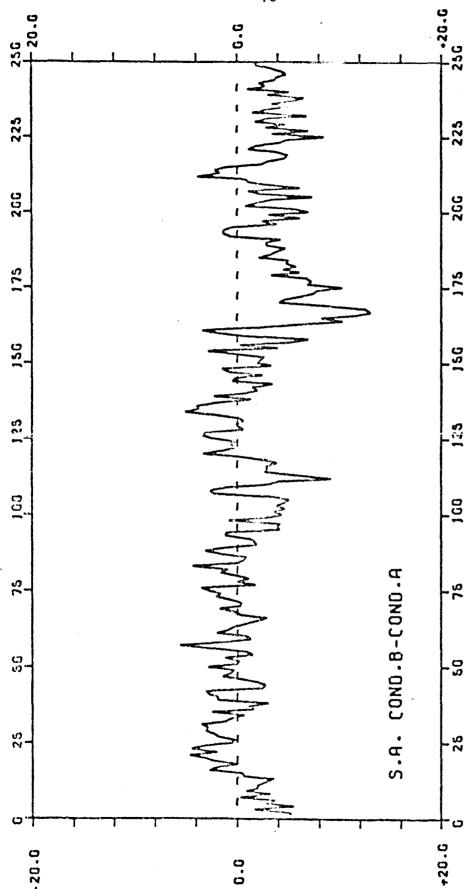












Bibliography

- Adey, W. R. Cerebral structure and information storage. In Progress in Physiological Psychology. Vol III.

 New York: Academic Press, 1970. Pp. 181-200.
- Aidley, D. The Physiology of Excitable Cells. Cambridge: Cambridge University Press, 1971.
- Amassian, V. E., Waller, H. J., Macey, J. Neural mechanisms of the primary somatosensory evoked potential. Annals of the New York Academy of Science, 1964, 112, 5-32.
- Bickford, R. G., Jackobson, J. L., & Cody, D. T. R. Nature of average evoked potentials to sound and other stimuli in man. Annals of the New York Academy of Science, 1964, 112, 204.
- Brazier, M. A. B. Evoked responses recorded from the depths of the human brain. Annals of the New York Academy of Science, 1964, 112, 33-59.
- Brookhart, J.M., Arduini, A., Mancia, M., & Moruzzi, G.
 Thalamocortical relations as revealed by induced slow potential changes. Journal of Neurophysiology, 1958, 21, 499-525.
- Bures, J., Petran, M., & Zachar, J. <u>Electrophysiological</u>
 <u>Methods in Biological Research</u>. Prague: Academia Publishing
 House, 1967.
- Castelluchi, V.F. and Goldring, S. Contribution to steady potential shifts of slow depolarization in cells presumed to be glia. <u>Electroencephalography and Clinical Neurophysiology</u>, 1970, 28, 109-118.
- Chiorini, J. R. Slow potential changes from cat cortex and classical aversive conditioning. <u>Electroencephalography and Clinical</u> Neurophysiology, 1969, 26, 399-406.
- Clare, M. H. and Bishop, G. H. Potential wave mechanisms in cat cortex. Electroencephalography and Clinical Neurophysiology, 1956, 8, 583-602.
- Cohen, M. W. The contribution by glial cells to surface recordings from the optic nerve of an amphibian. <u>Journal of Physiology</u>, 1970, 210, 565-580.

- Cooper, R. Osselton, J. W., & Shaw, J. C. <u>EEG Technology</u>. London: Butterworths, 1969.
- Creutzfeldt, O. D., Watanabe, S., & Lux, H. Relations between EEG phenomena and potentials of single cortical cells: I. evoked responses after thalamic and epicortical stimulation. Electroencephalography and Clinical Neurophysiology, 1966, 20, 1-18.
- Creutzfeldt, O. D., Watanabe, S., & Lux, H. Relations between EEG phenomena and potentials of single cells: II. spontaneous and convulsoid activity. Electroencephalography and Clinical Neurophysiology, 1966, 20, 19-37.
- Cronbach, L.J. and Gleser, G. The signal to noise ratio in the comparison of reliability coefficients. Educational and Psychological Measurement, 1964, 24, 467-480.
- Davis, H. Enhancement of evoked cortical potentials in humans related to a task requiring a decision. <u>Science</u>, 1964, 145, 182-183.
- Davis, H., Egbertson, M. Lowell, E.L., Mast, T., Satterfeld, J., & Yoshie, N. Evoked responses to clicks recorded from the human scalp. Annals of the New York Academy of Science, 1964, 112, 224-225.
- Dawson, G.D. A summation technique for the detection of small evoked potentials. <u>Electroe-cephalography and Clinical Neuro-physiology</u>, 1954, 6, 65-84.
- Deeke, L., Scheid, P., & Kornhuber, H. Distribution of readiness potential, pre-motion positivity, and motor potential of the human cerebral cortex preceding voluntary finger movements. Experimental Brain Research 1969, 7, 158-168.
- Delse, F.C., Marsh, G.R., & Thompson, L.W. CNV correlates of task difficulty and accuracy of pitch discrimination. Psychophysiology, 1972, 9, 53-62.
- Dill, R.C., Vallecalle, E., & Verzeano, M. Evoked potentials, neuronal activity and stimulus intensity in the visual system. Physiology and Behavior, 196%, 3, 797-801.
- Donchin, E. Data analysis techniques in average evoked potential research. In Donchin, E. and Lindsley, D.B. (Eds.), Average Evoked Potentials. Washington, NASA, 1969.

- Donchin, E. and Cohen, L. Evoked potentials to stimuli presented to the surpressed eye in a binocular rivalry experiment. Vision Research, 1970, 10, 103-106.
- Donchin, E., Gerbrandt, L.A., Leifer, L., & Tucker, L. Is the contingent negative variation contingent on a motor response? Psychophysiology, 1972, 9, 178-188.
- Donchin, E., Gerbrandt, L.K., & Pribram, K.H. While a monkey waits: electrocortical events recorded during the foreperiod of a reaction time study. <u>Electroencephalography and Clinical Neurophysiology</u>, 1971, 31, 115-127.
- Donchin, E. and Lindsley, D.B. (Eds.) Average Evoked Potentials.

 Methods, Results, and Evaluations. Washington: NASA (U.S. Government Printing Office), 1969.
- Donchin, E. and Smith, D.B. The contingent negative variation and the late positive wave of the average evoked potential. <u>Electro-Encephalography and Clinical Neurophysiology</u>, 1970, 29, 201-203.
- Eccles, J.C. <u>The Physiology of Nerve Cells</u>. Baltimore: Johns Hopkins Press, 1957.
- Eccles, J.C., Korn, H., Taborikova, H., & Tsukahara, N. Slow potential fields generated in cerebellar cortex by mossey fiber volleys. Brain Research, 1969, 15, 276-280.
- Evarts, E.V. Pyramidal tract activity associated with a conditioned hand movement in the monkey. <u>Journal of Neurophysiology</u>, 1966, 29, 1011-1027.
- Evarts, E.V. Relations of pyramidal tract activity to force exerted during voluntary movement. <u>Journal of Neurophysiology</u>, 1968, 31, 14-27.
- Fox, S.S. and Norman, R.J. Functional congruence: an index of neural homogeniety and a new measure of brain activity. Science, 1968, 159, 1257-1259.
- Fox, S.S. and O'Brien, J.H. Duplication of evoked potential waveform by curve of probability of firing of a single cell. Science, 1965, 147, 888-890.

- Freeman, W.J. Patterns of variation in waveform of averaged evoked potentials from prepyriform cortex of cats. <u>Journal of Neuro-physiology</u>, 1968, 31, 1-13.
- Fromm, G.H. and Bond, H.W. Slow changes in the electrocorticogram and the activity of cortical neurons. <u>Electroencephalography</u> and <u>Clinical Neurophysiology</u>, 1964, 17, 520-523.
- Fromm, G.H. and Bond, H.W. The relationship between neuron activity and cortical steady potentials. <u>Electroencephalography and Clinical Neurophysiology</u>, 1967, 159-166.
- Galambos, R. A glia-neural theory of brain function. <u>Proceedings</u> of the National Academy of Science, 1961, 47, 129-136.
- Geddes, L.A. <u>Electrodes and the Measurement of Bioelectric Events</u>
 Toronto: Wiley, 1972.
- Gerstein, G.L. Neuron firing patterns and the slow potentials.

 In Computer Techniques in EEG Analysis. <u>Electroencephalography</u> and Clinical Neurophysiology, 1961. (Supplement No. 20, Pp. 68-72)
- Gilden, L. Vaughan, H.G., & Costa, L.D. Summated human EEG potentials with voluntary movement. <u>Electroencepahlography and</u> Clinical Neurophysiology, 1966, 20, 433-438.
- Grossman, R.G., Whiteside, L. & Hampton, T.L. The time course of evoked depolarizations of cortical glial cells. Brain Research, 1969, 14, 401-415.
- Gumnit, R.J. DC potential changes from auditory cortex of cat. Journal of Neurophysiology, 1960, 23, 667-675.
- Gumnit, R.J. and Grossman, R.G. Potentials evoked by sound in the auditory cortex of the cat. <u>American Journal of Physiology</u>, 1961, 200, 1219-1225.
- Haider, M. Spong, P. & Lindsley, D.B. Attention, vigilance and cortical evoked-potentials in humans. Science 1964, 145, 180-182.
- Henn, F.A., and Hamburger, A. Glial cell function: uptake of transmitter substances. <u>Proceedings of the National Academy of Science</u>, U.S.A., 1971, 68, 2686-2690.

- Hernandez-Peon, R., Scherrer, H., & Jouvet, M. Modification of activity in cochlear nucleus during "attention" in anesthetized cats. Science, 1956, 331-332.
- Hild, W. and Tasaki, I. Morphological and physiological properties of neurons and glial cells in tissue culture. <u>Journal of Neuro-physiology</u>, 1962, 25, 277-304.
- Hillyard, S. The CNV and the vertex evoked potential during signal detection: a preliminary report. In Donchin, E. and Lindsley, D.B. (Eds.), Average Evoked Potentials. Washington: NASA (U.S. Government Printing Office), 1969. Pp. 349-363.
- Hillyard, S.A. and Galambos, R. Eye movement artifact in the CNV. Electroencephalography and Clinical Neurophysiology, 1970, 28, 173-182.
- Hodgkin, A.L. <u>The Conduction of the Nervous Impulse</u>. Springfield, 111.: C.C.Thomas, 1964.
- Horst, P. <u>Factor Analysis of Data Matrices</u>. New York: Holt, Rinehart and Winston, 1965.
- Horvath, R.S. Variability of cortical auditory evoked responses. Journal of Neurophysiology, 1969, 32, 1056-1063.
- Humphrey, D.O. Re-analysis of the antidromic cortical response. II On the contribution of cell discharge and PSP's to the evoked potentials. Electroencephalography and Clinical Neurophysiology, 1968, 25, 421-442.
- Humphrey, D.R., Schmidt, E.M., & Thompson, W.D. Predicting measures of motor performance from multiple cortical spike trains. <u>Science</u>, 1970, 170, 758-762.
- Irwin, D.A., Knott, J., McAdam, D.W., and Rebert, C.S.
 Motivational determinants of the "contingent negative variation".

 <u>Electroencephalography and Clinical Neurophysiology</u>, 1966, 21,
 538-543.
- Irwin, D.A. and Rebert, C.S. Slow potential changes in cat brain during classical appetitive conditioning of jaw movements using two levels of reward. <u>Electroencephalography and Clinical Neurophysiology</u>, 1970, 28, 119-126.
- John, E.R., Herrington, R.N., & Sutton, S. Effects of visual form on the evoked response. Science, 1967, 155, 1439-1442.

- John, E.R. and Morgades, P.P. Neural correlates of conditioned responses sutdied with multiple chronically implanted moving microelectrodes. Experimental Neurology, 1969, 23, 412-425.
- John, E.R., Ruchkin, D.S., & Villegas, J. Experimental background: Signal analysis and behavioral correlates of evoked potential configurations in cats. Annals of the New York Academy of Science, 1964, 112, 362-420.
- Kern, E.B., Cody, D.T.R., & Bickford, R. Vertex response thresholds to pure tones in guinea pigs. Archives of Otolaryngology, 1969 90, 315-325.
- Kohler, W. Held, R., & O'Connell, D.N. An investigation of cortical currents. <u>Proceedings of the American Philosophical Society</u>, 1952 96, 290-330.
- Kornhuber, H.H. and Deeke, L. Hirnpotentialanderungen bei Willurbewegungen und passiven Bewegundendes Menschen:
 Bereitschaftspoteitial und reafferente Potentiale. Pflugers Archiv, 1965, 284, 1-17.
- Kornhuber, H.H., von Becker, W., Taumer, R., Hoehne, O., & ...
 Iwase, K. Cerebral potentials accompanying voluntary movements in man: readiness potential potential and reafferent potentials.

 <u>Electroencephalography and Clinical Neurophysiology</u>, 1969, 26, 439.
- Kopell, B.S., Wittner, W.K., & Warrick, G.L. The effects of stimulus differences and selective attention on the amplitude of the visual evoked potential in man. <u>Electroencephalography and Clinical Neurophysiology</u>, 1969, 26, 619-622.
- Kuffler, S.W. Neuroglial cells: physiological properties and a potassium mediated effect of neuronal activity on the glial membrane potential. <u>Proceedings of the Royal Tociety</u> Series B, 1967, 168, 1-21.
- Kuffler, S.W., Nicholls, J.G., Orkand, R.K. Physiological properties of glial cells in the CNS of amphibia. <u>Journal of Neurophysiology</u>, 1966, 29, 768-787.
- Landau, W.M. Evoked potentials. In Quarton, G.L., Melnechuck, T., Schmitt, F.O. (Eds.), <u>The Neurosciences</u>. New York: Rockefeller University Press, 1967. Pp. 469-482.
- Lasansky, A. Nervous function at the cellular level: Glia. Annual Review of Physiology, 1971, 33, 241-256.

- Low, M., Borda, R.P., & Kellaway, P. Contingent negative variation in rhesus monkeys: an EEG sign of a specific mental process. Perceptual and Motor Skills. 1966, 22, 443-446.
- Low, M.D., Borda, R.P., Frost, J.D., & Kellaway, P. Surfacenegative slow-potential shift associated with conditioning in men. Neurology, 1966, 16, 771-782.
- Magnusson, D. <u>Introduction to Test Theory</u>. Reading, Mass.: Addison-Wesley, 1966.
- Mast, T.E. Short-latency human evoked responses to clicks. Journal of Applied Physiology, 1965, 20, 725-730.
- McAdam, D. Slow potential changes recorded from human brain during learning of a temporal interval. <u>Psychonomic Science</u>, 1966, 6, 435-436.
- McAdam, D. Increases in CNS excitability during negative cortical slow potentials in man. <u>Electroencephalograph</u> and <u>Clinical</u> <u>Neurophysiology</u>, 1969, 26, 216-219.
- McAdam, D., Knott, J.R., & Ingram, W.R. Changes in EEG responses evoked by the conditioned stimulus during classical aversive conditioning in the cat. <u>Electroencephaolgraphy and Clinical Neurophysiology</u>, 1962, 14, 731-738.
- McAdam, D. and Seales, D.M. Bereitschaftspotential enhancement with increased level of motivation. <u>Electroencephalography</u> and Clinical Neurophysiology, 1969, 27, 73-75.
- McGuigan, F.J. Covert oral behavior during the silent performance of language tasks. Psychological Bulletin, 1970, 309-326.
- Miller, R.F. and Dowling, J.E. Intracellular responses of the Muller (glial) cells of the mudpuppy retina: their relation to the beta-wave of the electroretinogram. <u>Journal of Neurophysiology</u>, 1970, 33, 323-341.
- Mnukhina, R.S. Electroencephalographic analysis of the mechanism of temporary connection closure. <u>Pavlov Journal of Higher Nervous Activity</u>, 1961, 11, 359-366.
- O'Leary, J.L. and Goldring, S. Changes associated with forebrain excitation processes: D.C. potentials of the cerebral cortex. In <u>Handbook of Physiology</u> Section I: Neurophysiology, Vol. 1. Baltimore: Williams and Wilkins, 1959. Pp. 315-328.

- O'Leary, J.L. and Goldring, S. D.C. potentials of the brain. Physiological Reviews, 1964, 44, 91-125.
- Orkand, R.K., Nicholls, J.G., & Kuffler, S.W. Effect of nerve impulses on the membrane potential of glial cells in the central nervous system of amphibia. <u>Journal of Neurophysiology</u>, 1966, 26, 788-806.
- Patton, H.D. and Amassian, V.E. The pyramidal tract: its excitation and functions. In <u>Handbook of Physiology</u> Section I: Neurophysiology, Vol. 2. Baltimore: Williams and Wilkins, 1959. Pp. 837-861.
- Perry, N.W. and Childers, D.G. <u>The Human Visual Evoked Response</u>. Springfield: Thomas, 1969.
- Peters, J.F. Eye movement recording; a brief review; <u>Psychophysiology</u>, 1971, 8, 414-416.
- Rebert, C.S. The effect of reaction time feedback on reaction time and contingent negative variation. <u>Psychophysiology</u>, 1972, 9, 334-339.
- Ritter, W., Vaughan, H., & Costa, L.D. Orienting and habituation to auditory stimuli: a study of short term changes in average evoked responses. <u>Electroencephalography and Clinical Neurophysiology</u>, 25, 550-556.
- Roland, V. Steady potential phenomena of cortex. In Quarton, 6.G., Melnechuck, T., & Smith, F.O. (Eds.) The Neurosciences. New York: Rockefeller University Press, 1967. Pp. 482-495.
- Roland, V. and Goldstone, M. Appetitively conditioned and driverelated bioelectric baseline shift in cat cortex. <u>Electroencephalo-graphy and Clinical Neurophysiology</u>, 1963, 15, 474-485.
- Ruchkin, D.S., Villegas, J, & John, E.R. An analysis of average evoked potentials making use of mean square techniques.

 <u>Annals of the New York Academy of Science</u>, 1964, 115, 799-876.
- Rulon, P.J., Tiedeman, D.V., Tatsuoka, M.M., & Langmuir, C.R.

 <u>Multivariate Statistics for Personnel Classification</u>. New York:
 Wiley, 1967.
- Shaw, J.C., and Roth, M. Potential distribution analysis.

 Electroencephalography and Clinical Neurophysiology, 1955, 7, 285-292.

- Sheatz, G.G. and Chapman, R.M. Task relevance and auditory evoked responses. <u>Electroencephalography and Clinical Neurophysiology</u>, 1969, 26, 468-475.
- Somjen, G.G. Evoked sustained focal potentials and membrane potentials of neurons and of unresponsive cells of the spinal cord. Journal of Neurophysiology, 1970, 33, 563-582.
- Stevens, C.F. Neurophysiology: A Primer. New York: Wiley, 1966.
- Surwillo, W.W. The contingent negative variation: some methodological problems in the recording of shifts in steady potentials.

 <u>Psychophysiology</u>, 1971, 8, 229-235.
- Sutton, S., Tueting, P, Zubin, J., & John, E.R. Information delivery and the sensory evoked potential. <u>Science</u>, 1967, 155, 1436-1439.
- Sypert, G.W. and Ward, A.A. Unidentified neuroglia potentials during propagated seizures in neocortex. Experimental Neurology, 1971, 33, 239-255.
- Takahashi, Y., Hsu, C.S., & Honma, S. Potassium and glutamate effects on protein synthesis in isolated neuroglial cells.

 <u>Brain Research</u>, 1970, 23, 284-287.
- Tecce, J.J. Contingent negative variation (CNV) and psychological processes in man. <u>Psychological Bulletin</u>, 1972, 77, 73-108.
- Tecce, J.J. and Scheff, N.M. Attention reduction and surpressed direct-current potentials in the human brain. Science, 1969, 164, 331-333.
- Thompson, R.F., Bettinger, L.A., Birch, H., and Groves, P.M. Comparison of evoked gross and unit responses in association cortex of waking cat. Electroencephalography and Clinical Neurophysiology, 1969, 27, 146-151.
- Thurstone, L.L. <u>Multiple-Factor Analysis</u>. Chicago: University of Chicago Press, 1947.
- Timset, M., Koninckx, N., Dargent, J., Fontaine, O., & Dongier, M. Variations contingentes negatives en psychiatrie. <u>Electroencephalography and Clinical Neurophysiology</u>, 1970, 28, 41-47.

- Vaughan, H.G. The relationship of brain activity to scalp recordings of event-related potentials. In Donchin, E. and Lindsley, D.B. (Eds.) Average Evoked Potentials. Washington: NASA (U.S. Government Printing Office), 1969. Pp. 45-94.
- Vaughan, H.G., Costa, L.D., & Ritter, W. Topography of the human motor potential. <u>Electroencephalography and Clinical Neurophysiology</u>, 1968, 25, 1-10.
- Vaughan, H.G. and Ritter, W. Averaged evoked responses in vigilance and discrimination: a reassessment. Science, 1969, 164, 326-328.
- Verzeano, M. and Calma, I. Unit-activity in spindle bursts. Journal of Neurophysiology, 1954, 17, 417-428.
- Verzeano, M., Dill, R.C., Vallecalle, E., Groves, P., & Thomas, J. Evoked responses and neuronal activities in the lateral geniculate. Experientia, 1968, 24, 696-698.
- von Becker, W., Deeke, L., Hoehne, O., Iwase, K., Kornhuber, H.H., & Scheid, P. Bereitschaftspotential, motorpotential und praemotorische Positivierung der menschlichen Hirnrinde vor Willurbewegungen. Naturwissenschaften, 1968, 55, 550.
- von Becker, W.O., Hoehne, O. Iwase, K. Kornhuber, H.H., & Taumer, R. Readiness potential and evoked potentials of the human cerebral cortex associated with voluntary saccadic eye movements. Pflugers Archiv., 1968, 300, R105-R106.
- Walter, W.G., Cooper, R., Aldridge, V.J., McCallum, W.C., & Winter, A. Contingent negative variation: an electric sign of sensorimotor association and expectancy in the human brain. Nature, 1964, 203, 380-384.
- Walter, W.G., Cooper, R., Cooper, H.J., McCallum, W.C., Warren, W.J., Aldridge, V.J., Storm von Leeuwen, W., and Kamp, A. Contingent negative variation and evoked responses recorded by radiotelemetry in free-ranging subjects. Electroencephalography and Clinical Neurophysiology, 1967, 23, 197-206.
- Wasman, M., Morehead, S., Lee, H., & Roland, V. Interaction of electroocular potentials with the contingent negative variation. <u>Psychophysiology</u>, 1970, 7, 103-111.
- Waszak, M. and Obrist, W.D. Relationship of slow potential changes to response speed and motivation in man. <u>Electroencephalography</u> and <u>Clinical Neurophysiology</u>, 1969, 27, 113-120.

Watson, J.B. <u>Behaviorism</u>. Chicago: University of Chicago Press, 1963 (1930).