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GRADUATE COLLEGE

WITHIN AVERAGE VARIABILITY OF THE ACOUSTICALLY-EVOKED RESPONSE

A DISSERTATION

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the

degree of

DOCTOR OF PHILOSOPHY

BY

LAWRENCE WILLIAM KEATING

Oklahoma City, Oklahoma

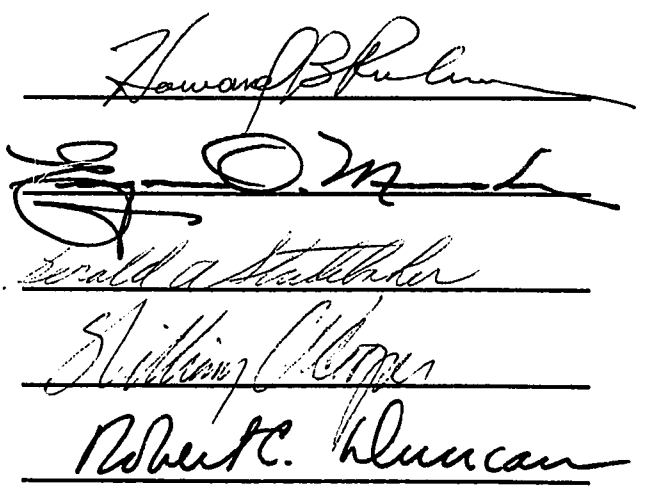
1968

WITHIN AVERAGE VARIABILITY OF THE ACOUSTICALLY-EVOKED RESPONSE

A DISSERTATION

APPROVED FOR THE DEPARTMENT OF COMMUNICATION DISORDERS

BY



ACKNOWLEDGMENT

So that the reader will know, the author expresses his sincere thanks to Dr. Howard B. Ruhm whose knowledge and insights led to this investigation and who, in addition, directed this study.

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WITHIN AVERAGE VARIABILITY OF THE ACOUSTICALLY-EVOKED RESPONSE

CHAPTER I

INTRODUCTION

One of the more common methods of studying central nervous system function is through the use of electroencephalography (EEG). This technique often provides information relative to neurological diagnoses on the basis of electroencephalic rhythms and relationships among various potentials.

P. A. Davis (30) was the first to describe observable changes in on-going electrical activity of the human brain in response to auditory stimulation. The most prominent change that she discussed was the on-effect which she described as a diphasic and sometimes triphasic wave observed most readily at the vertex. Other changes were the off-effect, similar to the on-effect but not as large, and a checking of the alpha rhythm. At about the same time, Davis et al. (25) reported similar findings and designated the acoustically-evoked response as the "K" complex. The "K" complex was described as a "multiple, diffuse, delayed, non-specific response to external sensory stimulation." This response ("K" complex) could be elicited by light and shock as well as by acoustic stimulation. These initial observations have since been confirmed and expanded by a host of other researchers.

The on-going electrical activity of the human brain varies from 10 to 200 μv (13) depending on the sites and relationships of the recording electrodes. This relatively high voltage tends to obscure in its record small voltages evoked by auditory stimuli whose amplitudes generally range from less than 1 μv to 15 μv or more. Thus, it is difficult to differentiate a single response from the large voltages contributed by the on-going activity. It is for this reason that techniques have been devised to minimize the representation of on-going activity in the record and to preserve the response.

A number of methods have been suggested to obtain a clear picture of the evoked response, the most common of which is the summing or "averaging" technique. This technique usually employs a special-purpose computer which stores in its memory the algebraic sum of many responses. The principle of averaging is based on two primary assumptions: (1) the evoked response has a fixed and consistent temporal relationship to the stimulus; and (2) changes in the on-going electroencephalic activity that are unrelated to the stimulus occur randomly. If the assumptions are valid, repeated sampling should produce a representative picture of the evoked response since the response is time-locked to the stimulus and will be added algebracially and grow proportionately to the number of samples, while the growth of the random activity will be approximately as the square root of the number of samples with repeated stimulation. The living organism is, however, in a continuous state of flux (14, 50). As a result of those "changes in state" it is rather hazardous to make the assumption that during a given sampling period the state of the organism does not change to

some degree. Rather, a more likely assumption is that the state of the organism does fluctuate during a given sampling period with the possibility of concomitant changes in responses. Any such differences among individual responses would be obscured in the record by the very nature of the averaging process.

Inherent in the procedure of obtaining an averaged acoustically-evoked response over time there are two obvious parameters of the response which can be sources of variation that may be obscured in the record: voltage amplitude and voltage latency. It is possible to construct hypothetical sets of individual responses which possess different changes in amplitude over time and yet yield the same average. In Figure 1 a hypothetical average is shown for two such sets during which there is considerable voltage variation in one condition relative to the other. Condition 1 shows less amplitude variation than does Condition 2. Yet, when averaged, these differences are obscured and the two sets yield the same result. An observer who examines only these averages is forced to decide that there was no difference between the two sets when, in fact, considerable difference existed.

A second source of variability is shown in Figure 2 where, under one set (Condition 1) an average (1a) consisted of individual responses (1b) that changed only in amplitude. Under a second set (Condition 2) individual responses shifted only in latency (2b) resulting in an average (2a) that would be interpreted as a lack of responsivity from the organism (discounting the dc level) when, in fact, the individual responses were as large as before but the overlapping of their time courses resulted in cancellation when averaged.

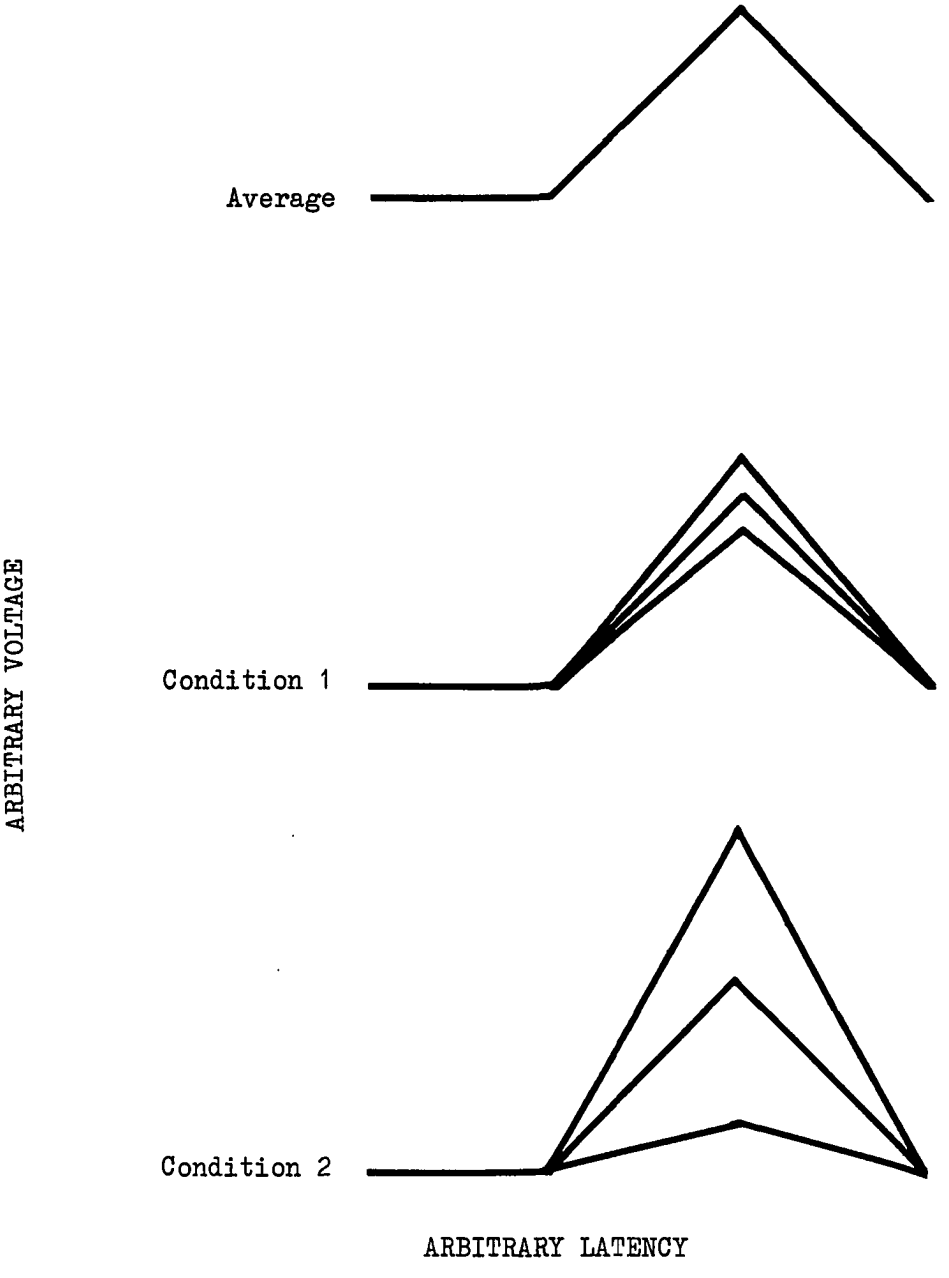


Figure 1.--Schematic diagram of a hypothetical average illustrating the ambiguity of the average relative to the amplitude of the evoked response. Condition 1: amplitude relatively constant. Condition 2: amplitude relatively variable.

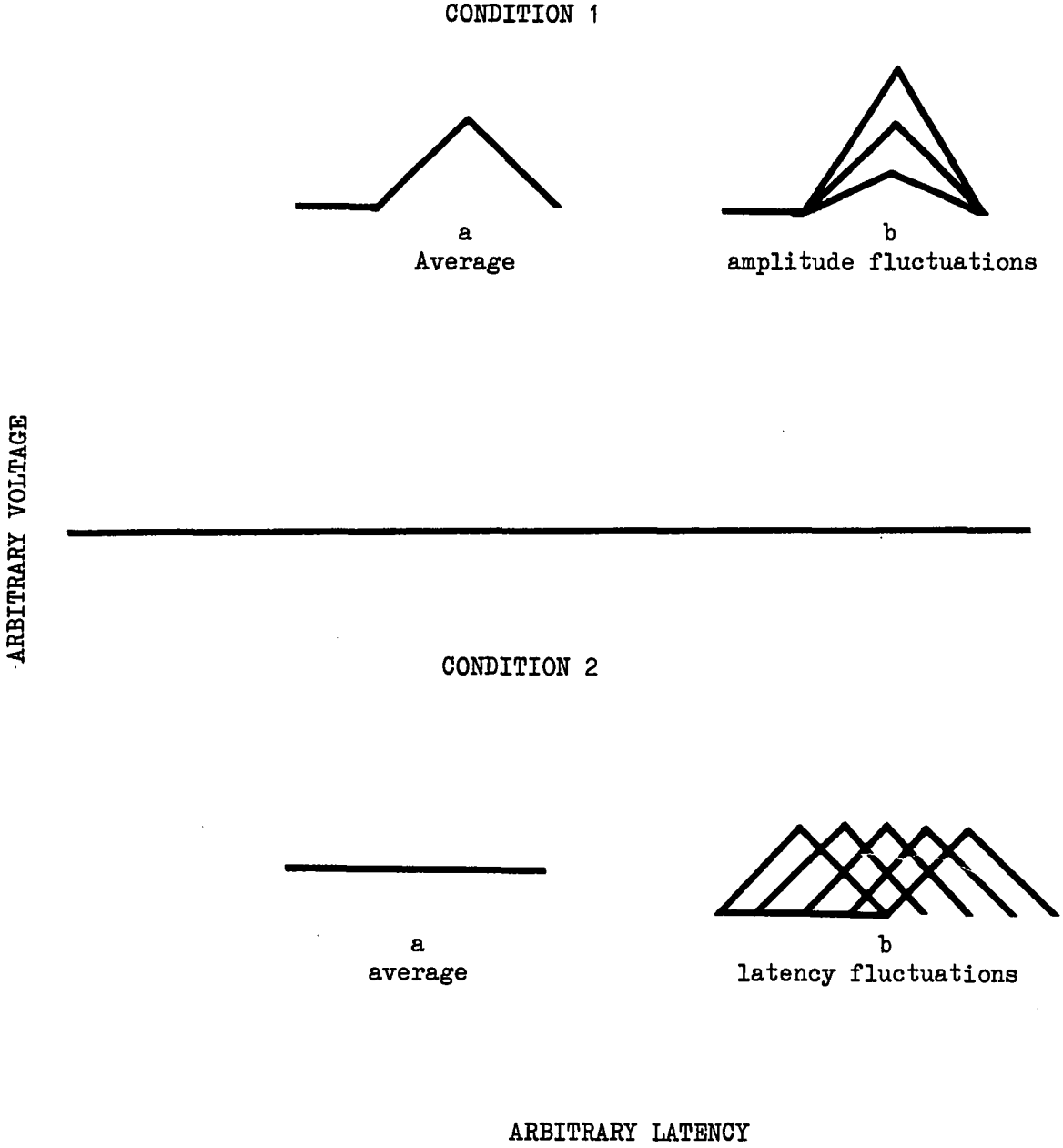


Figure 2.--Schematic diagram of a hypothetical average illustrating the ambiguity of the average relative to the latency of the evoked response. Condition 1: constant latency. Condition 2: variable latency.

It is obvious that interpretations of evoked responses that are drawn from their averages can lead to inappropriate conclusions. In order to enhance the value of such information, it is desirable to estimate the variability of the various components of the average evoked response. If, for example, an average is obtained from a set consisting of numerous repeated stimuli and the relative variability of its various components are small compared to a similar set with large variability, a conclusion that successive stimulation produced a somewhat different neurological effect might be in order. Furthermore, it has been suggested by various authors (12, 47) that certain components of the average evoked potential are contributed by specific neurological substrates in the central nervous system of the organism. It is reasonable to think that the relative lability of some components may be suggestive of their origins or areas of mediation.

As was shown earlier (Figures 1 and 2) there are two sources in the evoked potential which can cause variation in the voltages generated from the nervous system: latency and amplitude. It would be advantageous to measure the variability and to parcel out those portions due to latency and those to amplitude. However, due to the nature of the procedure used in obtaining the data it is impossible to make this determination since all measurements represent voltage over specific points in time. Variations in either voltage or latency would be reflected as differences in voltage in the average response. Accordingly, the present study will be concerned with the less ambitious task of determining the relative variability of the acoustically-evoked response regardless of the source of variation, be it voltage amplitude or

voltage latency. Specifically, this study was initiated to investigate the relative variability among several time-locked sample points which compositely make up the acoustically-evoked response and to specify the following: (1) the variance of selected components; (2) the most variable component in the acoustically-evoked response (AER); (3) the least variable component in the AER; (4) the effect of various tasks on the variance of the individual components; (5) the most variable point in the AER; (6) the least variable point in the AER; and (7) differences between responses to left and right ear stimulation under each of the above questions.

A discussion of pertinent literature as it relates to the variability of the evoked response is presented in the following chapter.

CHAPTER II

REVIEW OF THE LITERATURE

The purpose of this study was to investigate the variability of the average acoustically-evoked response. Specifically, it was designed to define a band of variance around certain specified components of the averaged response, to determine the most variable and least variable points along the averaged response, and to determine the relative variability among the major components of this average.

The following review of the literature consists, first, of a description of the acoustically-evoked response as a general orientation to the problem; then specific factors are reviewed which may influence the variability of responses evoked by auditory stimulation in humans.

Description of the Acoustically-Evoked Response

In the past decade much attention has been focused on measures of electrical potentials generated by the human brain as a possible indicant of auditory function. This interest has been generated by the observation that changes in the on-going electrical activity of the brain occur when a stimulus impinges on an intact sensory system. According to Brazier (13), Canton, in 1875, was the first to discover "persistent fluctuating potentials" of an electrical nature from the

cortex. The first actual recordings of this phenomenon were made by Berger in 1924 and reported in 1929. Since that time a multitude of researchers have expanded on these original findings and have increased our knowledge many fold relative to evoked responsivity of the central nervous system.

Chang (16) describes an evoked potential as ". . . the detectable electrical change of any part of the brain in response to deliberate stimulation of a peripheral sense organ, a sensory nerve, a point on the sensory pathway or any related structure of the sensory system." The acoustically-evoked potential was first described by P. A. Davis (30) in an experiment in which she used auditory, visual, and tactile stimuli to evoke responses. She and H. Davis et al. (25) have labeled the response as the "K" complex. Because observations in recording these potentials from various parts of the head have consistently shown them to be larger and more easily detectable at or near the vertex, the response was conveniently termed the "vertex" or "V potential" by Bancaud et al. (5).

The V potential has been described in some detail by various authors (2, 5, 25, 27, 29, 30, 33, 42, 89, 106, 107). There is general agreement that it is generated rather diffusely in the cerebral cortex and is non-specific for sensory modality since it can be elicited by stimulation of visual, tactile, olfactory, or auditory senses. It is thought by some (10, 19, 55, 65) that early potentials (to 50 msec) are mediated myogenically from the "sono-motor" reflex and have so distinguished these faster components from the V potential. Ruhm et al. (92) have presented evidence, obtained in simultaneous recordings from

the scalp and the cerebral cortex, that these early components exist even at low (dB) stimulation levels and concluded that they result from cochleoneurogenic mediation under these circumstances. There is agreement that the later components (50 to 500 msec) are not myogenic but do arise, either directly or indirectly, from neural pathways connecting the cochlea and the cortex.

Components of the Acoustically-Evoked Response

Davis et al. (25) described the evoked potential as:

> . . . multiple, diffuse, delayed, and a non-specific response to external stimulation. Often a fast wave of frequencies 8-16 cps are superimposed on a series of slow waves but either may occur independently.

It is generally agreed that the response is polyphasic in nature having fairly consistent latencies (24, 33, 75, 82). These waves are either positive or negative with respect to one another and are conventionally labeled P_1, N_1, P_2, and N_2. Those components labeled with P indicate that the potential is more positive; those with N, more negative relative to the slope of the waveform. Earlier and later components are sometimes observed but are not observed as frequently nor with as much consistency as are the previously-mentioned components.

When measured through electrodes on the intact human scalp the peak voltage of the on-going electroencephalic activity ranges from about 10 to 50 μv. Rarely does it exceed 200 μv (13). Although these on-going voltages are extremely small, they are relatively large when compared with the amplitude of the activity evoked by auditory stimulation. Keidel (57) showed responses obtained from the intact human scalp to have a peak to peak amplitude of 7.5 to 9.0 μv; however, he failed to

mention the intensity of the acoustic signals used for eliciting these responses. Davis (23) reported typical peak to peak voltages of 10 to 20μv in response to click stimulation of 70 to 80 dB and further claimed that in some rare individuals the response could be as large as 100μv. Zerlin and Davis (117) have obtained single evoked response data on one subject to 80-dB tone pips which averaged about 100μv. They comment that responses of this magnitude are unusual.

Price et al. (75) have reported that the N_1-P_2 component ranges from .5 to 28μv (N_1, 70-95 msec; P_2, 134-178 msec) with a mean of approximately 10μv. McCandless and Best (63) have measured the amplitudes of individual components as follows: P_1 (approximately 56 msec), 9μv; N_1 (approximately 96 msec), 21μv; and P_2 (approximately 163 msec), 39 μv. Rose and Ruhm (82) have presented findings showing that the amplitude of the evoked response increases with increases in signal intensity, but generally is in the 4 to 6μv range. Their findings agree with others (1, 2, 19, 23, 25, 30, 43, 44, 66, 72, 76, 87, 88, 100, 108, 111, 115).

Techniques for Extracting the Evoked Response

The fact that the amplitudes of evoked potentials are much smaller than are those of on-going electroencephalic activity makes these potentials difficult to distinguish in recording or observing this phenomenon.

In an attempt to measure evoked electrical potentials, Dawson (31) developed a technique whereby a number of evoked response configurations, recorded under similar conditions, are superimposed on a single display, thus allowing visual inspection of the points at which the

potentials are evoked with regular latency. Using this superimposition technique it is possible to detect small time-locked events in the presence of much larger background activity (1, 94, 100).

Rosenblith (85, 87) and his colleagues were some of the first to use a computer for averaging the evoked response. A procedure was developed in which the on-going activity was algebraically summed for a specified length of time after presentation of the stimuli. Since the report of this work, numerous other researchers have, through the use of high-speed computers, applied the summation technique with auditory as well as other types of sensory stimuli.

The summation method for detecting evoked responses is based on the assumptions that the response will occur at a constant latency relative to the signal and that the response is physiologically independent from the background activity. The technique of extracting the responses from the background activity involves time-locking the signal to the analysis period of the computer, thus, by algebraically adding the voltage that occurs at discrete temporal points after numerous repetitive stimuli, the average amplitude of the response increases in direct proportion to the number of samples (N) while the background activity, randomly related in time to the reference point, increases approximately as the square root of N.

Since the evoked responses are time-locked and their polarities and latencies are assumed to be relatively invariable, as algebraic summation occurs, the small response which is buried in the noise will grow with each repetition until it can be readily observed against the baseline of the summed random on-going activity. Thus, algebraic

adding causes the on-going activity to be small relative to the evoked response in the computer's memory and the average of the responses became readily discernable.

Upon obtaining an average of the amplitudes and latencies of the various components of the evoked response, it is of interest to determine the variability of these components. The variability of components derives importance from the possibilities that components may differ in relative lability, that normal and pathological nervous systems may differ in this respect, or that different psychophysiological states of the subject may cause more or less stability of the response.

Variability of the Evoked Response

Previous researchers have investigated the variability of the evoked response to sensory stimulation. The vast majority of these investigations have been carried out by comparing average against average under similar and different conditions of intrinsic and extrinsic factors. Few reports are available concerning "within average" variability. The remainder of this discussion will be limited to the literature as it pertains to the variability of the acoustically-evoked response and to selected studies of the variability of responses evoked through other sensory modalities.

Variability of the Average

Of great importance in obtaining evoked responses are variables which, during an averaging procedure, may alter or influence the waveform of the evoked response in one manner or another. These variables can originate intrinsically (within the individual) or extrinsically

(outside the individual). Intrinsic factors are somewhat controllable but much less so than extrinsic factors since it is much easier to control and monitor one's external environment than his internal environment.

Effects of Attention of the Acoustically-Evoked Response

The attentiveness of a human being to a stimulus can be thought of as lying on a continuum from maximum to deep sleep when there is no conscious recognition that the stimulus exists. The following studies show that the evoked response is affected by the state of awareness of the subject under examination.

P. A. Davis (30) noticed that the on-effect became more predictable as the subject progresses from alertness to sleep. She did not, however, indicate that during the alert stage the subject was attending to the stimuli. Likewise, Davis et al. (25) presented evidence that amplitude of the "on-effect" increases progressively as the subject progresses from a state of alertness to drowsiness and then sleep.

Roth et al. (90) investigated the "K-complex" in 80 psychological patients and 15 normals during consciousness, sleep, and under barbituate anesthesia. No differences were found between the groups but all groups showed an increased latency and duration for the "K-complex" during sleep and barbituate anesthesia.

Derbyshire and McDermott (34) studied subjects under three conditions: sleep, repose with inattention to stimuli, and repose interrupted by the task of closing a key in response to acoustic stimulation. They found that the latency of the "K-complex" at minimal intensities was shortest during sleep and longest for the key-closing condition. The reverse was true for higher intensity levels. They

also noted a greater percentage of observable responses under the "key-closing" condition. These results suggest that attention directed toward the stimulus renders the response more readily indentifiable. Kohler and Adams (58), using several visual stimuli and having the subject attend closely to a specific stimulus, found that not only was the evoked response enhanced to the stimuli receiving attention but responses were evoked at lower stimulus intensities as well.

Williams and his co-workers (114) undertook a study to determine the characteristics of the acoustically-evoked response during the various stages of sleep. They used three subjects who were stimulated by clicks via a speaker one foot from the head at 85 dB re .0002 dynes/cm^2. Clicks were presented at a rate of one per two seconds throughout the night. At the onset of sleep, low voltage patterns with irregular frequency were seen and termed Stage 1 sleep. There was then a slow progression through Stages 2 and 3 which is denoted by 14 cycle per second spindling and slower activity to Stage 4 where trains of 4 to 7 cycle-per-second waves appear in the record. The subject then traversed back through Stage 3 and Stage 2 to Stage 1 where rapid eye movements (REM) accompany the emerging low voltages. It was reported that during a normal night of sleep the above-described cycle is repeated every $1\frac{1}{2}$ to 2 hours. Their results showed that as the subject shifts from the waking pattern through the stages there is a consistent change in the waveform. The amplitude of P_1 increases but not so markedly as does the amplitude of N_2, while N_1 and P_2 decrease. A third positive wave emerges in Stage 2 and reaches relatively high amplitude in Stages 3 and 4. Responses during Stage 1 (REM) appear to be quite

like that seen in the waking state and at the beginning of sleep except the amplitudes of the components are smaller, especially P_2 and N_2. In addition, the peak-to-peak amplitude of P_2-N_2 is smaller in Stage 1 (REM) than in any other stage.

These data then tend to confirm Geisler's (43) observation that the K-component of the evoked response changes as the subject goes to sleep. Also these results indicate that the waveform changes across different stages of sleep and that the form of the evoked response is closely allied with the EEG stage of sleep reflected by the background electroencephalic activity.

Williams et al. (113) confirmed the above findings and in addition compared two waking conditions (reading and counting) with the various stages of sleep. The response obtained during reading corresponded closely to that seen during Stage 1 (REM). Since Stage 1 (REM) is associated with the dreaming stage of sleep, they hypothesized these similarities in waveform might be explained on the basis of similar CNS organization under the two conditions.

Weitzman and Kreman (109) averaged evoked responses to 50- to 60-dB clicks on ten normal subjects during different stages of sleep and reported the following latencies: N_1, 40-70 msec; P_1, 80-130 msec; N_2, 140-230 msec; P_2, 250-400 msec; and, N_3, 650-950 msec. Their results showed the latencies of N_1, P_1, and N_2 to remain the same throughout all stages of sleep. This finding is different than that of Ornitz and his associates (71) who, in a later study, found all components of the acoustically-evoked response to be shortened in latency during Stage 1 (REM) sleep relative to Stage 2 sleep. Their (109) finding that during

REM the evoked response was smaller and closely resembled the awake response agrees with results reported by others (71, 113, 114). The above studies would seem to indicate quite decisively that evoked potentials do change depending on the sleeping state of the subject and that they are also unlike the evoked responses elicited during waking stages. There is also evidence to show that differences exist in the evoked response during waking stages, as well as in sleeping states.

Most researchers dealing with evoked potentials agree with a certain amount of variation in average evoked responses can be attributed to changes in the observer's state of attentiveness.

Haider and his co-authors (52) investigated changes in visual-evoked potentials averaged during a vigilance task requiring visual discrimination and a response. The task required the subject to respond by pressing a key to dim light flashes which were periodically interspersed among more numerous brighter flashes that required no response. The results of this experiment indicated that reduced attentiveness in a vigilance task is paralleled by corresponding reductions in the amplitude of visual-evoked responses measured at the N_1-P_2 component. During the decline of attentiveness (80 per cent correct at the beginning of task to 50 per cent correct at the end of the task) the amplitude of the evoked potential decreased from 13μv to 10μv and, in addition, the mean latency increased from 155 to 165 msec.

Davis (22) observed that the evoked response was significantly enhanced in amplitude when the subject was required to judge the loudness of filtered tone pips as opposed to a reading condition or a condition which merely required the subject to signal after each stimulus.

These amplitude measurements were obtained between the largest negative peak at about 100 msec (N_1) and the largest positive peak at about 150 to 200 msec (P_2). Filtered tone pips were delivered at regular 2.5 second intervals with four pips constituting a cycle. The first pip was low pitched (600 Hz) and served as a warning followed by the second, third, and fourth, all of a higher pitch but equally intense except for a 3-dB increment or decrement that was added or subtracted from the third pip of the cycle in the "decision" trial. The responses to each pip of the cycle were averaged separately in the computer's memory. There were no differences in the evoked response between the control runs (reading) and the runs in which the subject was required to push a button each time after the third pip of the cycle. The amplitude of the average evoked response was increased "well beyond the usual range of variation" in the trial where the subject was instructed to press the button only when the second pip sounded louder than the first. It was impossible to determine the degree of change from the data presented.

Spong and his associates (96) investigated evoked potentials to clicks and flashes from subjects performing visual or auditory discrimination tasks. Three methods of inducing attention were employed: (1) a vigilance condition which required the subject to attend to one modality by attempting to respond to decrements in that stimulus while ignoring the stimuli presented alternately to the other sense; (2) a key-pressing condition in which the subject pressed a key following each stimulus of a given modality while ignoring the alternately-presented stimuli to the other sense; and (3) a counting condition whereby the subject was expected to count all stimuli of a given modality and,

again, ignore the stimuli presented to the other sense. Each sense was used as the "attend to modality" half the time. Their results led them to conclude that when an attentive set is established by requiring subjects to perform such discrimination tasks to every stimulus, the amplitude of the evoked response increases as the subject's attention becomes more closely focused on the eliciting stimulus.

Satterfield (93) studied evoked responses to click and shock stimuli in an attempt to determine if attending to a stimulus produces an enhancement of the response to that stimulus. Among 47 subjects he found 7 in which attending to the stimulus produced decreased amplitudes of the response to either click or shock, while in the remaining 40, attention caused an increase in response amplitude.

Gross and his co-workers (50) presented data which seems to confirm the hypothesis that directing the subject to count clicks produces greater amplitudes and shorter duration of the acoustically-evoked response than obtained during reading. The latencies of the components of the response were not decreased.

Donchin and Cohen (37) attempted to determine the effects of attention when subjects viewed a 50-msec flash superimposed on a fluctuating background. In one condition the subject was required to ignore the background and respond to the flash; for the other condition he responded to the background and ignored the flash. It was found that the stimulus to which attention was directed elicited an average evoked potential with a considerably enhanced late positive component having a latency peak of between 250 and 300 msec. These results are consistent with the findings of others (21, 99).

The above-mentioned studies and several others (38, 104, 110) confirm the hypothesis that the psychological state of the organism, when viewed on a continuum from vigilance to sleep, has an effect on the average acoustically-evoked response. Not all the results of the various studies are in agreement; however, it does appear that generally the evoked response is enhanced during tasks which require the subject to perform difficult perceptual discriminations and that it is more readily observable as a subject passes from awake stages to sleep. It has also been shown that individual components of the total response are affected differentially by these various states of attentiveness.

Effects of Age on the Acoustically-Evoked Response

According to Barnet and Goodwin (7) the largest component of the acoustically-evoked potential (P_2) of infants occurs at about 270 msec after the stimulus and appears to have much likeness to the "vertex" potential described by Davis et al. (25). In the adult the response latency for this component is in the range of 160 to 200 msec (26, 82, 114). Ellingson (41) traced the decreasing latency of response in infants from the newborn to several months old. Although the stimuli which he used were light flashes, the response appears to have many of the same properties as those elicited by clicks (17).

In the infant the stimulus intensity appears to be the most important parameter of the signal in eliciting an evoked response (7) whereas in adults this relationship, although seen, seems to be less acute (23). According to Barnet and Goodwin (7), factors associated with level of consciousness influence the response of babies to a lesser degree than adults because EEG patterns in the infant are relatively

undeveloped and are thus not as strikingly different from waking patterns.

Goodman and his co-authors (49) studied evoked potentials in newborns two to ten days old utilizing tone pips and found latencies as follows: P_1, 75 msec; N_1, 125 msec; P_2, 250 msec; and N_2, 450 msec. These latencies are longer than typical adult latencies. Suzuki et al. (101) found that at low sensation levels latencies of responses evoked from children between 11 and 15 years of age were longer than those obtained from adults. However, when the sensation level was raised to 50 to 60 dB no such differences were found.

Price and his associates (75), in studying a group of 160 subjects between 10 and 83 years of age, found frequency of occurrence and latency of responses to be unaffected by age; however, amplitude of the N_1-P_2 component was affected in that it decreased with increasing age to a low for the 40 to 49 year old group and then increased to a maximum at the 70+ age group. The authors suggested that seemingly large amplitudes for the older group might be explained on the basis of the recruitment phenomenon. McCandless and Best (63) reported that evoked response patterns of children are similar within a given age group but differ from other age groups and adults.

One of the most systematic studies of the effects of age has been carried out by Dustman and Beck (40) utilizing visually-evoked potentials of 125 subjects ranging in age from one month to 81 years. They reported that amplitudes of waves in the first 250 msec of the response changed dramatically with age. In recording from the occiput, there was a rapid increase of amplitude to a maximum in the 5 to 6 year old group with mean amplitudes about twice as large as the means of some

older age groups. With children 7 years and older, there was a rapid decrease in amplitude until age 13 to 14 at which time an abrupt increase in amplitude appeared. The amplitude seemed to stabilize at about age 16.

Effects of Drugs on the Acoustically-Evoked Response

The effects of different types of drugs on the acoustically-evoked response has not been fully investigated; yet, consistent effects have been observed to certain drugs. According to Price (73), ". . . it seems reasonable that any medication which affects the on-going EEG activity is likely to affect the evoked response."

Certain components of the evoked response, particularly those beyond 100 msec are adversely affected when the subject is injected with pentothal (74, 77). These effects are not noted when pentobarbital sodium is used as the sedating agent (74, 102). According to Price (73), ". . . the late negative component of the response at about 300 msec (N_2) which is often enhanced by certain stages of natural sleep is enhanced in an almost identical manner by pentobarbital sodium induced sleep." Rapin and Graziani (77) showed good evoked responses using chlorpromazine with children. Cody and his associates (20) have concluded that chloral hydrate does not appreciably alter the evoked response to acoustic stimuli.

Shagass and his associates (95) have shown data which indicates no significant affect from amobarbital, methadrine, and lysergic acid diethylamide on the evoked responses obtained on psychiatric patients with shock delivered to the ulnar nerve.

The sparse literature relative to drug effects on the

acoustically-evoked response suggests a need for a well-controlled systematic study covering the effects of drugs that are likely to be administered to the human organism.

Effects of Pathology on the Acoustically-Evoked Response

Rapin (76), in an attempt to determine any adverse effects on the evoked potential introduced by CNS disorders, investigated the acoustically-evoked response of 26 normal adults and children and 50 children with communication disorders, ages $6\frac{1}{2}$ to $12\frac{1}{2}$ years. She found peak latencies of the components of the response to be essentially the same for both groups. In addition, only one child out of the seventy-six that were studied showed no response. Rapin and Graziani (77) presented evoked-response data obtained by visual and auditory stimulation of sixty-one infants who were normal, brain damaged, or deaf. These data did not show differences between groups in the configuration of the response evoked by either auditory or visual stimuli.

Hogan and Graham (54) studied the waveform characteristics of the acoustically-evoked response obtained from a group of retarded adults and concluded that the waveforms were similar to those obtained from normal adults (59). They did, however, find that a lower percentage of retardates responded at 30-dB SL than did the normal group. They interpreted this as an adverse effect of abnormal EEG patterns rather than a reduced responsiveness of the subject.

Nodar (68) observed the acoustically-evoked response from ten mentally retarded adults and compared these responses to five normal adults. He found no major differences between the two groups and concluded that there is no difference between the acoustically-evoked

responses of normal and mentally retarded adults. Barnet and Lodge (8) have shown, in comparing normal and mongoloid infants' (age 0 to 14 months) acoustically-evoked responses, that the amplitude of the P_2-N_3 component of mongoloids is significantly greater than that of normals. The mean amplitude for fifteen mongoloid subjects was 50.2 μv as compared to 28.8 μv for the fifteen normal subjects matched by age to the mongoloid group. No differences were found in comparing the latency of the P_2 component of these same groups.

In observations carried out on thirty normal subjects and four patients with known lesions of the audiovestibular system, Bickford and his co-authors (11) concluded that varying degrees of myogenic contamination accompany the acoustically-evoked response, particularly the early (50 msec and less) componentry. Ruhm and his associates (92) have shown data, simultaneously recorded from the cortex and skin electrodes near the vertex, which indicate that responses evoked by moderate-level signals are neurogenic rather than myogenic.

Gross and his co-workers (51) studied the effects of delirium tremens on acoustically-evoked responses under the hypothesis that there was an acute disturbance of the response to acoustic stimulation. They found that when the patient was hallucinating the evoked potentials were consistently different from those obtained when hallucinations were not present. This observation applied particularly to the first major negative deflection (25-30 msec). During hallucination the component appeared lower in amplitude and broader than when there was no hallucination.

Straumanis and his associates (98) attempted to determine

whether average visual-evoked responses differed in normals and in chronic brain syndrome patients. Three groups were studied: (1) patients with severe cognitive impairment due to cerebral arteriosclerosis; (2) normal healthy controls of the same age as group 1; and (3) healthy young adults. Group 1's responses differed from those of age-matched controls in components occurring after 100 msec; latencies were prolonged and rhythmic after-activity was reduced. Differences associated with age were found primarily in components occurring before 100 msec, with prolonged latencies and greater amplitude in older subjects.

Brazier (14) was the first to explore evoked responses to electrical stimili via electrodes implanted in several human brain structures (hippocampus, hippocampal gyrus, amygdala, and temporal tip). These patients (seven cases of temporal lobe epilepsy) had electrodes placed for diagnostic and theraputic purposes and were made available for evoked response studies. Her results showed that impulses initiated in the amygdala evoke a response in the ipsilateral hippocampal gyrus and also reach the temporal tip. Repetitive stimulations of the amygdala were shown to lead to recruitment of response in the ipsilateral hippocampus and hippocampal sites. In addition to the electrical stimulation, visual stimulation was employed and it was found to evoke a response in the hippocampus and hippocampal gyrus but not in the amygdala. Ruhm (91) in a study presently being conducted has been successful in obtaining bilateral responses from chronically-implanted electrodes in the amygdala.

Effects of Stimulus Parameters on the Acoustically-Evoked Response

There are several means by which the acoustic signals used to elicit acoustically-evoked responses can be altered under controlled conditions. As a result of highly sophisticated and precision instrumentation a number of different variations have been investigated in an attempt to determine the effect on the evoked response of altering certain acoustic parameters.

Type of stimuli. Acoustic signals that appear to evoke the most readily recognizable responses are clicks and pure tones or noise bursts possessing rapid onsets (84). It has been hypothesized (4, 72, 111) on the basis of emperical observation that clicks are more effective than pure tones in evoking responses because of the shorter rise-time possessed by a click. Perl (72) has observed that clicks possess equal energy over a broad frequency range and thus excite a greater number of peripheral units which result in more cortical activity than discrete pure tones and, hence, supports the above hypothesis.

Davis (22) has studied evoked responses to filtered clicks, thus achieving different frequency spectrums and has more recently pointed out (24) that although generally good agreement exists between voluntary pure-tone thresholds and evoked responses to filtered clicks, the agreement between the two is not so good for individuals with sharply sloping hearing losses. Thus, he advocates the use of brief pure-tone bursts with 20-msec rise and decay times. McCandless and Best (64) feel that pure-tone stimuli should be utilized, thus enabling one to obtain information relative to the sensitivity of the auditory mechanism at different frequencies.

It has been shown (35, 39) that speech stimuli evoke auditory responses and is felt (35) that their use enables one to observe the effect of semantic content of the stimulus.

It is clear, therefore, that responses are evoked not only by discrete signals, but also by signal parameter increments.

Intensity. It appears that the stimulus parameter of intensity exerts the greatest influence on the auditory response. As a general rule, it has been shown that increases in the intensity of the auditory stimulus are accompanied by increases of the evoked response (29, 32, 34, 46, 59, 66, 82, 100, 116). Moore and Rose (67) have presented data indicating that the amplitude of the evoked response does not increase at levels above 70- to 90-dB SL.

Suzuki and Asawa (100) and Suzuki and Taguchi (102) utilizing pure tones have shown that as the stimulus intensity increases the number of positive evoked responses increases. Others (44, 61, 62, 82) have studied amplitude effects relative to the intensity of click stimuli and have reported larger responses at higher intensities. Rapin et al. (78) observed an irregular and non-uniform increase in the N_1-P_2 component in response to increased click stimulus intensity; however, the amplitude of the response to pure-tone stimuli was more regular and seemingly related to changes in intensity. Suzuki and Taguchi (102) observed the amplitude of the N_1-P_2 component to follow a linear function when plotted relative to dB above subjective threshold. Davis and Yoshie (28) also observed a linear function when the N_1-P_2 component of the vertex response to filtered clicks was plotted relative to sensation level (SL), at least for lower SL's. According to Moore and Rose (67),

the amplitude does not increase above 70 to 90 dB, hence, it might be that this relationship may become asymptotic at higher levels.

Abe (1) showed a linear relationship between the intensity of a 1000-Hz tone and a positive 150-msec component plotted re dB of intensity. Davis and Zerlin (29) observed a linear slope of 0.24 re .0002 dyne/cm^2 when the voltage of the vertex potential to a 2400-Hz tone pip was against dB in SL. They reported, however, that there was considerable intersubject variability. Likewise, McCandless and Best (64) reported that the relationship between the amplitude of the response and the stimulus intensity was evident for some subjects but not for all. In another study (63) they found that subjects performed similarly on test-retest but different from subject to subject. Teas (103) found that an increase in signal intensity which results in an increase in response amplitude was dependent upon the subject under observation. He observed that while the amplitude of the response varied with the signal intensity the on-going activity seemed to vary more with the state of the subject.

Rapin (76) found that as click intensity was reduced the amplitude of both early and late components were decreased. Further, components prior to 65 msec disappeared before components with longer latencies. McCandless and Best (64) have observed in testing young normal adults that the amplitudes of P_1 (50 to 70 msec) and N_2 (250 to 300 msec) do not vary as a function of intensity and that N_1 (100 to 130 msec) and P_2 (175 to 230 msec) do appear to vary with intensity. Rose and Ruhm (83) reported a significant increase in the number of observable evoked responses as a function of increased SL, the higher

SL yielding the greater number of responses.

The latency as well as the amplitude of the acoustically-evoked response is affected as a result of intensity changes in the acoustic signal. In general, latency can be expected to decrease as intensity is increased (1, 60). Davis and his associates (27) observed the latencies of the P_1 (60 msec), N_1 (110 msec) and P_2 (190 msec) components to tone pips at an intensity range from 20 to 75 dB (ASA 1951) between the frequencies of 300 and 4800 Hz and determined that the latencies were significantly increased at low stimulation levels.

Rose and Ruhm (82) studied three components of the response evoked by clicks at 20- to 40-dB SL. The components observed were the most negative point occurring after 40 msec and subsequent most positive and negative points. The latencies of the first and second components decreased significantly in latency when the signal intensity was increased; however, the latency of component three did not decrease. The conclusion which was drawn from this data was that later components are affected less by intensity changes than are earlier components.

Rapin and her co-workers (76, 78, 79) have studied the latencies of evoked responses to clicks and pure tones of varying intensities and found that the latencies of the N_1 and P_2 components remain constant for clicks but at 50-dB SL the latency for tone bursts is 10 to 15 msec longer than for clicks of the same intensity. The observed difference was progressively greater with decreasing intensity. The authors chose to explain these differences on the basis of Bekesy's (1960) theory of temporal integration and Daughty and Garner's (1947) threshold of tonality experiments. The results of this experiment may be open to question,

however, since the authors do not report taking into account the inherent 10- to 15-msec delay of the electronic switch which was incorporated into their instrumentation for the purpose of delivering pure-tone stimuli to the subjects.

Rise time. Acoustic stimuli that appear to produce the most readily recognizable evoked responses are clicks and pure tones or noise bursts possessing rapid rise times (84). Goldstein and Kiang (45), in studying the effects of various stimulus rise times from evoked responses recorded from the cortex of cats, found that as the stimulus rise time was increased the detectability of the evoked response tended to decrease. They reported that with rise times of 50 msec and more doubtful or negative results were obtained, while with rise times of 25 msec or less responses were readily observed. Although rapid rise times were better than slow rise times clicks were observed to be superior to either. Others (32, 34, 35) have reported that stimuli with rise times slower than clicks do not evoke responses as effectively. Rosenblith (84) has advanced the hypothesis that biological systems respond primarily to change and that they rapidly adapt to steady state stimulation. In this light, clicks and other acoustic signals with rapid onsets impose upon the auditory system an acoustic change which creates an excellent climate for neural response. Goldstein and Kiang (45) theorized that gross electrodes placed on the scalp record summed electrical activity of many neurons. The change in amplitude of the response is determined not only by the number of participating neural units but also by the synchrony of the unit discharges. The abrupt nature of clicks and fast rise time tones or noise bursts activates

large number of peripheral neural units more or less simultaneously. These can be expected to result in more concentrated neural bursts throughout the auditory system and be reflected in large responses recorded with scalp electrodes. Slow rise times, on the other hand, more closely approximate steady state conditions and may result in evoked responses which are much different than these evoked by abrupt stimuli. To investigate this they used clicks and slow rise time voice bursts against a background of masking noise reasoning that the masking noise would cause some of the neural units to be in refraction and, hence, modify the ability of the total neural population to respond synchronously. They found that for noise bursts the masking noise exceeded the intensity of the burst by 10 dB before evoked responses became undetectable while with clicks the response disappeared when the masking level was 10 dB below that of the stimulus. They explained these results on the basis of neural synchrony. The abruptness and brevity of the clicks allowed peripheral units only one chance to respond and the masking noise eliminated too many neural units to achieve synchronous activity. Due to the greater duration of the noise bursts, neural units that were unable to fire at the onset were activated later during stimulation, but still with enough synchrony to produce a detectable response.

Perl and her co-authors (72) performed Fourier analyses of acoustic clicks generated through an earphone and found that the acoustic energy is distributed about equally throughout the frequency range of 200 to 3000 Hz with an additional energy peak at 6000 Hz. Thus, when utilizing click stimuli broad areas of the basilar membrane will be stimulated causing greater numbers of neural units to fire relative

to pure-tone stimulation.

McCandless and Best (64) have indicated that pure tones are as satisfactory as click stimuli in eliciting evoked responses and have an advantage in that they provide more information relative to auditory functioning across frequency. They found, in comparing three frequencies (250, 1000, and 4000 Hz), that there were essentially no differences in latencies and overall pattern. This finding has also been reported by Davis and his associates (27).

Lamb and Graham (59) found that when using rise times of 10, 49, and 95 msec with 1K Hz tones at intensity levels of 10, 20, and 30 dB that the number of responses decreased with increases in signal rise time and that clicks were far more likely to evoke responses than a 1000-Hz pure-tone signal with a 95-msec rise time. Onishi and Davis (70) utilized computer-generated tone bursts with linear rise times of 3 or 30 msec and plateaus from 0 to 300 msec at four intensities. They found with a 30-msec rise time the amplitude (N_1-P_1) and the latency (N_1) to be unaffected by increasing the plateau from 0 to 300 msec. With a 3-msec rise time and 30-msec plateau, the amplitudes were significantly reduced. The latency at 45 dB (ISO) was slightly prolonged compared to 65 and 85 dB (ISO) and significantly so at 25 dB (ISO).

Ruhm and Jansen (91) found that when measured from an extrapolated behavioral threshold the latency of the P_2 peak seems not to vary systematically as a function of signal rise time over the range of 10 to 1000 msec.

Duration. Cody and his co-authors (19) have reported recording adequate vertex acoustically-evoked responses utilizing tone pulses

having durations of 4 to 64 msec. Derbyshire and his associates (35) have reported the same for tones of 0.5 to 4.0 seconds. McCandless and Best (64) found no difference in the waveforms of responses evoked by 30-msec and 700-msec duration tones. They do, however, report the occurrence of an off-response to the longer duration (700 msec) stimulus. Rose and Malone (81) have observed off-responses to occur at stimulus cessation to tones ranging from 800 to 1500 msec.

Davis and Zerlin (29) studied evoked responses to 1200-Hz tone bursts that ranged from 2 to 320 msec and observed no systematic differences in the voltages of the N_1-P_2 component. They report that the 20-msec tone bursts sometimes evoked the largest responses and hypothesized that this may be due to an interaction of the on- and off-effects. They were able to observe separate off-effects to stimulus duration as low as 500 msec while Rose and Malone (81) observed this phenomenon to stimuli with durations as short as 800 msec. The amplitude of the off-effect appears to grow larger as the duration of the tone is increased (29). These observations lead one to suspect, then, that the evoked response is initiated primarily by the sudden change in the environment to which the organism has become adapted rather than to the duration of the stimulus.

Rose and his associates (80) have recently studied the acoustically-evoked response to determine if it would show a change in amplitude or latency (40 to 300 msec) with changes in stimulus durations. They found no significant change in amplitude or latency as a function of the duration of stimuli 30, 50, 100, and 300 msec at two sensation levels of 20 and 40 dB.

Norkus and Mills (69) studied acoustically-evoked responses to 1K-Hz tone pulses of .03 to 10 seconds duration at intensities up to 60-dB SL. They found responses to be optimum with durations of 0.1 to 0.5 second while durations less than 0.1 second yielded less than optimum responses when the signal was 30-dB SL or less.

Frequency. The majority of reports relative to the effect of frequency on the acoustically-evoked response state that this parameter of the auditory stimulus does not affect the waveform of the response (27, 29, 64, 81, 102).

Observations contrary to this notion have been made by Rapin and her co-authors (78) who found that the N_1-P_2 component was enhanced greatest by increases in stimulus intensity to 1000-Hz tones, less to 250-Hz tones, and least to 6000-Hz tones. Vetter and Horwath (105) state that low frequency tones are more effective in evoking a response in sleeping subjects.

Recently, Antinoro and Skinner (3) reported a study in which they compared acoustically-evoked responses to different frequencies (250 to 8000 Hz at octave intervals). Stimuli were presented randomly at two levels, 30- and 60-dB SL in one experiment and at 30- and 60-dB SPL at 1000 Hz (equal loudness) in another. Their results, an overall decrease in amplitude of about 70 per cent from 250 to 8000 Hz under the equal SL condition and a 50 per cent drop under the equal loudness condition led them to conclude that the amplitude of acoustically-evoked responses could not be meaningful compared across frequencies in efforts to extrapolate to threshold.

Number of Stimuli

The averaging or summing process utilized by researchers in obtaining evoked responses is a statement to the effect that individual responses are often difficult or impossible to detect in the midst of on-going EEG activity. This implies that in order to see an evoked response one must in many instances use identical repetitive stimuli and observe the average of many responses.

A number of investigators have attacked the question of the number of stimuli that are needed in order to evoke an observable response. McCandless and Best (63) reported that reliable responses were obtained from adult subjects to 50-click stimuli. Cody and Bickford (19) found that generally 25 to 50 stimulus presentations were needed in order to obtain an average response. Rapin (76, 78) has stated that when stimuli are presented well above threshold 30 to 50 presentations are sufficient and that the number required is increased as the level of the stimuli are lowered toward threshold. At 20-dB SL she has found it necessary in some instances to present as many as 400 stimuli.

Because of the repetitive nature of the evoking stimuli (in averaging the evoked response) there have been some questions raised concerning habituation. Observations of this phenomena have been reported in the literature (60, 103, 107). Rose and Ruhm (82) have failed to substantiate habituation in obtaining evoked responses to 500-click stimuli.

The data of Davis and Zerlin (29) suggest that habituation does not constitute a significant problem in obtaining observable evoked responses, as does the data of Leibman and Graham (60). Brazier (14)

has found by obtaining an average composed of many summations and breaking it down into several averages of fewer serial summations that the waveform of the evoked potential does change. Price (72) has demonstrated habituation to 500-Hz tones of 60-msec duration presented at 88-dB SPL at a rate of one per two seconds over a period of two hours. Although habituation was not a smooth predictable function, there was a definite trend toward lower amplitudes of the response with time. The most dramatic reduction of amplitudes occurred between 20 and 30 minutes from onset of the test. This data also indicates that habituation is not a significant factor in obtaining an observable response.

Rate of Stimulus Presentation

Perl and her associates (72) were the first to notice that the number of discernable evoked responses were decreased when auditory stimuli were presented too rapidly; i.e., faster than 10 per second. Abe (1) found that with an inter-stimulus interval (ISI) of 0.75 second a reduced response could be evoked, whereas with ISI's of 0.5 second and less no response was observed. McCandless and Best (63) studied repetition rates of .5 per second to 3.0 per second and found that with stimulus rates of 2.0 per second and faster response components with latencies occurring after 80 msec were reduced in amplitude and shifted in latency. Response components were very little affected with repetition rates of .5 per second or less. Goodman et al. (49) and Appleby et al. (4) have stated that ISI's of 10 to 15 seconds are associated with the largest response in infants.

Rapin (75) and Derbyshire and his associates (34) advocate the

use of slow (10 to 30 second ISI) irregular stimulus presentations to obtain greatest response amplitude. Rapin uses rates which average 1 per 3 seconds although the stimuli are presented irregularly. Walker and his co-authors (106) state that components occurring after 70 to 80 msec can be driven by repetition rates as high as 100 per second while Cody and Bickford (19) report that satisfactory vertex responses can be obtained with rates of 1 per 2 seconds.

Davis and his associates (27) have stated that the P_1, N_1, and P_2 components show no latency shifts until ISI's are shorter than 0.5 second. With shorter ISI's the waveform is altered with concomitant reduction in amplitudes which might be explained on the basis of superimposition of relatively later components of the preceeding response on the early components of the succeeding response.

Davis' group (27) has stated that at least 10 seconds are required for full recovery of the vertex potential; however, their decision was based on an interpolated curve.

Recording Method

It appears that some confusion exists as to the exact nomenclature of various recording montages. Brazier (13) has said that bipolar recordings are taken between members of a pair of electrodes each of which is over active brain tissue and that monopolar recordings are made when one electrode of the pair is positioned over active brain tissue and the other over a relatively inactive point. She states, however, that such an inactive point cannot be found on the head. Goff and associates (48) have stated that there is no such thing as monopolar recordings; rather, that the term monopolar only means that the reference

electrode is located over an area which in the ideal case is isoelectric with regard to the evoked potential sought. They found in attempting to standardize recording sites and to locate an "ideal" site for placement of the reference electrode that the earlobe was probably the most isoelectric point on the head, particularly for enhancement of comparability of cross-modality studies. Thus, they have recommended using as the reference in future evoked response studies the earlobe contralateral to the stimulus.

Recording Site

Derbyshire and his associates (26) have indicated that the recording site which produces the greatest amplitudes for acoustically-evoked responses is 3 cm lateral to the midline in the plane existing between the subject's ears with the reference electrode on the contralateral earlobe or mastoid. Similarly, McCandless and Best (64) have found greater magnitude of responses when recordings are made from the central area of the interaural plane.

Davis (30), Abe (1), and Davis and Zerlin (29) have stated that the acoustically-evoked response is greatest when recorded from the vertex. The latter state that nasion, mastoid, or earlobe is acceptable for reference electrode placement. Many others have confirmed that maximal amplitude of the evoked response is obtained from vertex or near vertex recordings (4, 19, 75, 78, 103).

Variability of Individual Responses Within the Average

The previous section of this review has indicated the complex relationship that exists among the average acoustically-evoked response,

the various parameters of the psychophysiological state of the individual, the physical parameters of the stimulating signal, and the techniques utilized to obtain the information which gives rise to the average response. It is evident that tremendous interest has been generated relative to the study of acoustically-evoked responses as reflected by the rapidly increasing literature pertaining to response averages. It would seem that in addition to this information about means it would be useful to have concomitant knowledge about the individual responses from which the average response is derived. The need to average responses is brought about by the low "response" to "noise" ratio in electroencephalic activity. This need necessitates that repetitive identical stimuli, each one an entity unto itself, be administered to evoke successive responses for averaging. As a result of this requirement the effects of individual responses may be lost or altered by the repetition or averaging process and thus, the average may in some instances be non-representative of the true nature of individual responses at any period in time. (See Figure 2.) There are two main reasons why this information has not as yet been readily available. The first is because the evoked response is so small relative to the on-going activity with which it is present that it cannot be easily observed. Thus, if one cannot observe it, its variability cannot be measured. Secondly, we are limited instrumentally. All measurements are made in voltages at predetermined points in time after the onset of the stimulus. This second point can be illustrated by constructing a hypothetical situation. In order to make it meaningful it is useful to think of the evoked response as a series of instantaneous voltages

which maintain their respective time relationships with the onset of the stimulus.

The hypothetical situation is set up as follows: evoked responses are recorded and fed to a summing computer. The responses may vary to one degree or another, and furthermore, these variations can be either changes only in the amplitude of the evoked response or changes only in the latency of the evoked response. (See Figure 2.)

Latency and Amplitude Fluctuations

As was discussed earlier, it is not possible to definitively separate peak variance due to latency shift from that caused by amplitude changes. Some rationale can, however, be applied to such data in an attempt to achieve an estimated parcellation of the effects of these two factors. If we assume for a moment that all response variance that is displayed by a single component is due to latency shift, it might be expected that the variance measured at a point where the averaged component crosses the zero baseline would be a direct reflection of the total variance. Conversely, if the variance were entirely attributable to amplitude changes (no latency shift), one would expect to see less variance exhibited at the point of zero baseline crossing of the average; however, provided the response slope remained relatively constant across samples, changes in amplitude would be reflected as apparent latency shifts of the component peak. It should be understood, however, that this apparent latency variability is related to the peak amplitude rather than to any change in the latency of substrate firing. The key relationship in this approach is that which exists between the peak and baseline variances. The assumptions underlying this line of reasoning

are that (1) the slope of the component in question remains relatively constant across samples and (2) that amplitude changes are not reflected at the baseline. To the extent that these assumptions are valid, an approximate parcellation might be made between variability due to latency shift and that due to amplitude change.

Because of the rigorous assumptions which must be made in order to separate latency variance from amplitude variance, this study was designed to concern itself with the less ambitious but somewhat more sure-footed task of determining the variability of selected peak components.

Indications of Within-Average Variability

P. A. Davis (30) in her initial report of observations of the acoustically-evoked responses in the on-going EEG activity alluded to the variable nature of the response by indicating that the response could not always be observed in the written record. Others have also made this observation (27, 29, 36, 70, 116).

Davis and Zerlin (29) and Davis et al. (27) make reference to the variability within the average by indicating that variability is an outstanding characteristic of the evoked response even when the response is observed as an average.

Zerlin and Davis (116) investigated the variability of individual responses in a single subject who yielded unusually large evoked responses to auditory stimulation. The peak-to-peak voltage of the N_1-P_2 component measured from the vertex averaged about 100 μv to 80-dB HL (ISO 1964) tone pips at 10-second intervals. Responses were also measured to 70-dB tone pips. The responses at 80 dB were on the average

larger than at 70 dB, but with much overlap. They found that the voltages of these single responses varied widely in approximately Gaussian distributions and were unable to show a cyclic pattern of variation.

Burns and Melzack (15) presented examples of data obtained from waking dogs and sleeping humans to show that large changes in the amplitude and waveform of the evoked response occur in the time needed to obtain an average. Brazier (14) has shown that when an average response, recorded from a cat, to 480 light flashes were broken down into averages of each successive 60 stimuli the components of the response underwent a serial change as the animal became used to the stimulus.

Barlow (6) has utilized electrically generated signals and contrasted them with biological signals in an attempt to explore the limitations imposed upon the computer technique of studying evoked responses. Barlow contends from the results of his study that one cannot talk about the variance of an evoked response if the evoked response has been obtained from physiological potentials in the midst of background activity. The variance in this instance is due to the response as well as to the background activity. He further states that it may be impossible to sort out the amount of variance due to the response from the total variance and, hence, one must speak of the variance of the analyzed signal rather than the variance of the evoked response.

Justification for the Present Study

The findings regarding the summing of EEG activity synchronized with sensory stimulation, specifically auditory stimulation, indicates

that the summation method is a reasonable approach to the study of the functioning of the central auditory mechanism. Thus far, the primary focus of attention has been directed toward determinations of the differences in the averaged waveforms both across and within subjects under different and similar experimental situations. Some authors have suggested while others have shown that the responsivity of the CNS can and does change during the time course of obtaining an average evoked response. Hence, interpretations based on average waveforms might lead to erroneous conclusions based upon this type of data. If, however, one were able to specify the variability of the signals which compositely yield the average the experimenter could at least be either more or less confident of future observations on the acoustically-evoked response. Certainly interpretation would be enhanced if it were possible to specify these parameters.

The present study was designed to investigate the intra-average variability of the acoustically-evoked response under different conditions which have been shown to cause response variations. A description of the experimental apparatus, subjects, and procedures utilized to obtain this information is outlined in detail in the following chapter.

CHAPTER III

PROCEDURE AND INSTRUMENTATION

This experiment was designed to investigate the relative variability of the numerous time-locked sample points which compositely make up the average acoustically-evoked response. The specific goals of this study are found in Chapter I.

The electroencephalic activity was picked up by electrodes attached to the scalp of the subject and fed to the instrumentation described in later sections of this chapter. The method of obtaining this activity was via a bipolar technique, with one electrode positioned on the vertex and referenced to parallel electrodes at both mastoids. The ground electrode was placed on the subject's forehead. The subject's scalp was measured with a metric rule to determine the exact location of the vertex, i.e., the intersection of a line from nasion to inion and a line connecting the two preauricular notches. Prior to placing the electrodes, the proper area of placement for each electrode was cleaned with alcohol. The electrodes, filled with an electrolytic suspension (Sanborn EKG Sol), were then held in place by collodion over a gauze patch.

Subjects

Data were collected from eight normal-hearing adult males. All subjects had a negative history of ear pathology and a negative neuro-

logical history. They were required to demonstrate normal hearing by passing a pure-tone hearing-screening test at 25-dB HL (re ISO, 1964) at octave frequencies from 125 through 8000 Hz. All subjects were either graduate students or staff members of the Department of Communication Disorders, University of Oklahoma Medical Center. Each subject was tested on a pilot run to determine if his responsivity was large and clear. Some prospective subjects were excluded from the experimental group on the basis of either a non-definitive response or excessive myogenic noise which made the response difficult to see. Once a subject was chosen for inclusion in the experimental group the responses obtained during an experimental session, regardless of how definitive the response happened to be, were used as data.

Apparatus

All testing was accomplished in a sound-isolated two-room suite at the Speech and Hearing Center, University of Oklahoma Medical Center.

Screening Apparatus

A pure-tone audiometer (Beltone, Model 15C) feeding either of two air-conduction receivers (Telephonics, TDH 39, ten ohm) located in a sound-isolated two-room suite was used in the hearing screening tests. The acoustic output of the receivers was measured with an audiometric calibration unit (Western Electric 640AA Condensor Microphone; Western Electrical-Acoustical Laboratory, Inc., Condensor Microphone Complement, Type D/E) at two week intervals by the clinical staff at the University of Oklahoma Medical Center Speech and Hearing Clinic. The corrections

for dial readings (posted on the audiometer) were incorporated in the measurements thus compensating for any deficiencies in acoustic output of either earphone.

Experimental Test Apparatus

The experimental test apparatus used in this experiment is represented by a block diagram shown in Figures 3 and 4. For purposes of clarity, data paths are depicted by solid lines and trigger and signal paths by interrupted lines. It is obvious from these figures that many of the same components were utilized in both phases.

Stimulus presentation apparatus. A central programming unit (Grason-Stadler Modular Programming Series 1200) was preset to control the temporal sequence of the stimuli which were delivered to the subject. The programming unit triggered either of two waveform generators (Tektronix, Type 162) which, in turn, triggered either of two pulse generators (Tektronix, Type 161). The outputs of the pulse generators, a click of 0.1 msec duration was delivered to either of two matched insert receivers (Radio Ear, M92, 200 ohm) through either of two attenuators (Hewlett-Packard, Model 350 AR) loaded with minimum loss pads to match impedances. The acoustic output of the receivers was checked to insure that 0.1 msec voltage increments produced identical waveforms. The receiver was mounted in a 2-cc coupler with a 640 AA microphone as a pick-up source which in turn was connected to a Western Electric Acoustic Laboratory (Type D/E) calibration unit. The output of the calibration unit was fed to a storage oscilloscope (Tektronix, 564) on which the waveform was visually inspected and measured. This procedure revealed that the click was a highly damped sinusoid whose initial

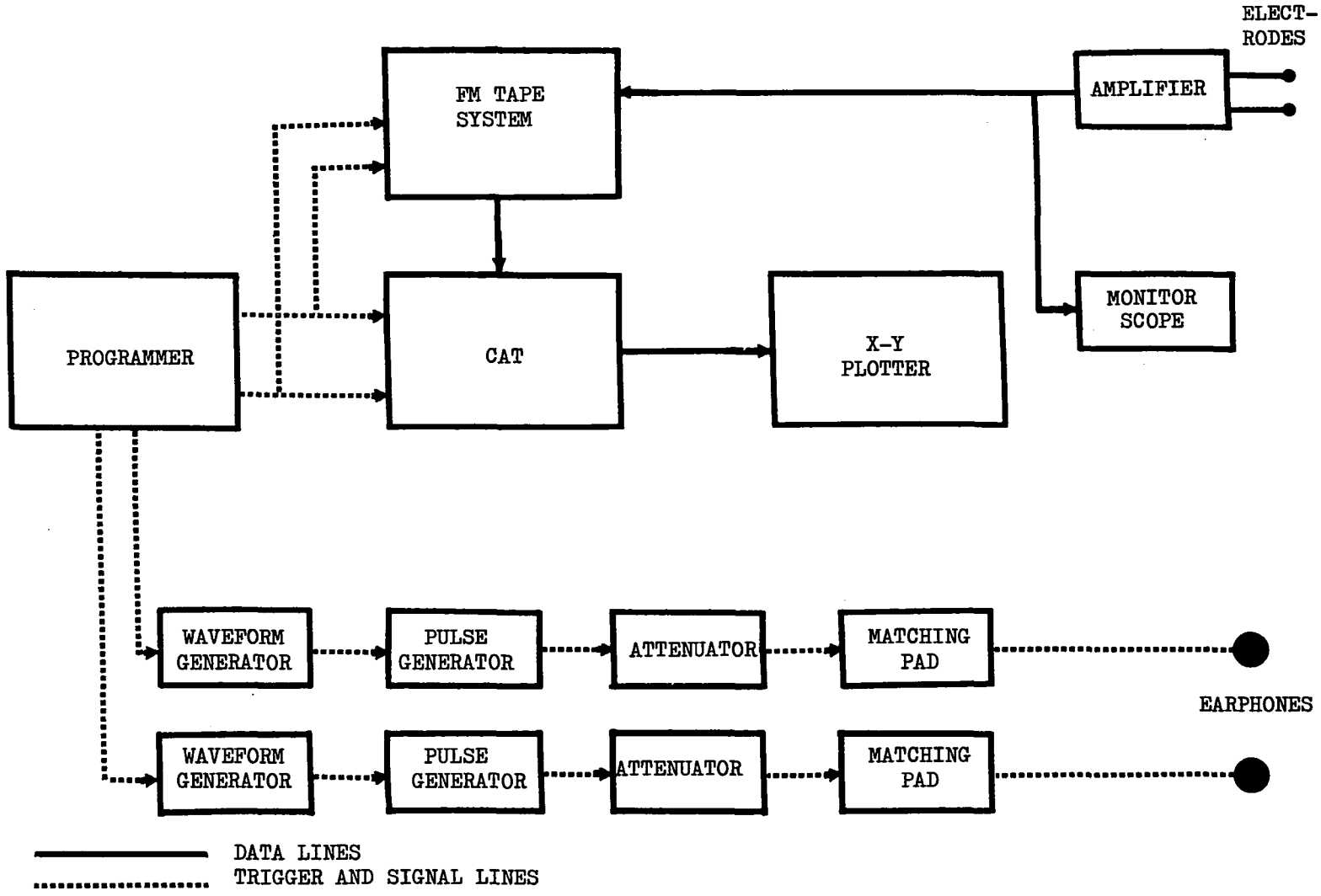


Figure 3.--Simplified block diagram of recording and signal presentation apparatus.

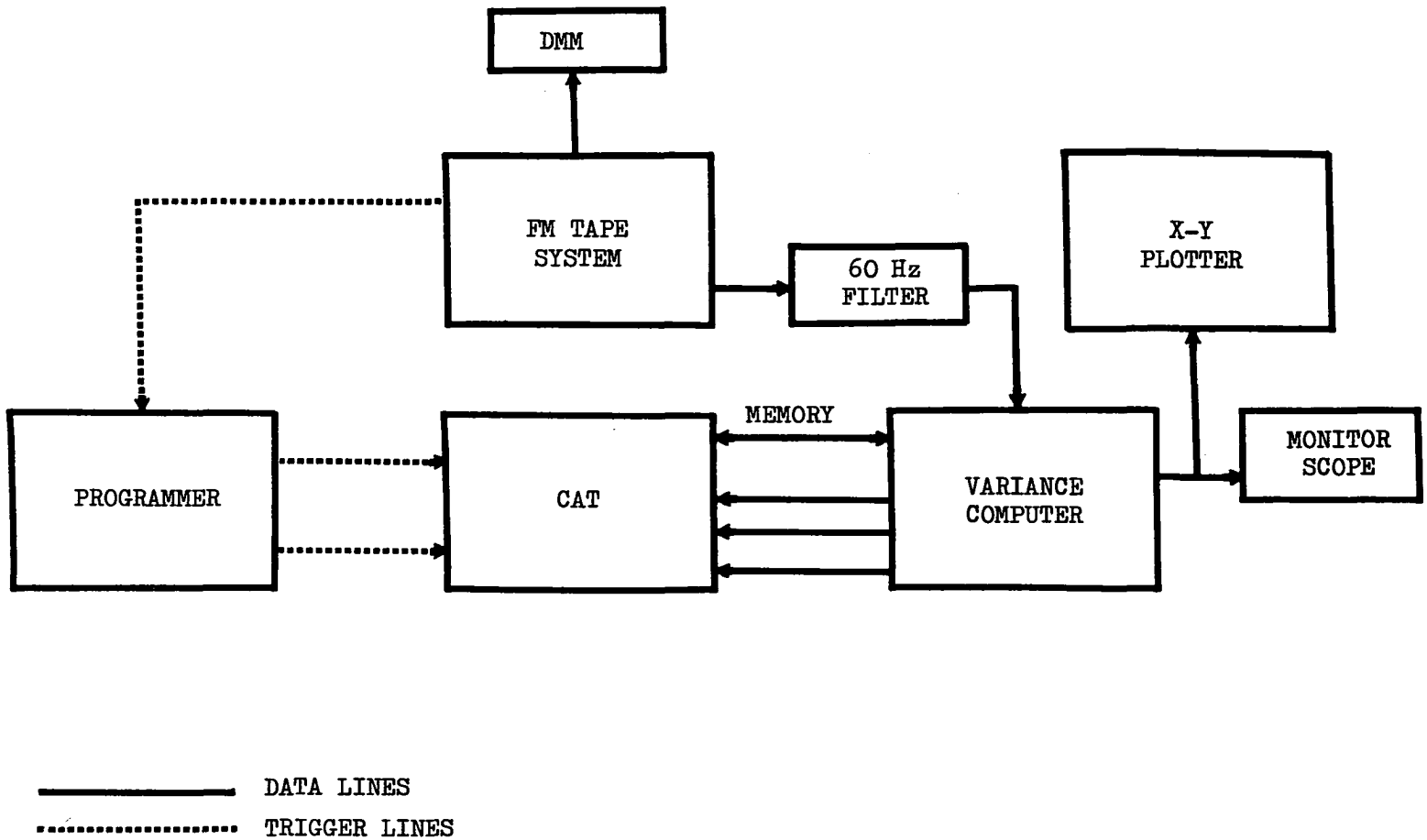


Figure 4.--Simplified block diagram of data analysis apparatus.

components followed a positive-negative-positive fluctuation. The initial peak was approximately 0.5 msec from the initiation point and was damped to within ten per cent of zero baseline within 1.75 msec from initiation. Clicks were presented at a rate of one per four seconds alternately to each of the two receivers.

Recording apparatus. A schematic diagram of the recording apparatus is shown in Figure 3. The electrodes which conducted the electrical activity from the scalp were connected through a patch board to an A.C. amplifier (Grass, P5 Series, Type P511) set at a gain of 25,000. This amplified activity was fed to an FM tape system (TMC, Model 700/1400) where it was stored on one track of the magnetic tape. The signal was paralleled to a monitor oscilloscope (Tektronix, Model 561-A with Type 3A74 four trace amplifier and Type 67 base) to enable the experimenter to visually monitor the on-going EEG activity.

An averaging computer [TMC, Computer of Average Transients, Model 400-A (CAT)] was pre-set to average the incoming data taken from a monitor output of the FM tape system. During the recording phase of the experiment the CAT was used only as a monitor. Responses to stimuli delivered to the right ear were monitored on one channel of the CAT and responses evoked by stimulation of the left ear on another channel. The programming unit was adjusted to deliver a trigger to start the CAT sweep concurrently with each stimulus presentation, except in the discriminating condition. During this condition 10-dB increments were presented alternately to the two ears on every eighth, tenth or twelfth presentation. When the intensity increment was delivered to the ear the CAT did not accumulate data and triggers were not put on tape.

This was accomplished by the experimenter's manual activation of a switch which blocked the trigger to the CAT and the CAT trigger units but allowed the signal to pass to the subject's earphone.

Triggers were recorded on two tape channels through two sync trigger units (TMC, Model 1056-A) which were utilized in retreving, separately, left and right ear responses in the analysis phase of the study.

Analysis apparatus. A schematic diagram of the analysis apparatus is shown in Figure 4. The tape-recorded data were fed through a 60-Hz notch filter (A.P. Circuit Corporation, APN-60) to the variance computer (Bio-Data, Model 204) which shares the memory of the CAT.

The trigger pulses recorded on one track of the magnetic tape were utilized to trigger the CAT in the accumulation of responses to right ear stimulation, while those on a second track provided synchronization for responses to left ear stimulation. These triggers corresponded temporally to stimulus presentation. Silent control runs were analyzed by time-locking the computer sweep to a temporal point between stimuli (3 seconds following the trigger). This approach provided an estimate of responsivity to a "no-signal" condition within the time domain of each experimental treatment. Responses to left ear stimulation, right ear stimulation and silent control periods for each ear were recovered separately. This required that the tape be run four times and the appropriate pulse was utilized to trigger the CAT.

At the termination of each condition, the data which were stored in the computer's memory were normalized to the true average level. The contents of the memory were then non-destructively fed to

the X-Y plotter (Moseley, Model 2DR-2) and the average and variance at each of the 200 addresses were traced on calibrated graph paper. The calibration signals supplied internally by the variance computer to the first eight addresses of the memory were aligned in the normalizing process with the use of a digital multimeter (Darcy, Model 440).

Calibration Procedures

The procedure used to calibrate the various components of the system are recorded in APPENDIX A. Calibration procedures for the CAT, variance computer, and X-Y plotter were supplied by the respective manufacturers.

Experimental Procedures

This study was a repeated experiment of identical format performed with eight subjects. The experimental conditions under which subjects were tested were: (Q) subject sitting, eyes open; (C) subject sitting, eyes open, counting; (D) subject sitting, eyes open, discriminating 10-dB increments; (R) subject sitting, reading. Each condition consisted of 100 stimuli to the right ear and 100 stimuli to the left ear. A balanced order of treatments as shown in Table 1 was used on the assumption that such ordering would reduce the influence of possible sequence effects on the data.

Each experimental session consumed approximately three hours which included electrode placement, threshold measurement, rest periods, and the four experimental conditions.

The equipment was calibrated before and checked after each experimental session. If, for any reason, the equipment was found to

TABLE 1

BALANCED ORDER OF TREATMENTS FOR EIGHT SUBJECTS

		Ear							
		Left				Right			
		Subject				Subject			
Trial	Run	1 5	2 6	3 7	4 8	1 5	2 6	3 7	4 8
1	1	Q Q	C C	D D	R R	Q Q	C C	D D	R R
	2	C C	Q Q	R R	D D	C C	Q Q	R R	D D
	3	D D	R R	Q Q	C C	D D	R R	Q Q	C C
	4	R R	D D	C C	Q Q	R R	D D	C C	Q Q
2	1	Q Q	C C	D D	R R	Q Q	C C	D D	R R
	2	C C	Q Q	R R	D D	C C	Q Q	R R	D D
	3	D D	R R	Q Q	C C	D D	R R	Q Q	C C
	4	R R	D D	C C	Q Q	R R	D D	C C	Q Q

Q, quiet; C, counting; D, discriminating; R, reading.

be out of calibration or otherwise malfunctioning following or during an experimental session, the data was discarded and the session repeated at a later time. Data was discarded and re-collected for four of the sixteen sessions because of instrumentation failures.

After the subject was seated and the electrodes positioned, instructions were given for the determination of sensitivity threshold for clicks. Threshold was then measured using an ascending technique in 2-dB steps and was defined as the lowest stimulus intensity to which a subject responded at least two out of three times. The stimulus intensity was increased 50 dB re threshold for all presentations.

The subjects were instructed relative to their task immediately before each experimental condition was initiated. For the quiet condition, subjects were asked to pay no particular attention to the clicks and, above all, not to count the clicks. For the counting condition, subjects were requested to count the total number of clicks heard in

both right and left ears. For the discriminating condition, the subjects were instructed to push a button whenever they detected a stimulus which was louder than the preceding stimulus. For the reading condition subjects were given small pamphlets concerning medical research to read. Subjects were requested to sit as quietly as possible throughout each of the experimental conditions. Rest periods of from ten to twenty minutes were given between each condition at which times the subject was allowed to get out of the chair and move about freely.

Experimental and sampling errors were mitigated by using the following controls: (1) appropriate calibration checks before and after each experimental session; (2) the use of young normal-hearing adult subjects; (3) a pilot evoked response session to ensure observable evoked responsivity; (4) short experimental runs to reduce fatigue; (5) experimental sessions in the morning hours to ensure maximal alertness; (6) alternation of stimuli between left and right ears to obtain bilateral data during the same time domain; and (7) the use of shielded suites to reduce the possibility of stray electrical interference.

Measurement of the Data

After the data had been recorded, played back, and read out on the X-Y plotter the evoked responses were visually inspected in order to determine which components were to be analyzed. Upon inspection of the data, it seemed that there were nine such components which appeared with sufficient frequency to warrant consideration. These nine components were as follows:

Component 1 - first prominent negativity after 30 msec.

Component 2 - first prominent positivity after component 1.

Component 3 - second prominent positivity after component 1.

Component 4 - largest prominent negativity after component 3.

Component 5 - first prominent positivity after component 4.

Component 6 - second prominent positivity after component 4.

Component 7 - third prominent positivity after component 4.

Component 8 - first prominent negativity after component 7.

Component 9 - first prominent positivity after component 8.

The positivity or negativity of the components described above represents the polarity of the component with respect to the slope which immediately precedes it and does not represent polarity relative to the zero baseline. In many instances some of the components did not appear in the readout from a particular experimental session. In these cases no data measurements were made and data for that component was left blank to be estimated later if need be. Final decisions were made by a researcher who has had nine years of experience in investigating the complexities of the acoustically-evoked response.

After the response components were selected for analysis they were measured for latency, amplitude relative to a zero baseline, and the amplitude of the variance at corresponding latencies. (See APPENDIX A for calculation of amplification for system and readout.) The measured data for each subject under each test and retest condition can be found in APPENDIX B.

When statistical analysis was undertaken it was decided to specify all amplitude measurements relative to the second negative peak at approximately 90 msec. It was thought that this procedure would facilitate the interpretations of the differences between amplitudes of the

various components since the mean amplitudes of each component would be referenced to a fixed point and not be confounded by the shifting baseline. Furthermore, this component was prominent in all tracings and was usually the most negative point on the waveform of the average evoked response.

Classification of Components

It was decided that the traditional method of classifying the components of the evoked response (P_1-N_1-P_2-N_2) would not be utilized in this study for two main reasons: (1) identification of more consistant peaks than are denoted by the traditional system were made and (2) such a classification makes cross-modality comparison of results difficult in the event that future research of other sensory modality stimulation yields similar data. The method of classification of components is that recommended by Goff and his associates (48) (Figure 5). The total time of the evoked response is divided into six latency ranges. All components within each latency range are designated by the same number with a prefix which denotes polarity and a suffix denoting the order of appearance within a specific latency range. Thus, if components change, either within or across subjects or within or across sensory modality stimulation, only the suffix need be changed. For example, the N4b component is negative and is the second component to appear in the fourth latency range.

Since the method of classifying components has only been recently introduced, the component classification in this study represents an effort of agreement with Goff and his colleagues (48).

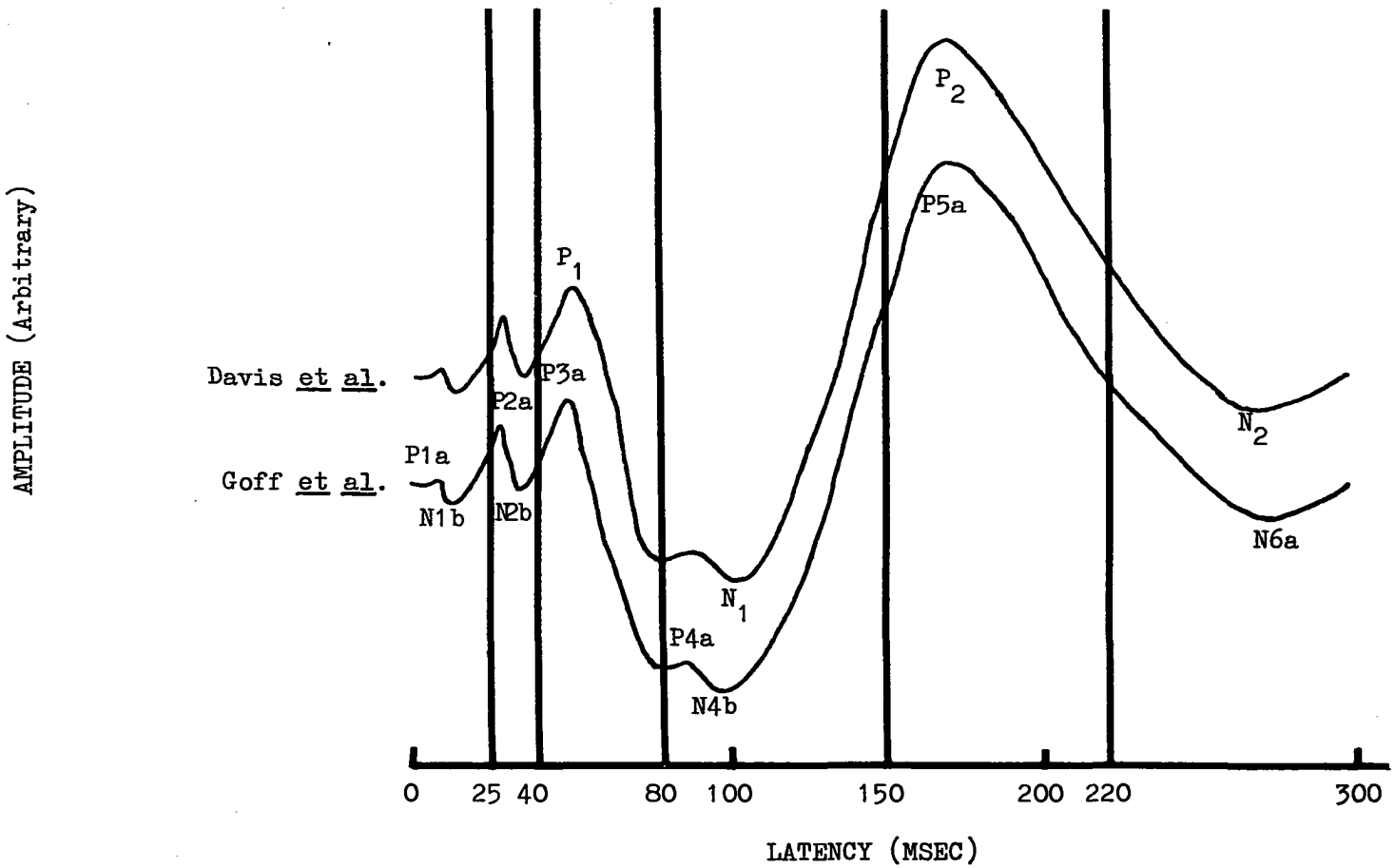


Figure 5.--Representation of latency breakdown for component classification in the present study contrasting the traditional system and the constant latency system.

Statistical Analysis of Data

The overall analysis of variance (115) employed to analyze the data which resulted from this investigation is a four treatment, four period change-over design applied within a two-by-two factorial experiment with repeated measures on each factor.

When the actual analysis of the data was undertaken it became readily apparent that not all subjects exhibited data points for all components. This problem prevented the utilization of the entire group of eight subjects for analysis purposes without estimating an inordinate number of missing values. It was, therefore, decided to analyze the data by component, utilizing at a minimum four subjects for each component. Thus, estimation of missing data values was kept at a minimum. The missing data were estimated by means of the following formula: (97, p. 150)

$$X = \frac{r\,(R + C + T) - 2G}{(r-1)\,(r-2)}$$

where: r = number of subjects,

R, C, T = totals of the observed values for the row, column, and treatment containing the missing value, and

G = grand total of the observed values.

It was found that component 3 and component 9 did not occur with sufficient frequency to enable analysis, hence these two components were dropped from consideration leaving a total of seven components. The subjects utilized for statistical analysis for each of the seven different components are found in Table 2.

Since the N4a component was exhibited by all eight subjects

TABLE 2

SUBJECTS UTILIZED FOR INDIVIDUAL COMPONENT ANALYSIS

Component	Subjects	Total
1 (N3a)	1 4 6 7	4
2 (P3b)	1 3 4 6	4
4 (N4a)	1 through 8	8
5 (P4b)	1 3 4 6	4
6 (P5a)	1 2 4 7	4
7 (P5b)	1 2 3 8	4
8 (N6a)	1 2 4 7	4

all data were used in the analysis of this particular component thereby increasing the precision and power of the statistical analysis for that component.

These data are presented and discussed in the following chapter.

CHAPTER IV

RESULTS AND DISCUSSION

The purpose of the present investigation was to delineate certain aspects of the within-average variability of acoustically-evoked potentials obtained from the vertex of the intact human scalp. Potentials evoked by 0.1-msec acoustic clicks at 50-dB SL delivered alternately every four seconds to the left and right ears of eight subjects were recorded, separately stored on magnetic tape and later printed out on calibrated graph paper from which the data were manually measured. Each average consisted of one hundred summations of EEG activity for 500 msec following stimulus onset. Criteria for subject selection included measured normal auditory acuity, admitted history of negative ear pathology and central nervous system disorders, right handedness, and judged at a pilot session to produce "adequate" evoked responses.

The effects of psychophysiological state and left and right ear stimulation, as well as the effect of stimulus presentation on different days, was determined by obtaining responses under four conditions (sitting quietly, counting the number of stimuli, discriminating 10-dB intensity increments and reading) from both ears separately and then repeating the same procedure on the same subject on a different day.

Latencies of seven points on the evoked potential, as well as the amplitudes relative to the N4a component and relative to a zero

baseline, the associated variance, and a silent variance estimated from on-going EEG activity just prior to stimulus onset were measured and are reported.

— Since there were a considerable number of missing data points, i.e., not all subjects produced an observable or measurable response at each of the seven defined components, each component was analyzed separately utilizing the four most responsive subjects that would still maintain the treatment and run balance in the experimental design. All eight subjects were utilized in the analysis of the N4a component since there were no missing data points here. The subjects utilized in analysis of the remaining components are found in Table 2. This same division of subjects holds for the analysis of latencies and amplitudes for the individual components. Where applicable the data will be compared to that reported by other investigators.

Variance

Since each variance datum was based on 100 samples it was felt that the variance measurements should be approximately Gaussian. Frequency plots confirmed this and consequently Analyses of Variance were performed without transforming the variance data.

Ear Effects

The mean variance for individual components averaged over treatments and trials for each ear are recorded in Table 3. The overall AOV showed no significance between ears ($P > .05$) for components except for the P5b component (Tables 32 through 38, APPENDIX C). Since the finding for the P5b component did not follow the trend of results across compon-

TABLE 3

MEAN VARIANCE IN MICROVOLTS OF EACH COMPONENT FOR LEFT AND RIGHT EARS AVERAGED OVER ALL TREATMENTS AND BOTH TRIALS

Ear	N3a	P3b	N4a	P4b	P5a	P5b	N6a
Left	1.48	1.45	1.93	1.52	1.61	1.87	1.67
Right	1.50	1.57	1.89	1.61	1.66	1.74	1.75

ents, inspection of the error terms was indicated. This revealed that the error term for the P5b component was much smaller than the error terms for the rest of the components. Thus, the significance found may be the result of a poor estimate of error for that component.

The overall AOV showed no significant differences ($P > .05$) between the test and retest sessions for any of the seven components.

Treatment Effects

The overall analysis of variance showed that the treatments had an effect on the variance. For all components, except the P4b and P5a components, significance was obtained ($P < .05$). The mean variances for each component by condition is graphically depicted in Figures 6 and 7. A Duncan's Multiple Range Test (97) was employed within each component to determine where differences were located. For the N3a and P3b components, the quiet and counting conditions showed significantly greater variance than the reading condition. Analysis of the P4b component showed only that the quiet condition was significantly more variable than the reading condition. Inspection of Figures 6 and 7 reveal that the quiet condition shows the highest variance for all components except

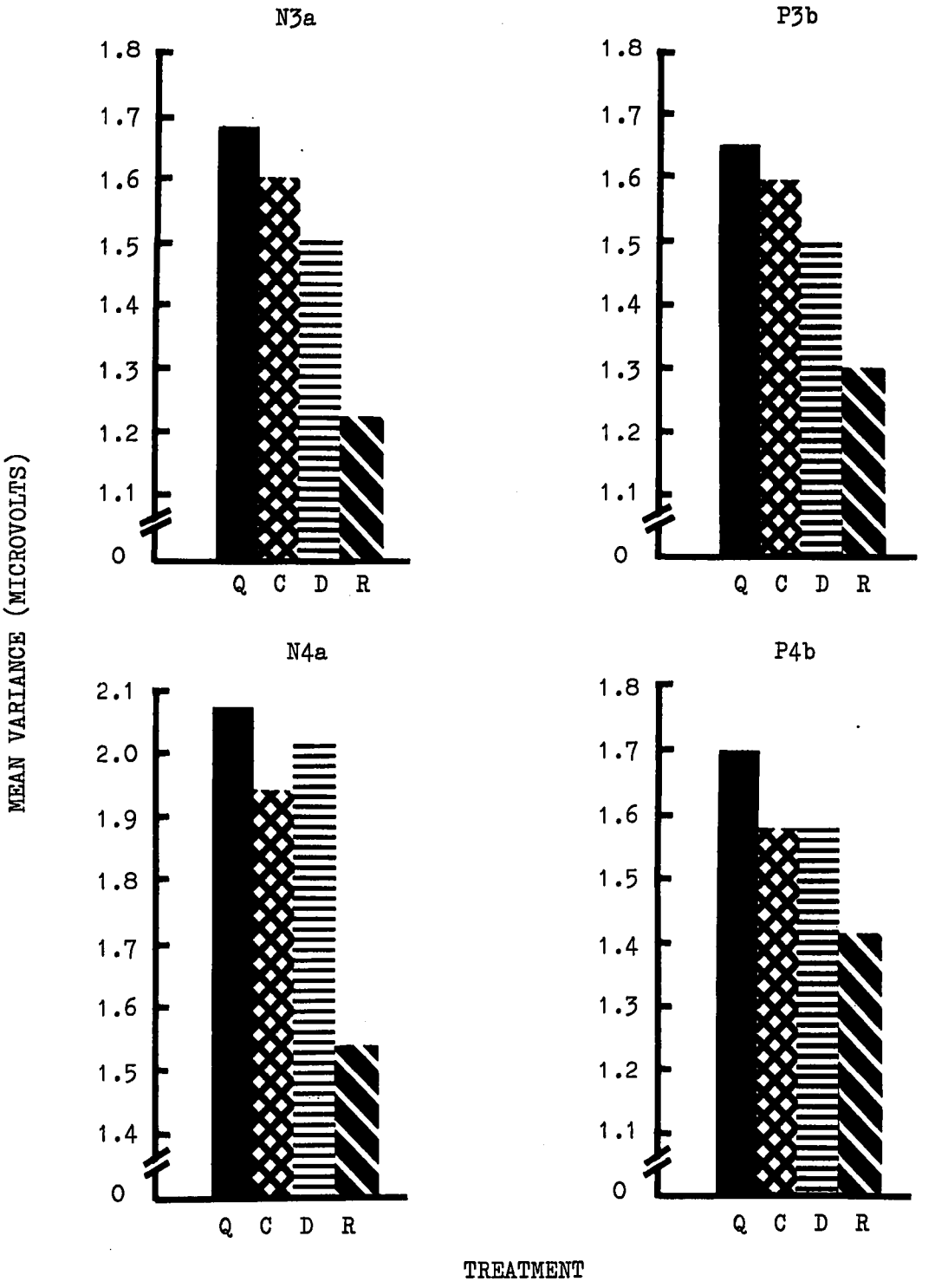


Figure 6.--Mean variances of the N3a, P3b, N4a, and P4b components in microvolts across treatments. (Note different ordinate values.) (Q: quiet, C: counting, D: discriminating, R: reading)

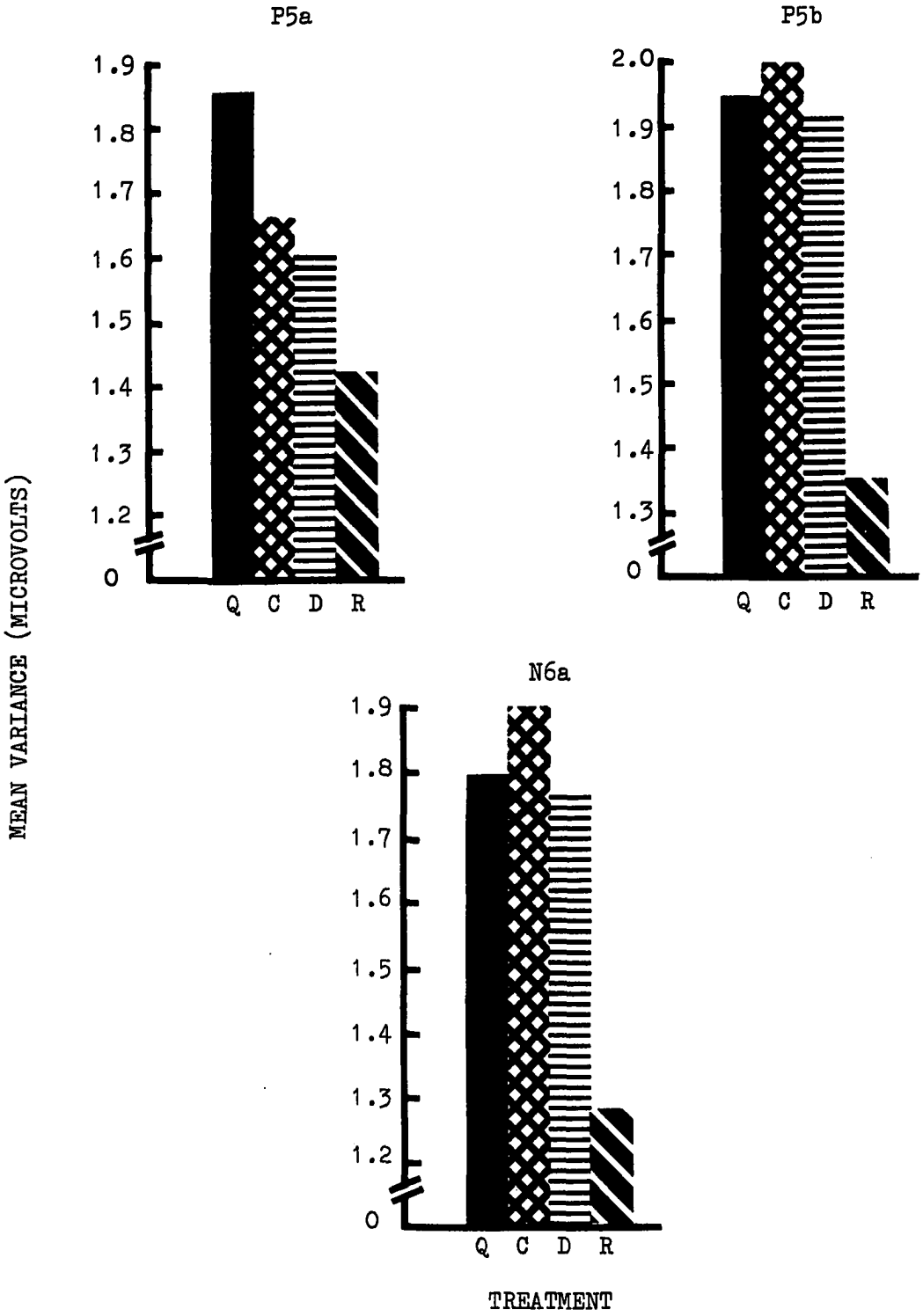


Figure 7.--Mean variances of the P5a, P5b, and N6a components in microvolts across treatments. (Note different ordinate values.) (Q: quiet, C: counting, D: discriminating, R: reading)

for the P5b and N6a components while the reading condition always produces the lowest mean variances. From these results it is obvious that the different tasks performed by the subjects during data accumulation affected the variability across components with the reading condition seemingly resulting in a more stabile neurological environment.

Run Effects

Figures 8 and 9 depict the run-by-component variance. Run 1 is composed of the set of data resulting from the treatments across subjects which occurred first in the experimental session regardless of the treatment. Run 2 is the set of data resulting from the treatments presented second, Run 3 the third set and Run 4 the fourth set. The overall AOV showed significance on runs for the N3a, P3b, and P4b components. The Duncan's Multiple Range Test revealed that Run 1 showed significantly less variability ($P < .05$) than Run 4 for the N3a, P3b, N4a, and P4b components. In addition, Run 1 showed significantly less variability than Run 2 for the P3b and N4a components and significantly less variability than Run 3 for the N4a and P4b components. Run 2 showed significantly less variability than Runs 3 and 4 for the P4b component. Visual inspection of Figures 8 and 9 shows that Run 1 tends to result in the least variance while there is a tendency for greater variance in Run 4.

Apparently, regardless of the treatment, the variability of the electrical activity of the brain, at least until the occurrence of the N6a component, increased from the time of the initiation of an experimental session until its termination. This result is not unexpected since it seems reasonable to assume that after a subject has

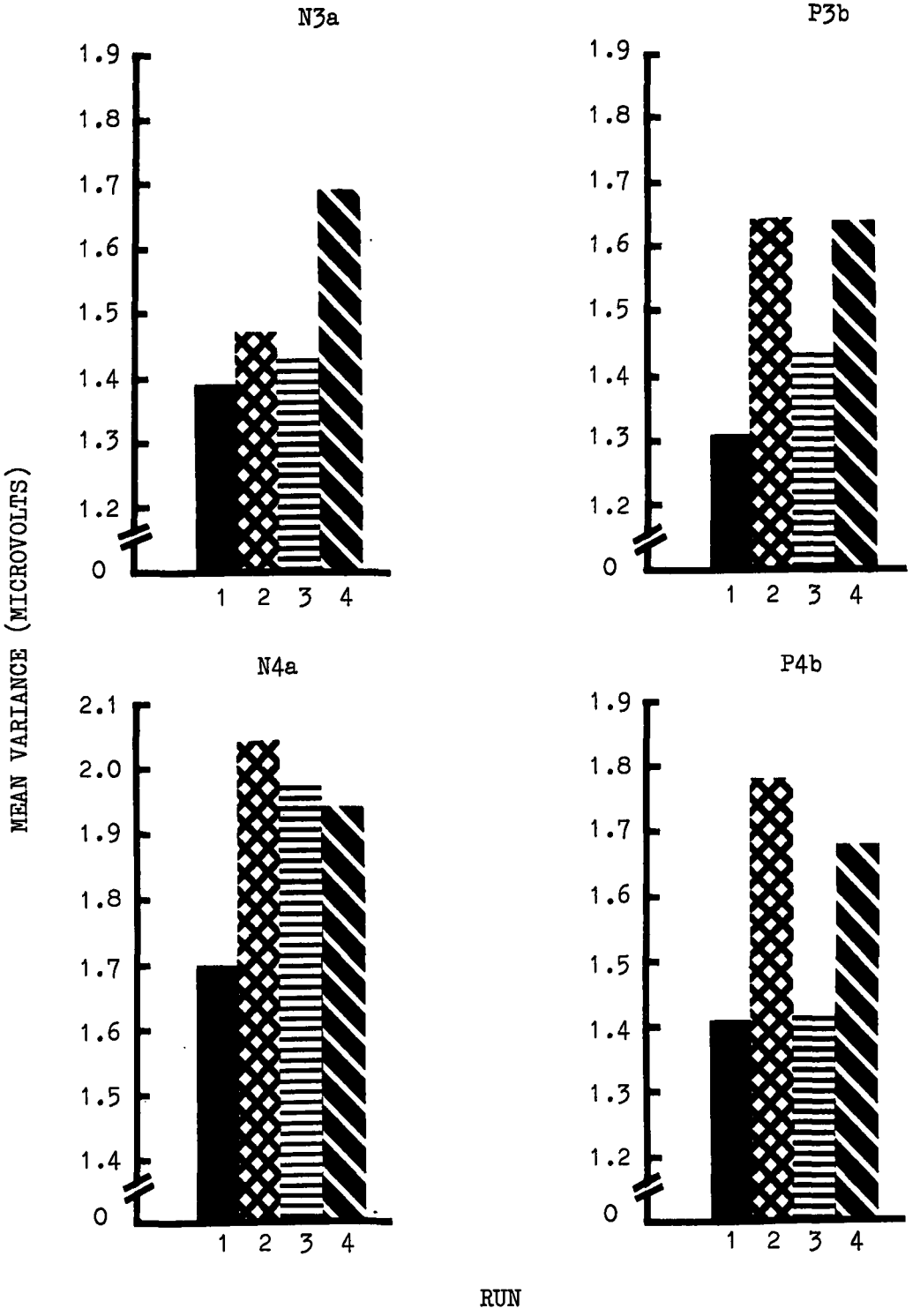


Figure 8.--Mean variances of the N3a, P3b, N4a, and P4b components in microvolts across runs. (Note different ordinate values.)

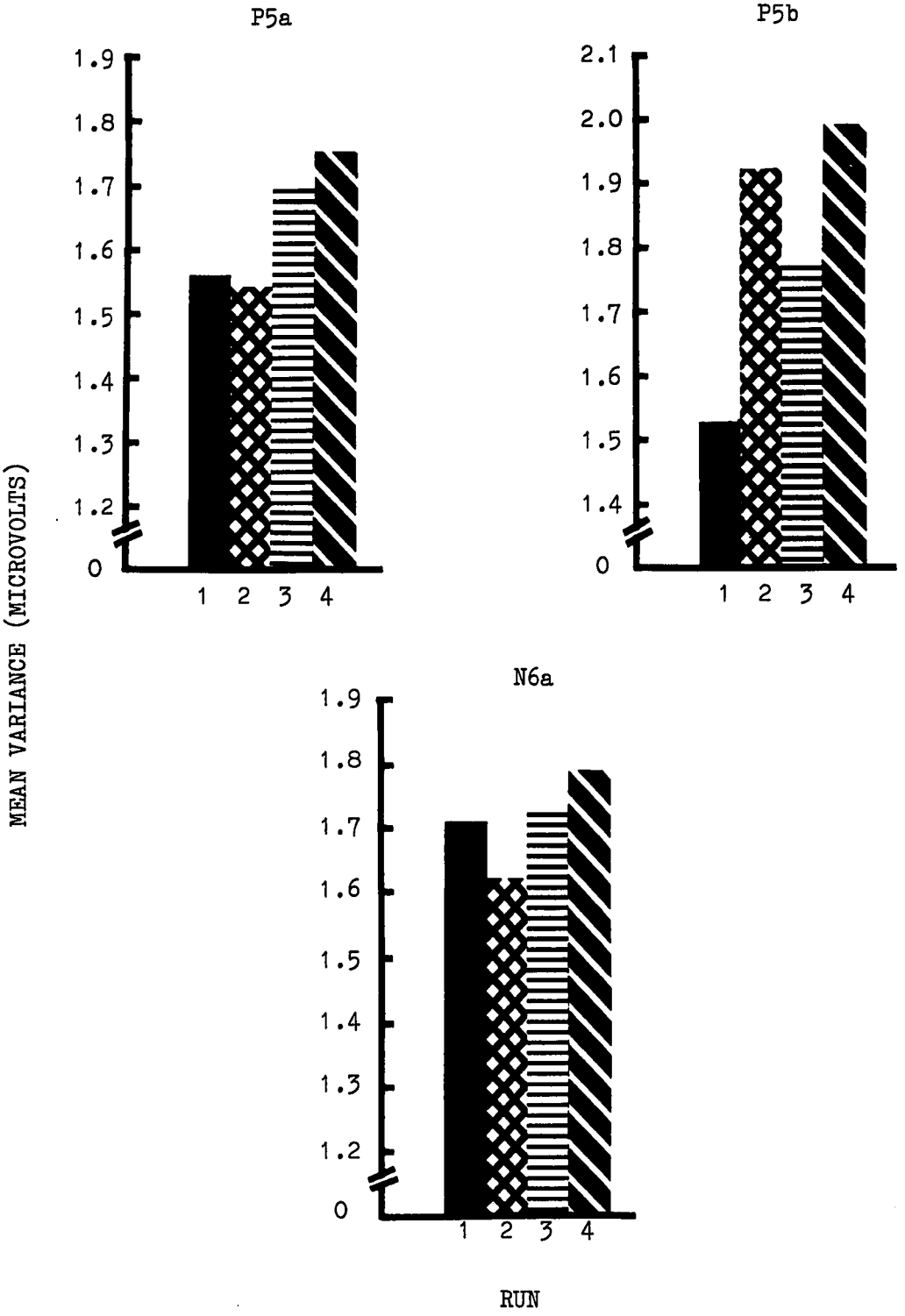


Figure 9.--Mean variances of the P5a, P5b, and N6a components in microvolts across runs. (Note different ordinate values.)

been in a restricted environment for a time he may tire of the situation and thus become restless. This observed increase in variability may be the result of fatigue which could conceivably manifest itself in increased brain or myogenic activity, thus causing the variance measures to be higher for Run 4.

Treatment Effects Across Components

In order to observe the variance across components it was necessary to re-arrange the data so that the same subjects' data would be represented at each component. In other words, the data in the previous analyses has been looked at by treatment effects within a given component. The present analysis looks across components within a given treatment. It is, therefore, imperative that if valid comparisons are to be made across the components the data must be derived from identical subjects. Due to the arrangement of the missing data the same subjects' data could not be utilized across all seven components. Hence, the N3a and P3b components were compared within a particular set of subjects and the N4a, P4b, P5a, P5b, and N6a components were compared within another set of subjects. Subjects 1, 4, 6, and 7 were utilized for the N3a and P3b components while subjects 1, 2, 3, and 4 were utilized for the N4a, P4b, P5a, P5b, and N6a components. Ordering of runs and counterbalancing of treatments were thus maintained. (See Table 2.) The silent variance was estimated separately for the two groups of components from the silent variance of those subjects making up the groups and was treated as a component in the analysis. The silent variance was estimated by deriving the mean of the highest and lowest points of the

variance for the silent period.

The analysis of variance was equivalent to randomized complete block design (with subjects as blocks) for each treatment and for each group (97) and showed no significant difference ($P > .05$) for any treatment across all components. As a matter of course, Dunnett's procedure was employed utilizing the silent variance as the control. Out of the 28 comparisons the only difference found was under the counting condition where the variance of the N6a component was significantly greater ($P < .05$) than the silent variance. This significance is surprising and is unexplained. It is in opposition to the rest of the results without apparent reason and may simply represent a random deviation.

Figures 10 through 15 graphically depict the data upon which the above-discussed analysis was performed. Inspection of these figures reveals several interesting aspects. Figure 10 shows that the silent variance exceeds the variance for the N3a and P3b components for all treatments with the exception of the N3a component under the counting condition. In Figures 11 and 12, the same type of comparison is made for the N4a, P4b, P5a, P5b and N6a components. Inspection of these figures reveals that at times the silent variances are smaller than component variances, while at other times the converse is true. Further inspection does not reveal any pattern for this phenomenon.

The variance which is seen for the components is a combination of the variance due to the response plus the variance of the on-going activity. The variance of the silent period sampled just prior to stimulus on-set is assumedly representative of just the variance of the on-going activity. The original idea of arriving at the variance of the

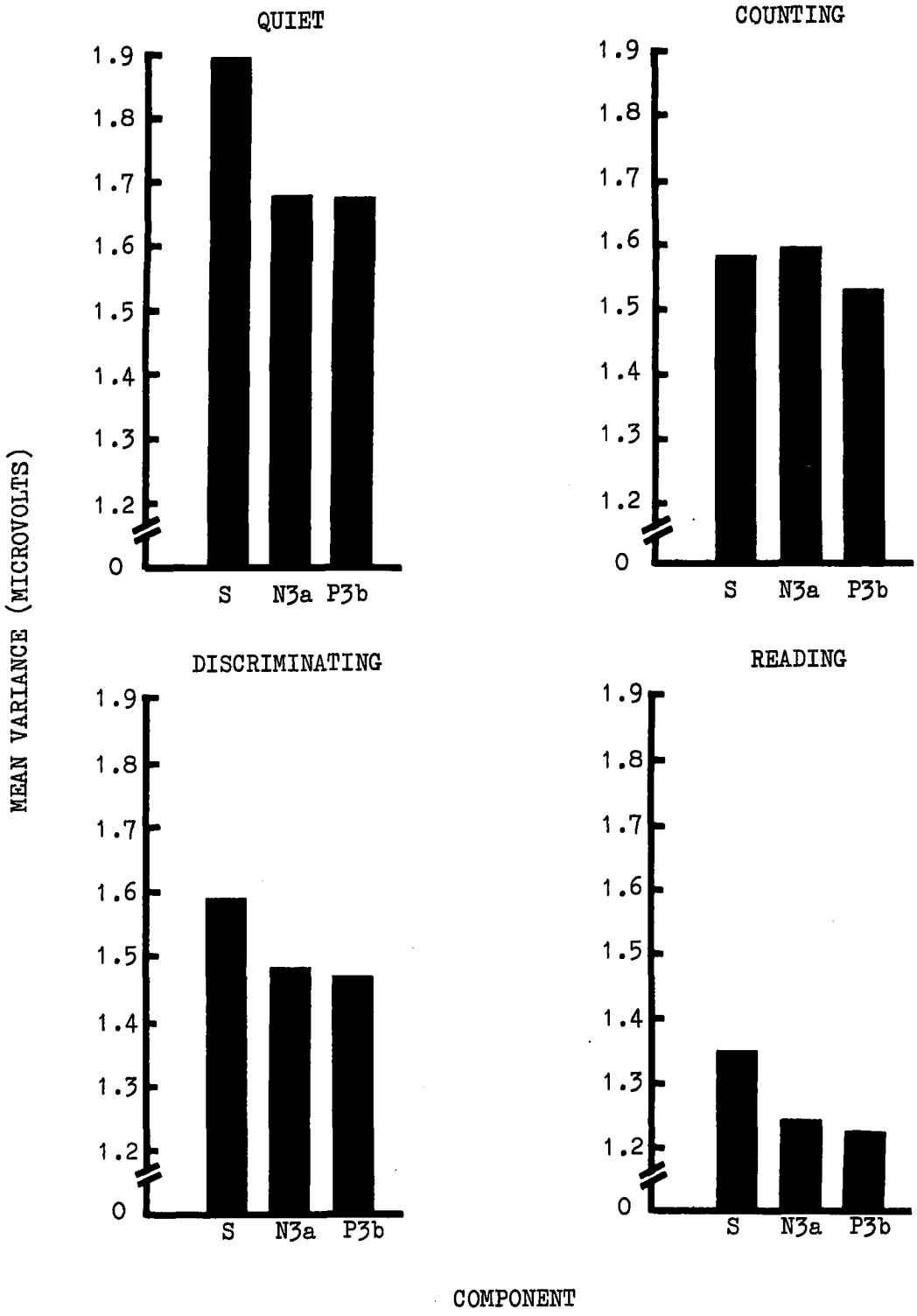


Figure 10.--Mean variances of the N3a and P3b components and their associated silent variance estimates for the quiet, counting, discriminating, and reading conditions. (S: silent)

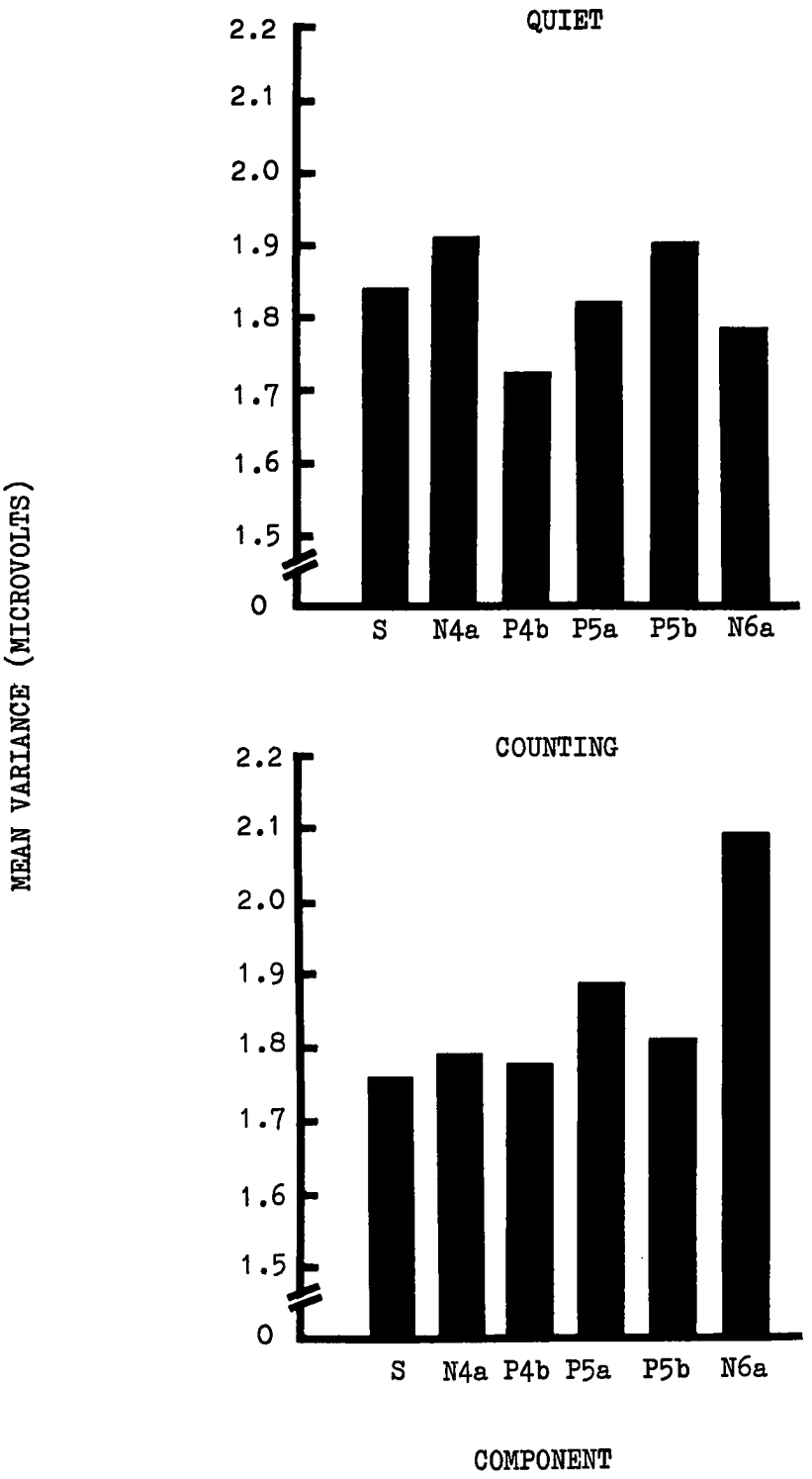


Figure 11.--Mean variances of the N4a, P4b, P5a, P5b, and N6a components and their associated silent variance estimates for the quiet and counting conditions. (S: silent)

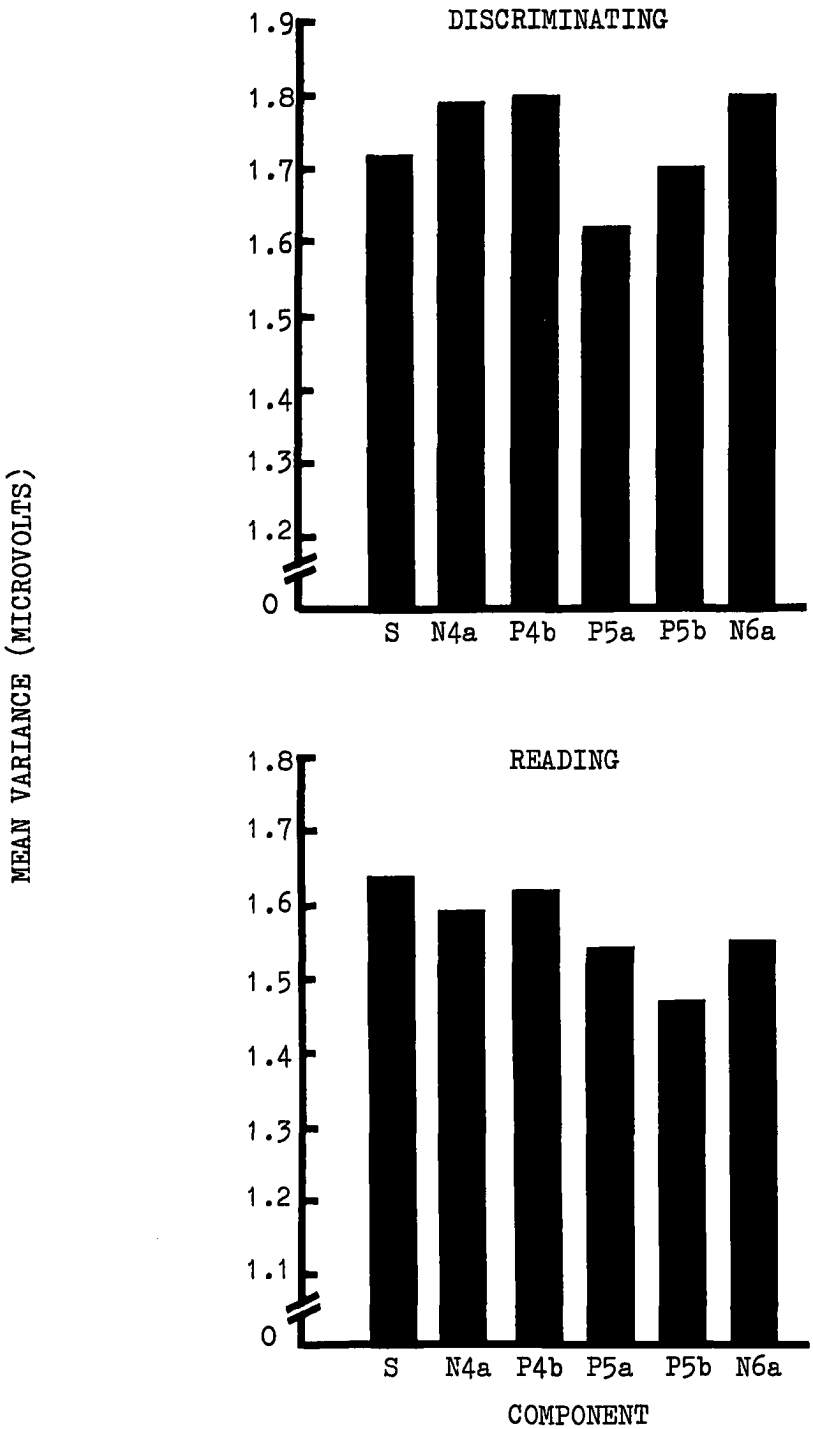


Figure 12.--Mean variances of the N4a, P4b, P5a, P5b, and N6a components and their associated silent variance estimates for the discriminating and reading conditions. (Note different ordinate values.) (S: silent)

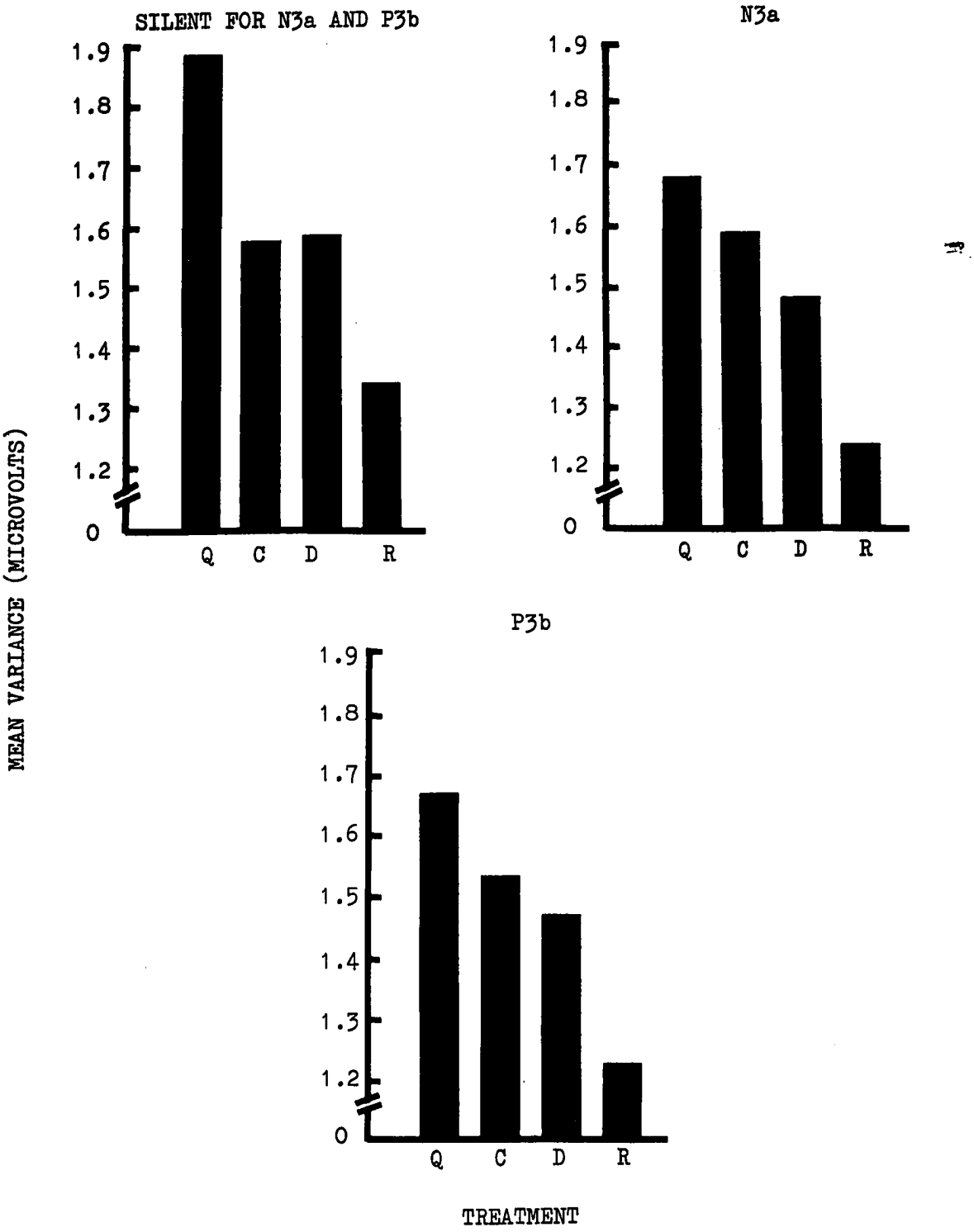


Figure 13.--Mean variances of the four treatments for the N3a and P3b components and their associated silent variance estimates. (Q: quiet, C: counting, D: discriminating, R: reading)

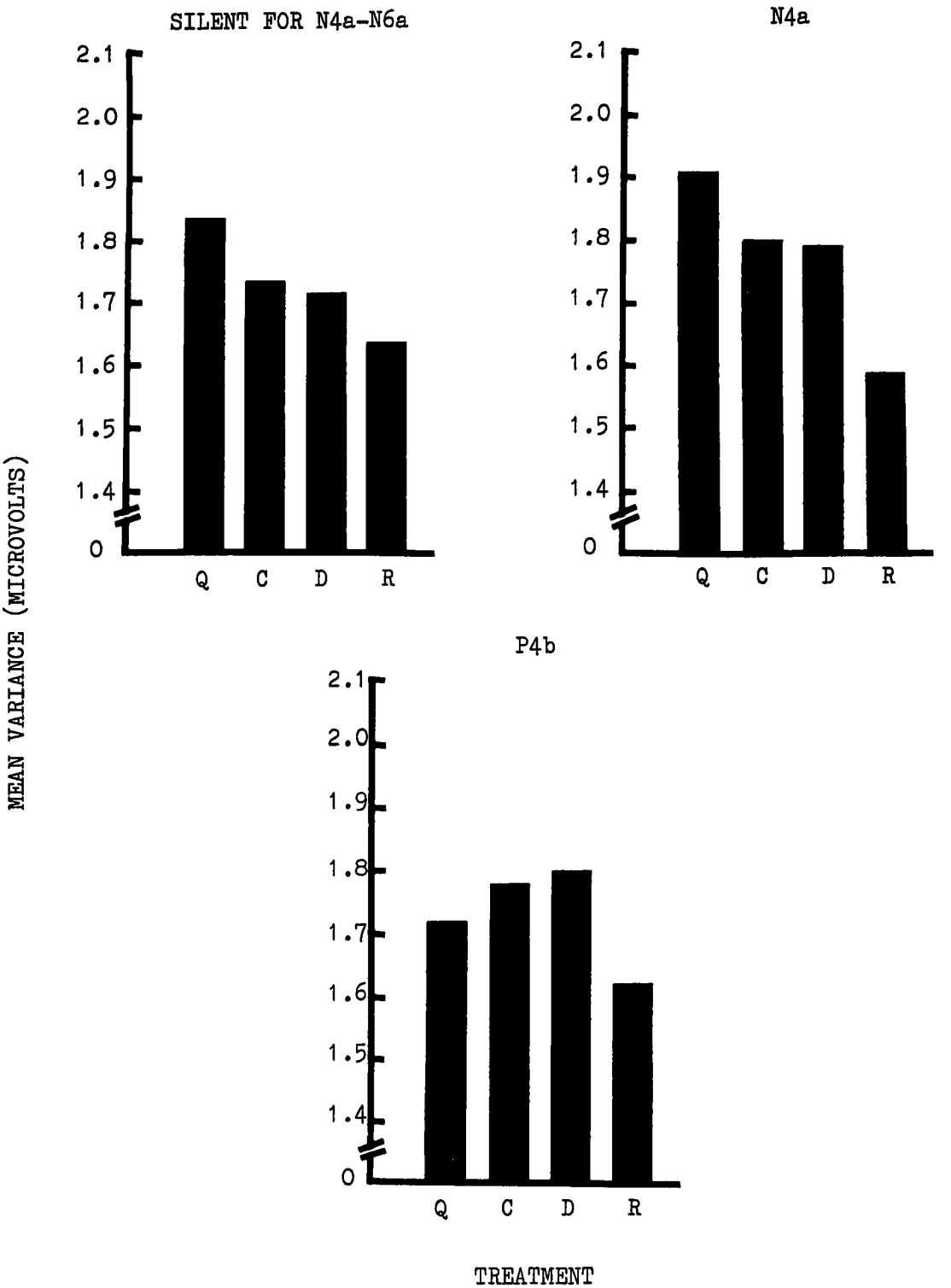


Figure 14.--Mean variances of the four treatments for the N4a and P4b components and the associated silent variance estimates for the N4a-N6a components (Q: quiet, C: counting, D: discriminating, R: reading).

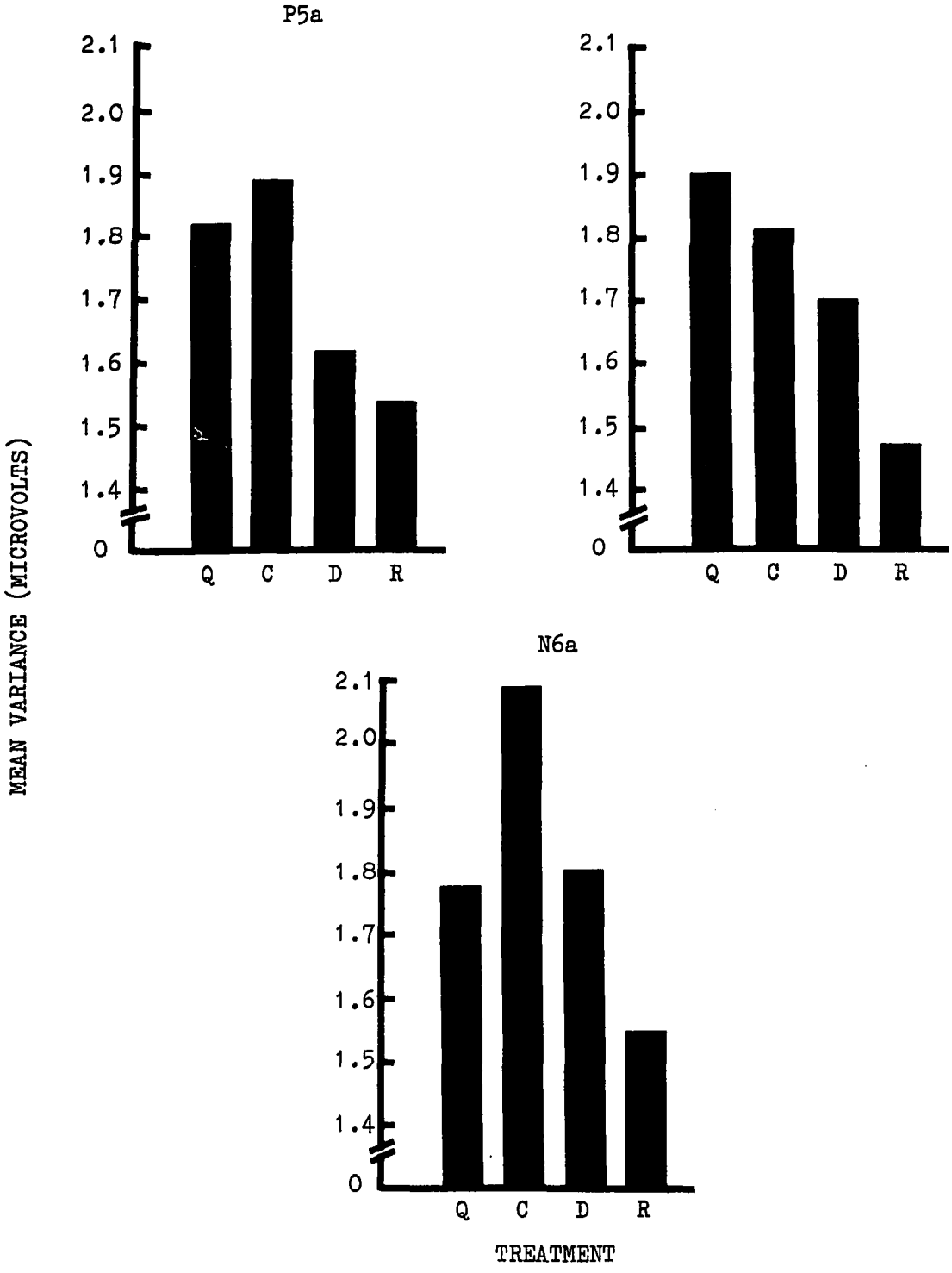


Figure 15.--Mean variances of the four treatments for the P5a, P5b, and N6a components (Q: quiet, C: counting, D: discriminating, R: reading).

response by subtracting the silent variance from the response variance and having left only the response variance cannot be done with this data because some of the component variances are smaller than the silent variances. This observation leads to the conclusion that either the estimate of the silent variance, that of the component variance, or both are poor; or, that the presence of the signal causes variance to be reduced. Looking at all components, the first explanation seems to be more tenable. However, in looking at the N3a and P3b components alone the second explanation seems worthy of consideration, particularly for relatively early componentry (Figure 10).

Figures 13, 14, and 15 reveal that there is a tendency for the quiet condition to generally possess the highest variance across components and silence, while the reading condition consistently shows the lowest variance across components and silence. This finding seems to support the hypothesis that restlessness on the part of the subject may be a contaminating factor for the variances as it seemed to be when the data was analyzed by runs wherein the fourth run showed more variance than the first.

Visual inspection of the data did not reveal a consistent place for points of highest variance nor for points of lowest variance along the 500-msec write-out. Furthermore, since there were no differences found between the level of the variance between components and across treatments, there does not appear to be any component that is either more or less variable than the others. There is, however, a tendency for early componentry (before 85 msec) to show less variability than those components which occur later.

Latency

The mean latencies for each of the seven components for left and right ear stimulation are shown in Table 4. As can be readily

TABLE 4

MEAN LATENCIES IN MILLISECONDS OF THE SEVEN COMPONENTS FOR LEFT AND RIGHT EARS AVERAGED OVER ALL TREATMENTS AND BOTH TRIALS

Ear	N3a	P3b	N4a	P4b	P5a	P5b	N6a
Left	42.13	55.53	98.95	129.31	160.63	191.91	245.53
Right	42.62	53.44	99.17	128.41	158.16	186.22	235.63

observed there is a tendency for left ear responses to occur later than right ear responses. The N3a and N4a components are the only two which show longer latencies as a result of right ear stimulation. The overall analysis of variance (Tables 39 through 45, APPENDIX C) shows that the only significant difference between left and right ear stimulation is for the P3b component in which responses to left ear stimulation are significantly later ($P < .05$) than responses to right ear stimulation. When the raw means are displayed pictorially, as in Figure 16, it appears that large differences exist between left and right ear stimulation, particularly for later components. However, the impression of the differences is brought into a more accurate perspective (Figure 17) by taking into account the standard error of the left-right difference, utilizing the error mean square for each component, and standardizing these differences. Even though the P3b component is the only one which shows a significant difference between ears the other components (except N3a

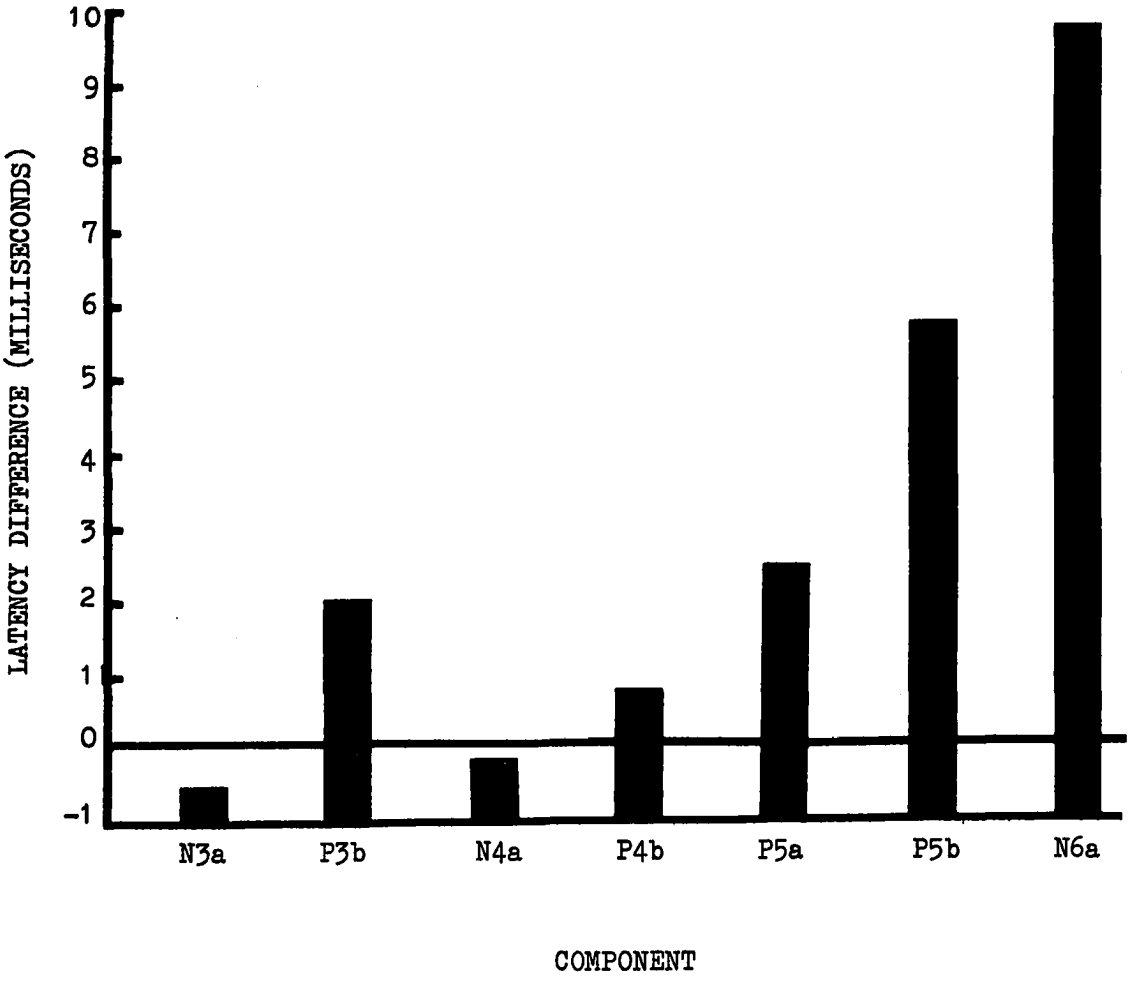


Figure 16.--Mean latency differences between left and right ear stimulation across the seven components plotted relative to right ear stimulation.

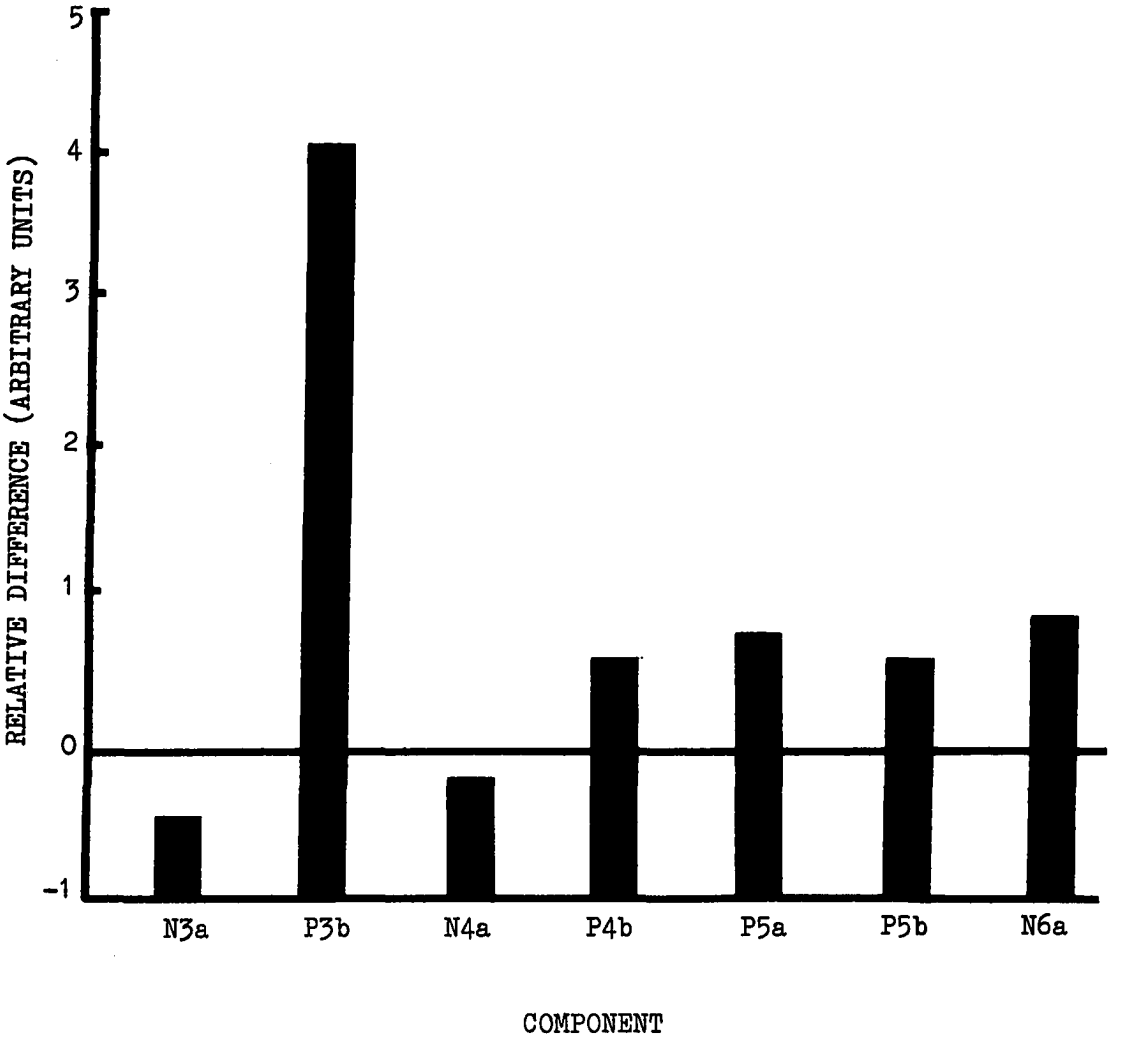


Figure 17.--Standardized differences between left and right ear stimulation across the seven components plotted relative to right ear stimulation.

and N4a) do show responses to left ear stimulation to be longer. If there were no differences between responses from left and right ear stimulation, the probability of observing responses to left ear stimulation longer than responses to right ear stimulation five times out of seven is 0.0625. There seems to be sufficient evidence for the question to be raised regarding a left-right dichotomy and to provoke further investigation about "earedness" being reflected in the latency of slow components.

Treatment Effects

Figures 18 and 19 show latencies for the seven components by treatment. The overall analysis of variance shows that treatment effects were significant ($P < .05$) for the P3b, N4a, and P4b components. All other components were not significant ($P > .05$). The Duncan's Multiple Range Test applied within components revealed the following:

1) For the N3a component, latencies under the reading condition were significantly longer than those under the discriminating condition.

2) For the P3b component, latencies under the reading condition were significantly longer than those under the discriminating, counting, and quiet conditions.

3) For the N4a component, latencies under the discriminating condition were significantly longer than for the counting, quiet, and reading conditions.

4) For the P4b component, latencies under the discriminating and reading conditions were significantly longer than latencies under the counting and quiet conditions.

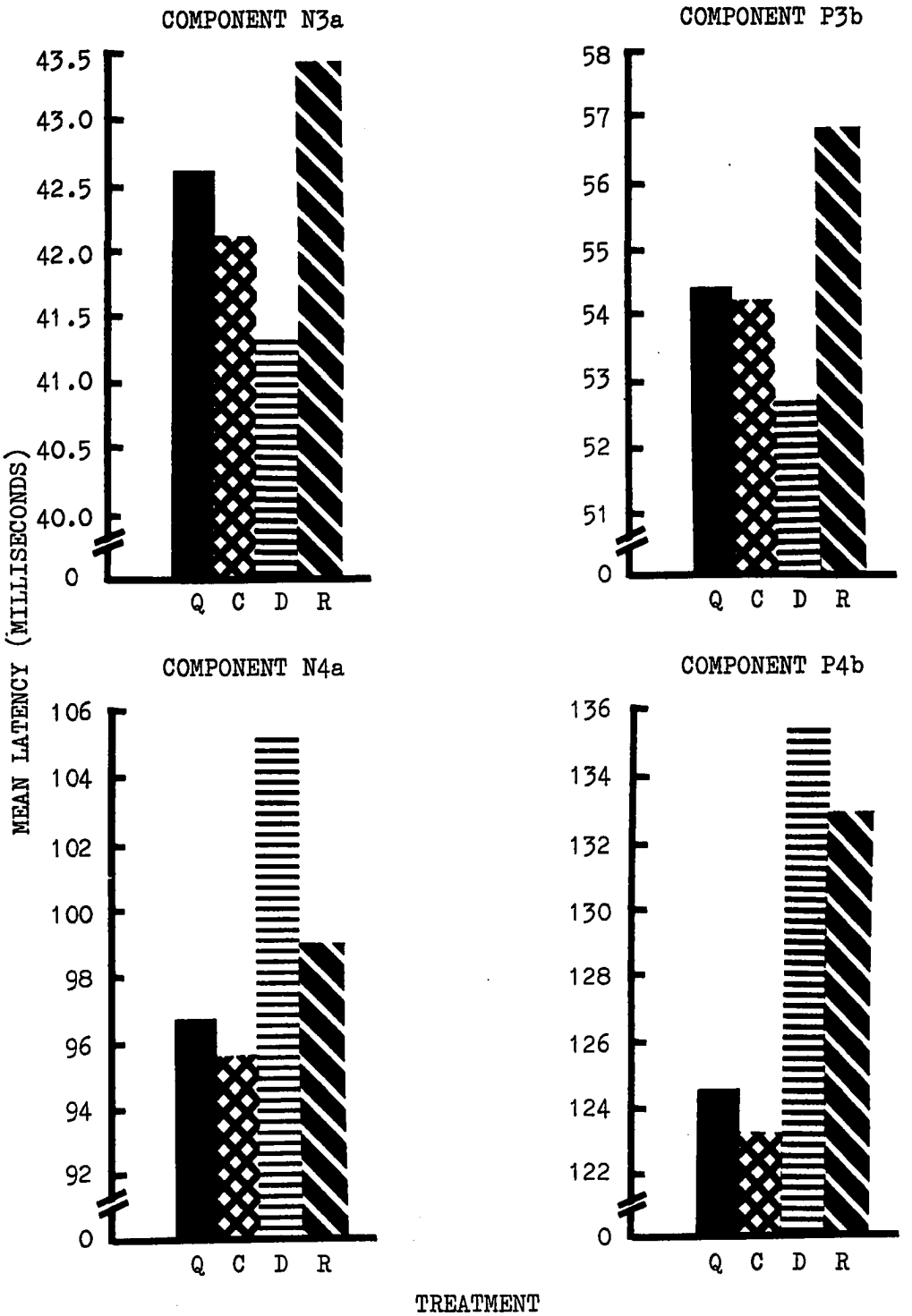


Figure 18.--Mean latencies of the N3a, P3b, N4a, and P4b components for the four treatments. (Note different ordinate values.) (Q: quiet, C: counting, D: discriminating, R: reading)

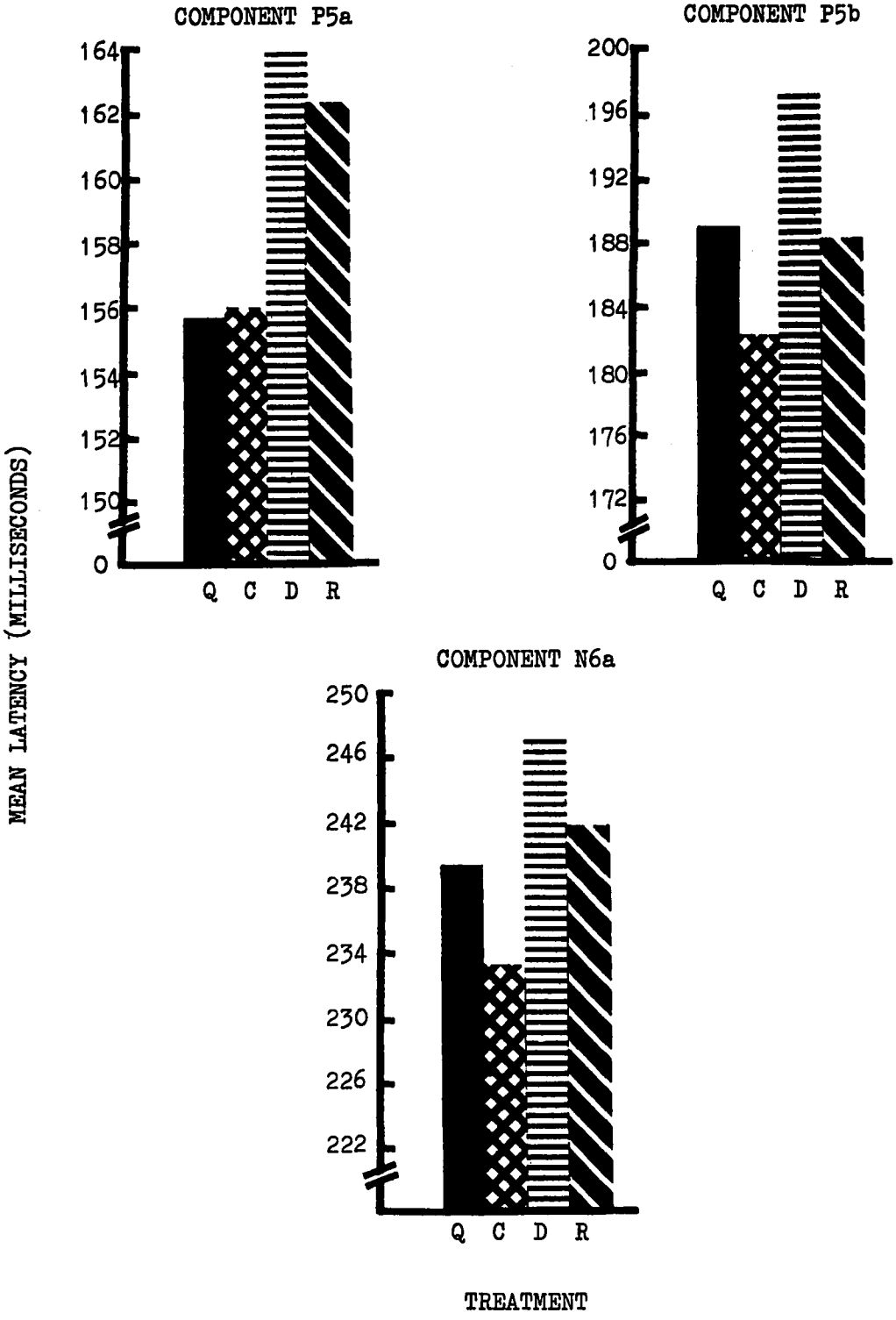


Figure 19.--Mean latencies of the P5a, P5b, and N6a components for the four treatments. (Note different ordinate values.) (Q: quiet, C: counting, D: discriminating, R: reading)

5) For the P5b component, latencies under the discriminating condition were significantly longer than latencies under the counting condition.

It is interesting to note that the latencies for the N3a and P3b components as revealed by Figures 18 and 19 do not show a great deal of dispersion across treatments. The N4a through the N6a components, however, show considerably more dispersion. One possible explanation for these findings is that the earlier components in the evoked potential may arise from neurological substrates whose primary function is the transmission of neural impulses to the primary auditory reception areas of the cortex; hence, small differences are seen among latencies for the various treatments because speed of neural transmission might be little affected as a result of the influence of structures such as the reticular activating system. The discriminating condition, in which it is assumed that the subject's attention was most highly focused on the stimuli, shows the fastest latencies, followed by the counting condition (the next most salient attraction to stimuli), the quiet, and finally the reading condition in which the subject's attention was presumably most highly diverted from the stimuli. Continuing this explanation, the later components (those following P3b) may arise from neurological substrates whose primary function is the processing and elaboration of information contained in the stimulus. The dispersion between treatment means of these components might be expected if such were the case since subjects were required to perform different mental tasks during stimulus presentation periods, ostensibly activating different combinations of neurological circuits across treatments.

Run Effects

The overall analysis of variance showed no significant differences ($P > .05$) across runs. Thus, latency does not change significantly from the first to the later part of an experimental session as the subject assumedly becomes more fatigued and less alert.

Interactions

The only significant interaction (overall analysis of variance, $P < .05$) for latency is ear-by-trial for the P3b component. An analysis of variance for simple effect interactions (randomized complete block design) was performed on all components to determine ear differences within treatments with the following results:

1) The N3a component under the quiet condition possessed latencies for the left ear stimulation that were significantly shorter ($P < .05$) than those for right ear stimulation.

2) The P3b component under the quiet condition showed that latencies for left ear stimulation were significantly longer ($P < .05$) than those for right ear stimulation; under the counting condition, latencies for left ear stimulation were significantly longer ($P < .05$) than those for right ear stimulation.

3) The N4a component under the discriminating condition showed latencies for left ear stimulation that were significantly shorter ($P < .05$) than those for right ear stimulation; under the reading condition, latencies for left ear stimulation were significantly longer ($P < .05$) than those for right ear stimulation. Significance was not observed for any of the remaining components.

If these observed differences are real, the results do not

appear to emerge into any readily observable tendency. It may be that errors in the identification of peak latencies are largely responsible for a relatively high degree of variability and the resultant lack of a clear trend.

Amplitude

Amplitude measurements were originally made relative to the zero voltage baseline displayed on the write-out through a calibrated circuit within the 204 Variance Computer. Visual inspection of the tracings of the averages revealed that the averages shifted around this reference, i.e., at times the trace was relatively negative with respect to the reference while at other times it was relatively positive for any given average. Such a phenomenon might confound the interpretation of the results; for example, if the average were shifted extensively in a positive direction, the measured amplitude of the N4a component would appear to be quite small relative to the zero voltage baseline when in fact it might be quite large with respect to adjacent components. In addition, positive components might appear to be much larger than they really are. It was, therefore, decided to choose the N4a component as the reference for comparison of componentry amplitude. The IBM 1800 computer was used to perform the mathematical manipulations required to obtain these data.

The mean amplitude relative to the N4a component averaged across treatments and trials is recorded in Table 5. The overall AOV (Tables 46 through 51, APPENDIX C) showed no significant difference ($P > .05$) between left and right ear stimulation for any of the seven components.

TABLE 5

MEAN AMPLITUDE IN MICROVOLTS RELATIVE TO THE N4a COMPONENT FOR LEFT AND RIGHT EARS AVERAGED OVER ALL TREATMENTS AND BOTH TRIALS

Ear	N3a	P3b	N4a	P4b	P5a	P5b	N6a
Left	1.31	2.51	0.00	3.45	4.79	5.94	1.56
Right	0.90	2.34	0.00	3.34	5.01	5.74	1.73

Treatment Effects

The overall analysis of variance was performed on the data as measured from the baseline (Tables 51 through 58, APPENDIX C) as well as the data as measured from the N4a component. These data are recorded in Tables 6 and 7 and are graphically presented in Figures 20 through 23. For the data measured relative to the N4a component the overall analysis of variance shows that the only significant difference ($P<.05$) in amplitude is for the P5b component. A Duncan's Multiple Range Test shows that this difference results from the fact that the amplitude under the discriminating condition is greater in magnitude than the amplitudes under the other three treatments.

For the same data measured relative to the zero baseline the overall analysis of variance shows that significant differences ($P<.05$) exist for the P5a and P5b components. The Duncan's Multiple Range Test shows that the amplitudes within the P5b component for the discriminating and counting conditions are significantly larger ($P<.05$) than the amplitudes for the quiet and reading conditions. The Duncan's Test did not show any significant differences ($P>.05$) for treatments within the P5a component.

TABLE 6

MEAN AMPLITUDE IN MICROVOLTS RELATIVE TO THE N4a COMPONENT ACROSS TREATMENTS

Treatment	Component					
	N3a	P3b	P4b	P5a	P5b	N6a
Quiet	0.97	2.27	2.90	5.01	4.51	1.60
Counting	0.93	2.71	3.65	5.27	6.03	1.89
Discrim.	1.41	2.60	3.76	5.10	7.79	2.29
Reading	1.13	2.14	3.27	4.23	5.03	0.79

TABLE 7

MEAN AMPLITUDE IN MICROVOLTS RELATIVE TO THE ZERO VOLTAGE BASELINE ACROSS TREATMENTS

Treatment	Component						
	N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	-1.50	0.26	-2.61	0.88	2.93	2.70	-0.61
Counting	-1.71	-0.05	-2.65	0.89	3.29	4.05	-0.22
Discrim.	-1.33	-0.18	-4.26	1.05	2.57	4.26	-0.44
Reading	-1.01	0.21	-2.78	0.84	2.28	2.58	-1.44

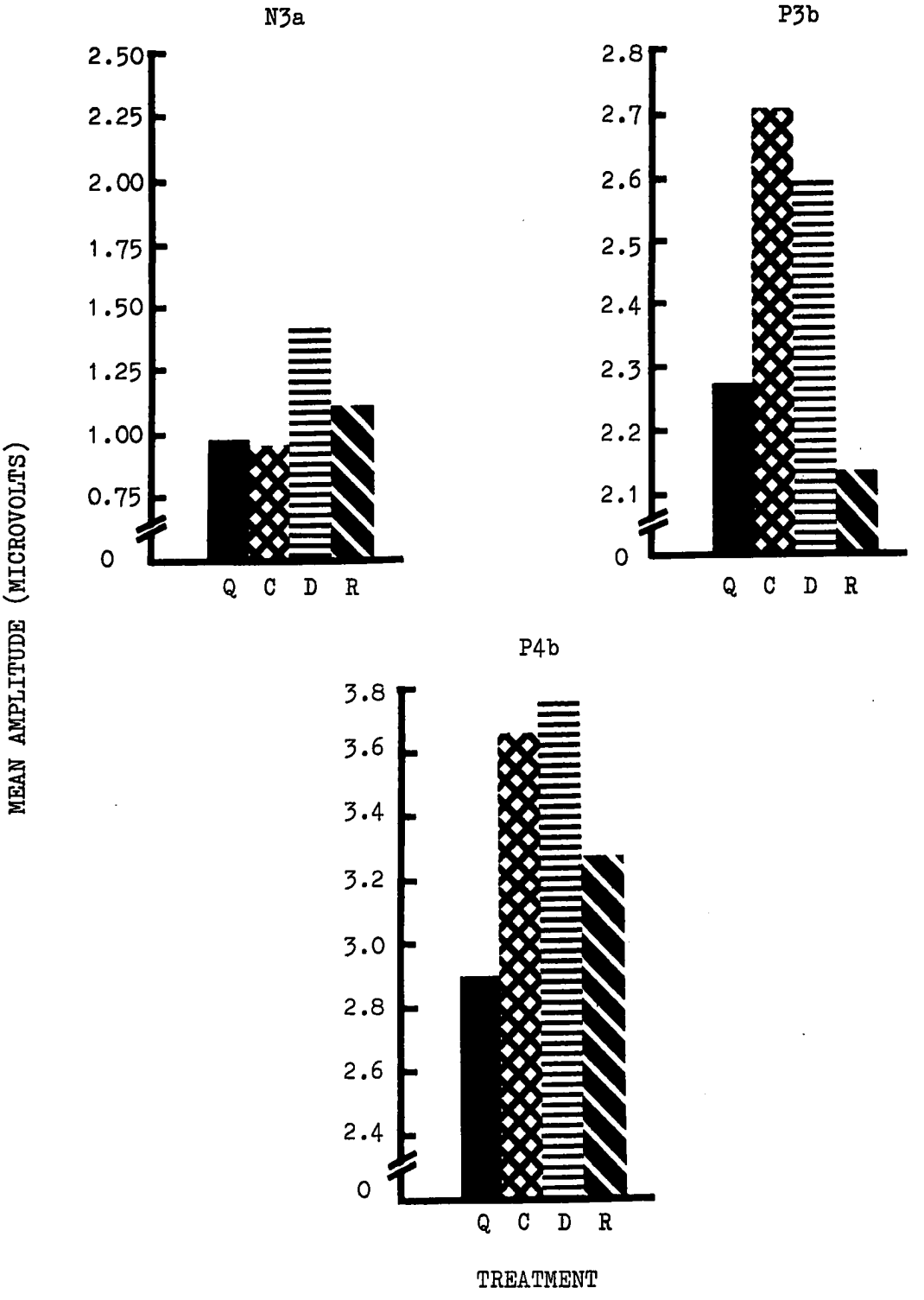


Figure 20.--Mean amplitudes of the N3a, P3b, and P4b components across treatments relative to the N4a component. (Note different ordinate values.) (Q: quiet, C: counting, D: discriminating, R: reading)

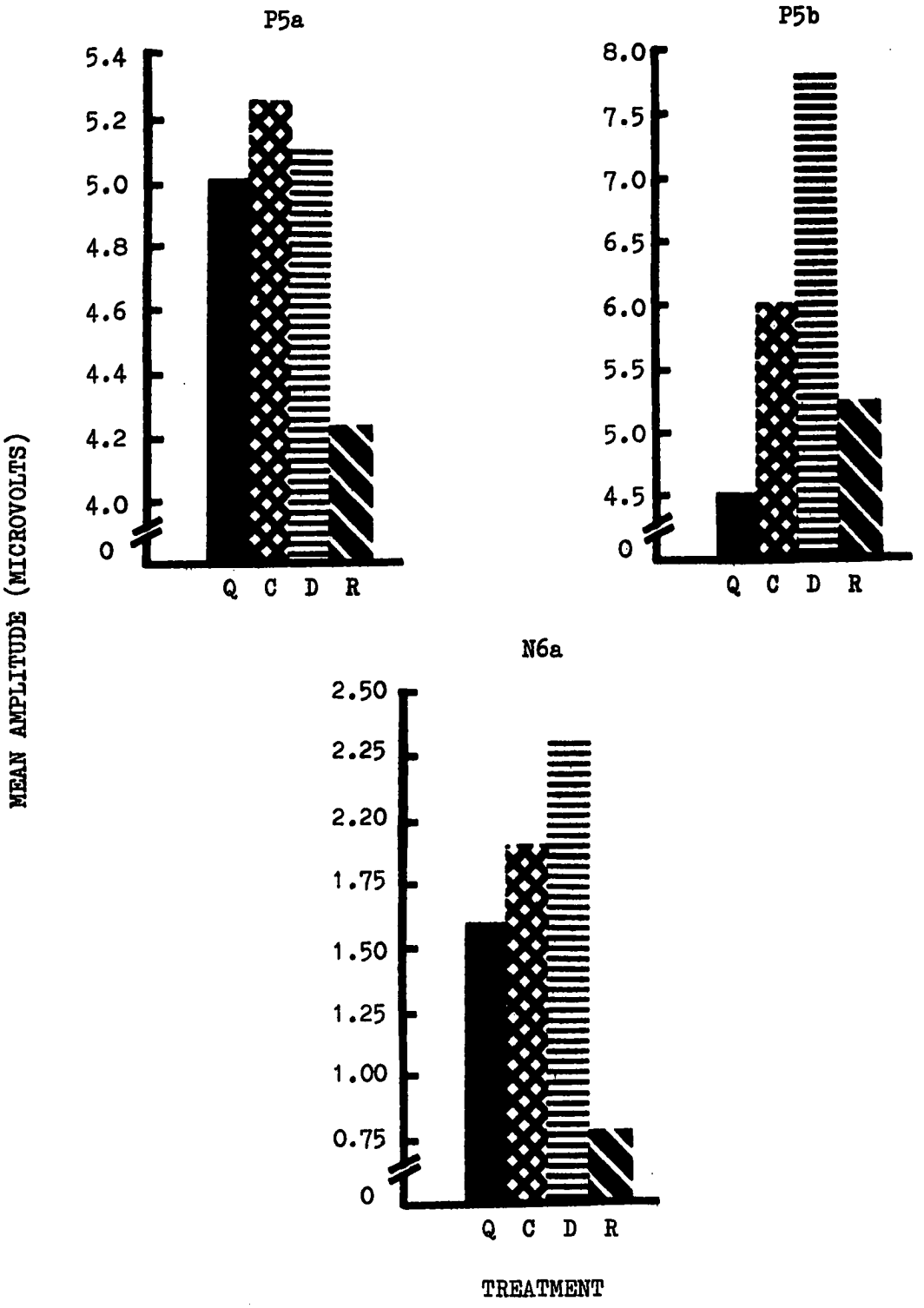


Figure 21.--Mean amplitudes of the P5a, P5b, and N6a components across treatments relative to the N4a component. (Note different ordinate values.) (Q: quiet, C: counting, D: discriminating, R: reading)

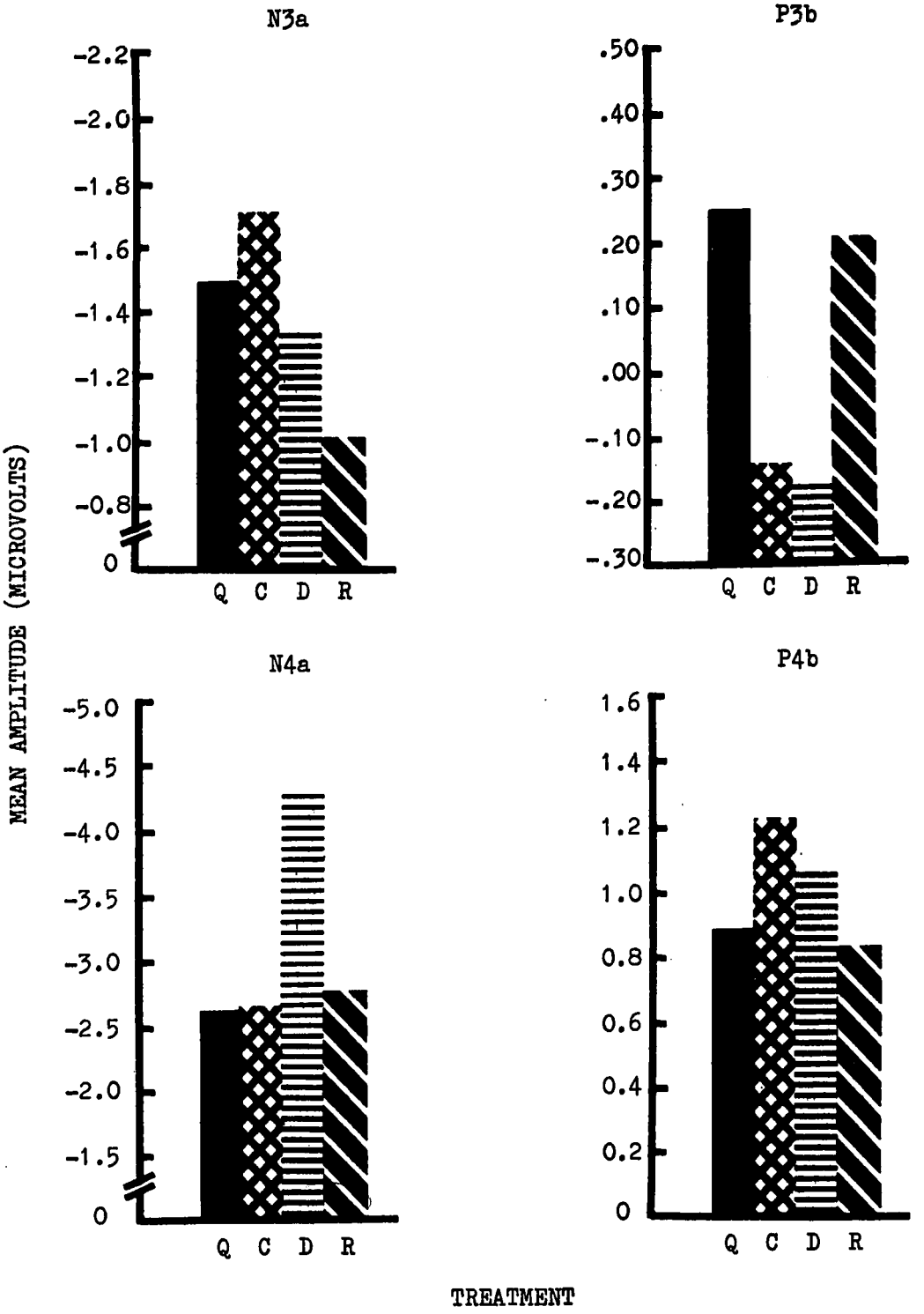


Figure 22.--Mean amplitudes of the N3a, P3b, N4a, and P4b components across treatments relative to the zero baseline. (Note different ordinate values.) (Q: quiet, C: counting, D: discriminating, R: reading)

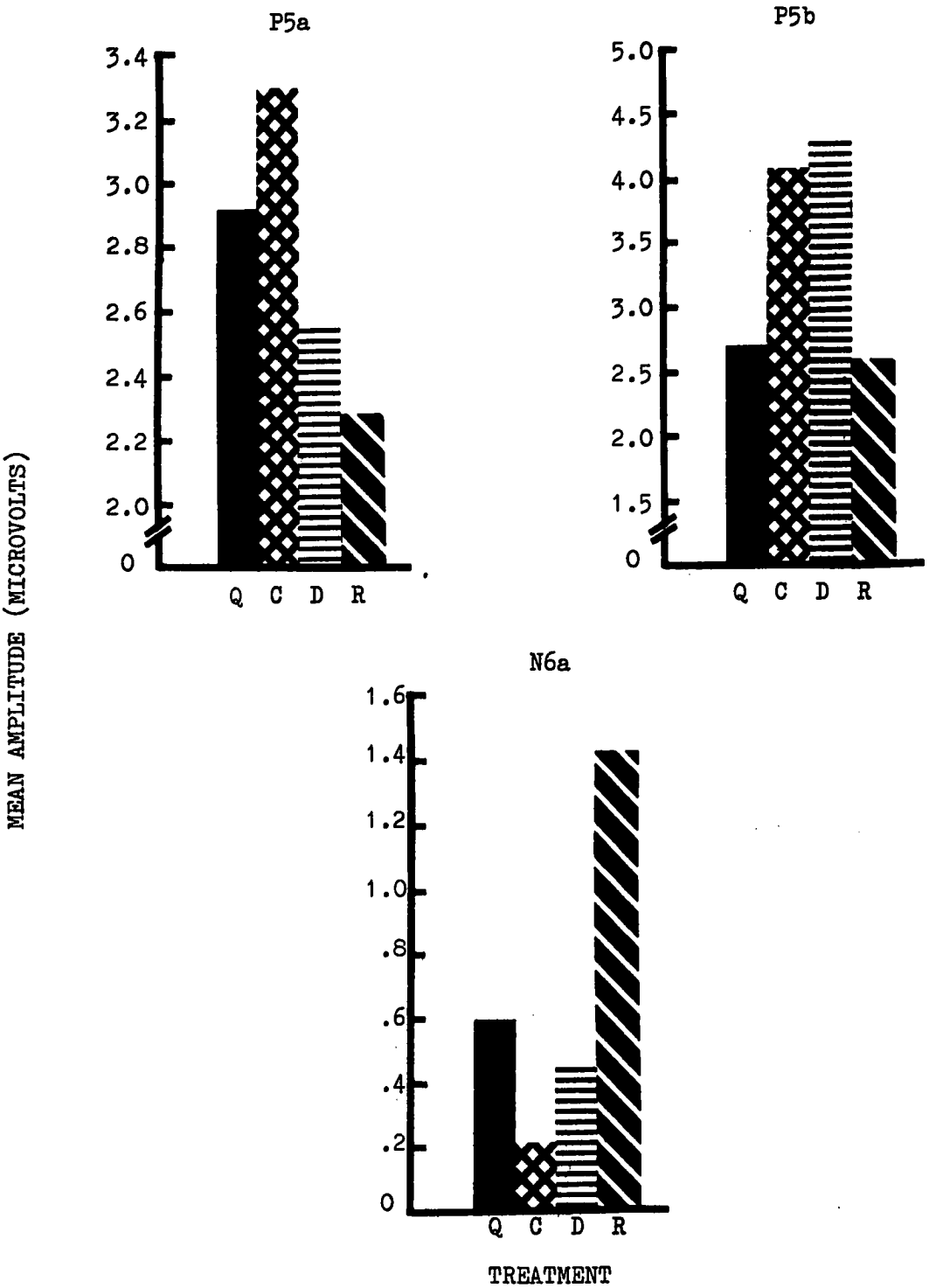


Figure 23.--Mean amplitudes of the P5a, P5b, and N6a components across treatments relative to the zero baseline. (Note different ordinate values.) (Q: quiet, C: counting, D: discriminating, R: reading)

The above results are interesting in that Davis (22) has advanced the idea that the acoustically-evoked response is enhanced only by a task requiring considerable vigilance on the part of the subject, i.e., loudness judgements, as opposed to a less demanding task such as merely responding after each stimulus. The counting condition, it seems, would best be categorized with Davis' responding task. On the other hand, Gross et al. (50) have presented evidence that the less demanding task of counting produces an increase in amplitude relative to a reading task. The results of the present study indicate that both the discriminating and counting conditions produce a significant increase in amplitude relative to the quiet and reading conditions. However, this difference for the counting condition is dependent upon the reference for measurement of the amplitude.

Run Effects

The data for the effect of runs on amplitude is presented in Figures 24 and 25. There was a tendency for Run 1 to produce greater amplitudes for components although the overall AOV did not show any significant differences ($P > .05$). The emergence of this tendency is interpreted to mean that there was some novelty effect, i.e., relatively greater amplitudes were obtained on the first treatment than would have been obtained if that treatment would have been second, third, or fourth.

Interactions

A trial-by-run interaction for the P3b component was the only interaction which showed significance ($P < .05$) on the overall analysis

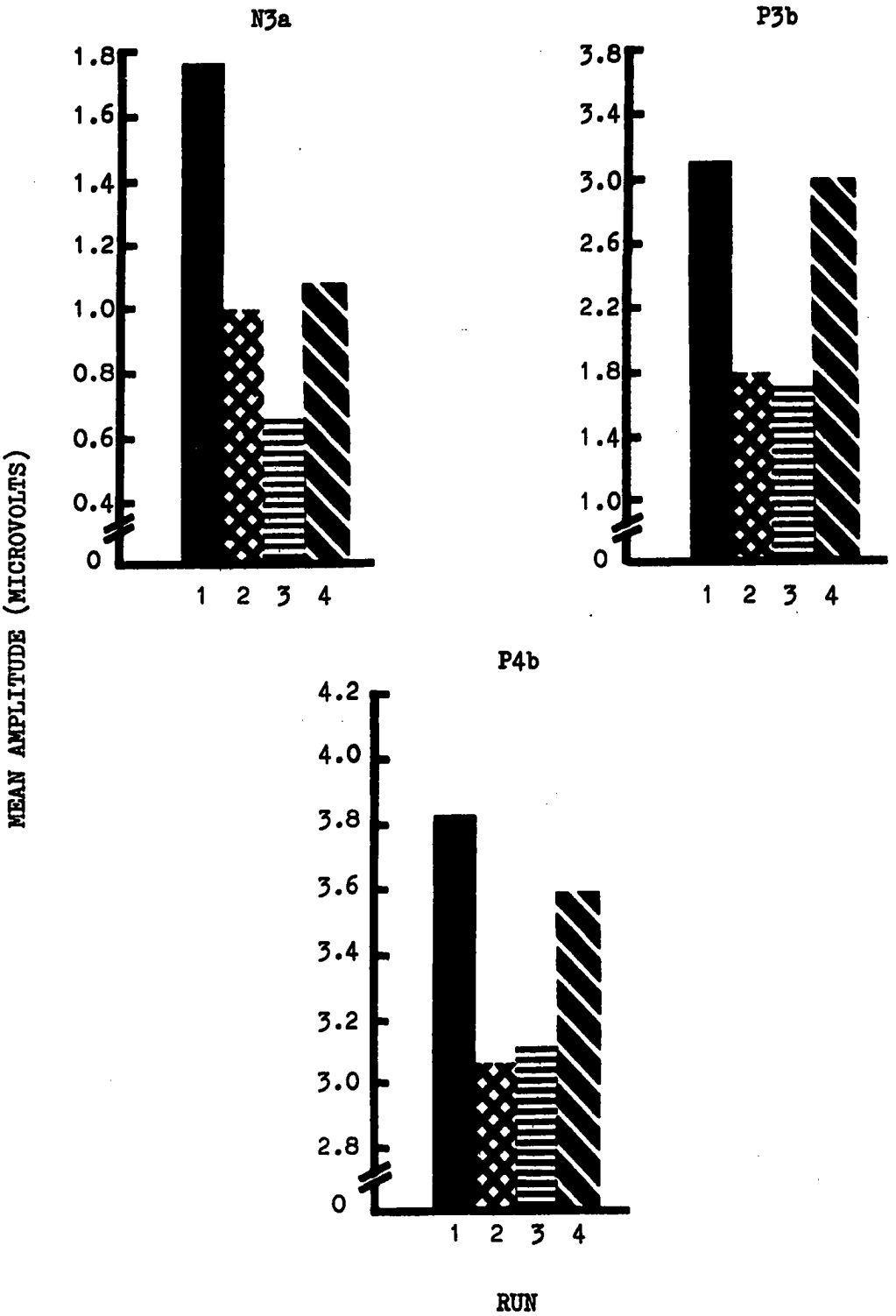


Figure 24.--Mean amplitudes of the N3a, P3b, and P4b components across runs relative to the N4a component. (Note different ordinate values.)

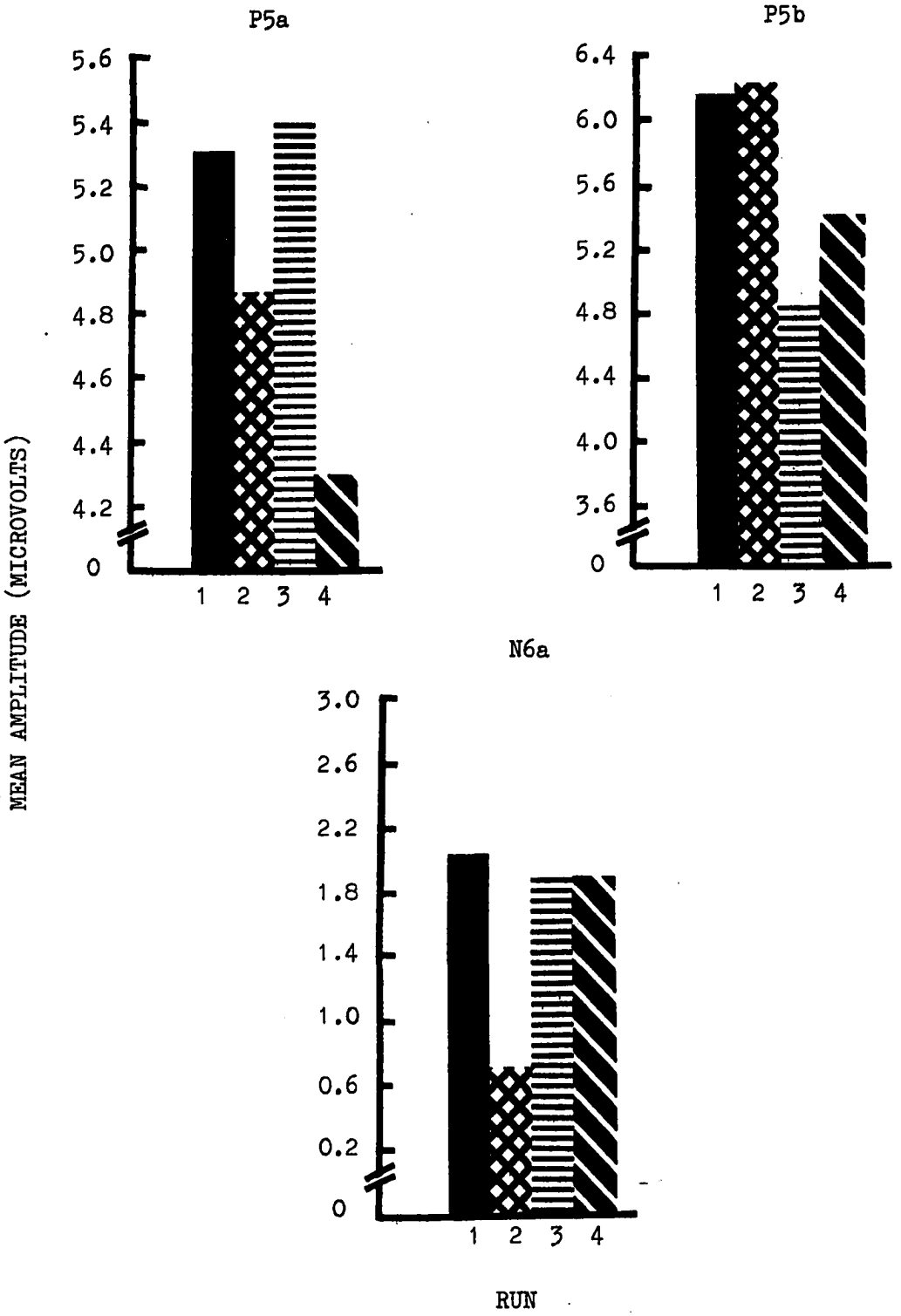


Figure 25.--Mean amplitudes of the P5a, P5b, and N6a components across runs relative to the N4a component. (Note different ordinate values.)

of variance. Because of this significant interaction, an analysis of variance for simple effects on runs was performed. This showed no significant differences ($P > .05$) between runs for each of the four treatments. Thus there were no differences between trials either over all or within individual runs.

Correlations

Simple and partial correlations (97) were computed separately for the variance, latency, and amplitude across the seven components to determine the degree of relationships between each component. These correlations were computed on the mean values of the measurements for each type of data (variance, latency, and amplitude) by treatment and component across subjects.

Correlation of Variances

The simple correlations for variance across components showed that all components under each of the treatments were rather highly correlated for the most part. Many of the correlations were .70 or better and only rarely were they as low as .40. When partial correlations for this same data were computed, i.e., all components fixed except the two under consideration, the results showed that under the counting and discriminating conditions all the components were either highly positively (.92 or better) or highly negatively (-.93 or better) correlated. Under the quiet condition, correlations were not quite as high as under the counting and discriminating conditions, but overall were higher than those observed under the reading condition which showed the least amount of correlation for the four treatments.

From these results it appears that the counting and discriminating tasks create a neurological climate in which the variation in any component is reflected by all other components (as defined in this investigation) in the evoked response.

Correlation of Latencies

When simple correlations were computed for latencies no systematic pattern emerged. Occasionally a correlation in excess of .80 appeared; however, the large majority of the computations resulted in correlations of between -.50 and .60. The partial correlations, on the other hand, revealed a very interesting finding in that the quiet condition resulted in extremely high correlations. All latencies under this condition were either highly negatively correlated (-.97 or better) or highly positively correlated (.97 or better). Under the counting, discriminating, and reading conditions, no pattern appeared to emerge and computed correlations were much smaller than for the quiet condition. These results suggest that under the quiet condition the latencies of the acoustically-evoked response assume a relatively fixed pattern whereas under the conditions requiring some mental task (counting, discriminating, and reading) the temporal-spatial relationships observed under the quiet condition are in some way affected.

Correlation of Amplitudes

The simple correlations resulting from the amplitude data, as with variance and latency data reveal nothing in the way of a pattern. The majority of the computed correlations fall between -.40 and .60. The computed partial correlations for the quiet, counting, and

reading conditions are unremarkable. All the correlations under the discriminating condition, however, are exceedingly high in either a negative (-.94 or better) or positive (.93 or better) direction. Apparently the discriminating condition causes the amplitudes of the herein-defined components to the acoustically-evoked response to become amplitude-locked in some manner which the other conditions fail to show.

Findings

The major positive findings which resulted from this investigation are as follows:

1) The task which the subject is required to perform during the accumulation of the average evoked response affects the variability of the N3a, P3b, N4a, P5b, and N6a components. Generally, the quiet task results in the greatest variability while the reading task produces the least variability.

2) In an experimental session where more than one treatment is applied, the first treatment generally produces less variability at specified components than subsequent treatments. This is especially true for components occurring in the first 130 msec post stimulus.

3) There is a tendency for the estimated silent variance to be greater than the variance for the N3a and P3b components and approximately equal to the other components.

4) There is a tendency for the quiet condition to possess the highest variance across components and silence.

5) The reading condition consistently shows the lowest variance across components and silence.

6) There is a tendency for left ear stimulation to produce longer latencies for components than right ear stimulation in right-handed individuals.

7) The reading condition prolongs the latency of the N3a and P3b components of the response while the discriminating condition shortens the latencies of these two components.

8) The discriminating condition has a tendency to prolong the latency of components occurring after the P3b component.

9) The discriminating condition produces greater amplitude of the P5b component than the quiet, counting, or reading conditions when measured relative to the N4a component.

10) When amplitude is measured relative to the zero voltage baseline the discriminating condition produces greater amplitudes than the other conditions at the P5a component while the discriminating and counting condition produces greater amplitudes than the quiet or reading condition at the P5b component.

11) There is a tendency within an experimental session consisting of more than one treatment for the first treatment to produce relatively greater amplitudes of response than if that treatment would have been subsequently presented.

12) Variance across components tends to be highly correlated within an average acoustically-evoked response, particularly when the subject's attention is directed to the stimulus.

13) Latency of components of an average acoustically-evoked response seems to be fixed rather rigidly across components under a condition where the subject is not required to perform any special mental task.

14) The amplitude of the components of the average acoustically-evoked response tends to be more highly correlated when the attention of the subject is directed to the stimulus.

Suggestions for Future Research

Considerable research has already been conducted as to the nature of evoked potentials; however, the state of the science at this time has as yet not filled all the gaps in our knowledge about this phenomenon. There are suggestions that various components in evoked potentials are contributed by different neurological substrates. With increased knowledge relative to open-brain surgery and the more frequent occurrence of such, the effects of sensory stimulation measured at various substrate levels should be investigated by researchers in accessible positions.

The variance of components of average evoked responses to stimuli other than acoustic stimuli should be investigated and cross-modality comparisons made with the results of the present study. Other methods of investigating this variability should be devised in an attempt to parcel out the variability and assign to the various generators of the response the respective amounts of variability due them.

The effects of different states of attentiveness to stimuli should be further investigated in an attempt to discover whether it is possible to selectively suppress or enhance componentry of the acoustically-evoked response.

CHAPTER V

SUMMARY AND CONCLUSIONS

The recording of EEG activity through the use of an averaging computer immediately following sensory stimulation has provided insights into central nervous system functioning. In recent years considerable information has been published relative to the summing or averaging of specific evoked potentials resulting from repetitive identical stimuli presented to a specific sense modality. Inferences and conclusions relative to these averages have been drawn by various authors, thus generating perhaps more accurate information relative to the manner in which the central nervous system processes sensory information.

Because of the nature of evoked potentials and on-going EEG activity, i.e., on-going activity is approximately 10 times or more larger than acoustically-evoked responses, the evoked potential is sometimes obscured by the magnitude of the other potentials. Thus, it has been necessary to resort to the averaging technique whereby evoked potentials are summed algebraically and grow in magnitude on the record proportionally to the number (N) of samples taken while the non-time-locked on-going activity grows approximately as the square root of N. As a result, studies have not been conducted to observe the individual evoked potentials which make up the average evoked potential.

Experimental Design

The purpose of this study was to investigate the within-average variability of certain specified components which compositely make up the average acoustically-evoked response.

Evoked responses to acoustic clicks at 50-dB sensation level were obtained. Clicks of 0.1 msec duration were presented every four seconds alternately to the left and right ears of eight normal-hearing adult subjects. A total of 100 clicks were presented to each ear to obtain an average. The EEG activity for left and right ear stimulation was stored on separate channels of a multi-channel FM tape recorder along with a trigger which enabled the separation of the same during the analysis phase of the study.

Separate average evoked responses were obtained from each subject while the subject was engaged in four different tasks designed to alter his attention relative to the stimulus. The four tasks were as follows: sitting quietly, eyes open; sitting quietly, counting each click; sitting quietly, discriminating 10-dB intensity increments; and, sitting quietly, reading.

The experimental procedure followed a four-treatment four-period change-over design applied within a two-by-two factorial arrangement with repeated measures on each factor. The average evoked response was, therefore, recorded from each subject under 16 (4 x 2 x 2) different experimental conditions. These conditions represent treatments and ears under the test and retest sessions.

After all the data were collected and stored on tape, they were retrieved through the variance computer and averaging computer and

written out on calibrated graph paper from which the variance, mean amplitude and mean latency of each component was measured. The variance of silent periods was estimated from a write-out by sampling the on-going EEG activity just prior to the onset of the acoustic stimulus.

Results and Conclusions

The results of this investigation show that there is no significant difference between the variability of on-going EEG activity sampled at a time when acoustic stimulation is not present as opposed to the EEG activity sampled immediately following the onset of an acoustic stimulus when the on-going activity is contaminated by the presence of the acoustically-evoked response. Although these data were non-significant, there was a tendency for the estimated silent variances to exceed the variances for the two defined components occurring before 85-msec post stimulation, while those occurring later did not reveal this tendency. It may be, then, that the estimation of the variance of the peak components and the silent periods were poor; or, that the presence of the stimulus had a short-time stabilizing effect on the brain potentials. In addition, the N4a component (approximately 85 msec) tended to show greater variability than the other components while the N3a and P3b components had a tendency to produce the least variability although differences were not significant. The variability of components was affected by the task which the subject was required to perform during the accumulation of an average. The quiet condition generally produced the greatest variability while the reading condition resulted in the least variability. This same phenomenon was reflected in the estimation of silent variances for each of the conditions. It is,

therefore, concluded that the variability of brain potentials is less when the subject is required to perform some specified mental task than when he is not required to perform specified mental manipulations. It was also found that in an experimental session there is less variability of the individual components at the beginning of the session as opposed to the end of the session. It is likely that fatigue and restlessness give rise to sources of potential contamination and thus the EEG and evoked potentials are more stabile during the initial stages of an experimental session than later in the session.

The latency of the defined components was affected only by the treatments applied. An interpretation of this finding is that these differences result from the activation of different undefined neurological substrates incorporated for the processing of information contained in the stimulus, depending upon the subject task required by the treatment.

The results of these data support previous findings (22, 50) that tasks requiring attention produce a greater amplitude in the average response than do tasks which do not require the subject to pay attention to the signal. Davis (22), however, has maintained that a vigilance task, such as discriminating intensity increments is required to enhance the amplitude. It is concluded from the results of this investigation that both counting and discriminating activities produce enhanced amplitudes of the P5a (approximately 160 msec) and P5b (approximately 190 msec) components depending upon the reference of measurement.

It will be obvious that the conditions of the present

experiment do not begin to tap the possible environmental effects on the variability of the evoked response. It is not unlikely, for example, that more difficult discrimination tasks than were used would serve to enhance responsivity to an even greater extent than was observed in the present study. Electroencephalic responsivity to stimuli such as those employed in signal detection procedures might well be useful in further clarifying the substrates underlying auditory behavior.

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APPENDIX A

CALIBRATION PROCEDURES

Recording Instrumentation

Step 1. Make sure equipment has been on and allowed to warm up for at least one hour.

Step 2. Adjust output of audio oscillator (Hewlett-Packard 201C)

(a) Connect output to input of Darcy DMM (VAC) in parallel with EXT. CAL. Input of P511.

(b) Adjust attenuator to read 1.73v RMS.

Step 3. Calibrate the gain of the P511 amplifiers.

(a) Set Input switch to CAL.

(b) Set Fall Time Constant switch to 1.

(c) Set Amplification switch to 50 x 1000

(d) Set Rise Time Constant switch to .3.

(e) Connect output of oscillator (100 Hz at 1.73v RMS) to EXT. CAL input.

(f) Connect output of P511 to DMM (VAC) and read 1.76v by adjusting ADJ. CAL. knob.

Step 4. Set P511 to operating position.

(a) Set Input switch to USE.

(b) Set Calibrator switch to 200 μv.

(c) Set CAL. switch to A.C.

(d) Set Fall Time Constant switch to 1.

(e) Set Amplification switch to 50 x 1000.

(f) Set Rise Time Constant switch to .1.

The calibration procedure for the CAT, FM Tape System, and X-Y plotter were supplied by the respective manufacturers.

Analysis Instrumentation

Step 1. Make sure equipment has been on and allowed to warm up for at least one hour.

Step 2. Set the DC level of the 60-Hz filter (A.P. Circuit Corporation, APN-60).

(a) Short the input to ground.

(b) Connect output to DMM (Darcy 440).

(c) Set DMM on VDC.

(d) Adjust DC pot on filter to read Ov on DMM.

The calibration procedure for the CAT, variance computer, FM tape system, oscilloscope, and X-Y plotter were supplied by the respective manufacturers.

Step 3. Calibrate the gain of the variance computer (Bio-Data Corporation Model 204) for a gain of 4.

(a) Connect output of audio oscillator (Hewlett-Packard 201C) to input of oscilloscope (Tektronics 561A).

(b) Adjust output to read 0.5 VAC on oscilloscope.

(c) Connect output of audio oscillator to input of variance computer.

(d) Connect output of variance computer to input of oscilloscope.

(e) Adjust amplification pot on variance computer to read 2 VAC on oscilloscope.

Record Calibration Signal

Step 1. Calibrate frequency output of audio oscillator (Hewlett-Packard 201C) for 40 Hz.

(a) Connect output of audio oscillator to input of interval timer and universal counter (TSI Model 361).

(b) Adjust frequency control on audio oscillator to read 40 Hz on TSI.

Step 2. Calibrate P511 amplifier for 25,000 gain. (See Step 3, Recording Instrumentation.)

Step 3. Connect output of audio oscillator to 160-ohm voltage splitter.

Step 4. Connect one side of voltage splitter to a 500-ohm load in parallel with input to an input converter (Grason-Staddler 1211) set for sine wave operation. Input converter triggers CAT and CAT trigger units (Grason-Staddler 1056A) to put triggers on tape.

Step 5. Connect the other side of the voltage splitter to attenuator (Hewlett-Packard 350 AR).

Step 6. Connect output of H. P. attenuator to microvolter (General Radio Type 548C) set for 20 μv output.

Step 7. Load microvolter output with 600 ohms to input of P511 amplifier.

Step 8. Output of P511 amplifier to input of data converter (Mnemotron M1000).

Step 9. Parallel output of data converter to monitor scope and CAT monitor.

Step 10. Adjust output of audio oscillator for 2.2v input to microvolter.

Step 11. Record the calibration signal.

Step 12. Play back the recorded 20μv signal through the data converter (Mnemotron M1000) to the 60-Hz filter (A. C. Circuit Corporation APN-60) to the variance computer (Bio-Data 204) and the CAT. (CAT is triggered by 1056A.) Accumulate 100 samples.

Calculation of System Amplification and Readout

Step 1. Determine the gain of the system for the average readout.

(a) Write out, via the X-Y plotter the 100 accumulations of the 20μv signal.

(b) Determine the number of squares covered on the calibrated graph paper (50).

(c) Divide the number of squares by 2 to obtain the value for 10μv input (25).

(d) Divide 10μv by 25 to determine the value for one square (.4μv).

Step 2. Determine the gain of the system for the variance readout.

(a) Determine the number of divisions covered by the 2.25v calibration signal supplied internally by the variance computer (4.5).

(b) Determine the voltage represented by one division (.5v).

(c) Divide .5v by 16, the square of the gain factor (0.3125v).

(d) Divide .03125v by 25,000, the gain of the system during the recording phase (1.25μv).

(e) Divide 1.25μv by 10 to determine the value of 1/10th of a division (.125μv).

APPENDIX B

INDIVIDUAL SUBJECT DATA

TABLE 8

INDIVIDUAL VARIANCE DATA FOR SUBJECT NUMBER ONE

Treatment	Trial	Ear	Variance of Component in Microvolts							
			N3a	P3b	N4a	P4b	P5a	P5b	N6a	Silent
Quiet	1	L	1.20	1.07	1.49	1.56	1.08	1.31	1.51	1.32
Quiet	2	L	0.76	0.72	0.78	0.96	1.16	1.08	1.18	1.46
Quiet	1	R	0.96	1.22	1.08	0.81*	1.22	1.18	1.01	1.30
Quiet	2	R	0.76	0.76	1.00	0.90	1.18	1.16	1.20	1.41
Count.	1	L	1.68	1.78	1.59	1.60	1.92	1.52	1.76	1.20
Count.	2	L	1.08	1.40	1.40	1.56	1.38	1.36	1.88	1.39
Count.	1	R	1.22	1.18	1.32	1.16	1.18	1.20	1.28	1.45
Count.	2	R	1.32	1.71	1.85	1.32	1.17	1.20	1.50	1.37
Discrim.	1	L	0.98	1.14	1.22	1.14	1.32	1.20	1.30	1.30
Discrim.	2	L	0.94	0.72	1.10	1.00	1.02	1.16	1.06	1.33
Discrim.	1	R	0.84	1.08	1.12	0.92	1.22	0.98	1.24	1.37
Discrim.	2	R	2.00	1.92	1.40	1.62	1.54	1.42	1.42	1.38
Read.	1	L	1.52	1.60	1.38	1.70	1.66	1.62	1.32	1.37
Read.	2	L	0.82	0.72	1.32	1.18	0.86	0.82	0.98	1.36
Read.	1	R	0.84	0.96	1.11	0.84	1.20	1.00	1.04	1.46
Read.	2	R	1.03	1.24	1.18	1.18	1.28	1.24	1.44	1.40

*Estimated data

TABLE 9

INDIVIDUAL VARIANCE DATA FOR SUBJECT NUMBER TWO

Treatment	Trial	Ear	Variance of Component in Microvolts							
			N3a	P3b	N4a	P4b	P5a	P5b	N6a	Silent
Quiet	1	L	2.44	2.52	2.04	2.30	2.20	2.90	1.80*	2.52
Quiet	2	L	2.00	----	1.92	2.35*	2.46	2.40	2.40	1.78
Quiet	1	R	----	----	2.70	2.04	1.68	2.77*	2.28*	2.48
Quiet	2	R	1.30	1.60	2.12	2.20*	2.26	2.17	2.20*	1.92
Count.	1	L	----	2.01	1.92	2.32	2.10	2.48	2.76	2.34
Count.	2	L	----	2.28	2.58	2.52	2.16	2.60	2.52	2.53
Count.	1	R	----	----	2.22	2.34*	2.40	2.37	3.48	2.22
Count.	2	R	2.88	----	2.14	2.51*	2.86	2.62	2.70	2.15
Discrim.	1	L	2.40	2.32	3.08	2.70*	2.80	2.72	2.00*	2.94*
Discrim.	2	L	----	----	2.28	2.76	2.04	2.76	2.44	1.94
Discrim.	1	R	----	----	3.20	3.24	3.24	3.12	4.18	2.93
Discrim.	2	R	2.04	----	1.86	1.96	1.52	1.74	2.28	2.22
Read.	1	L	1.80	----	1.92	2.40	2.04	2.40	2.40	2.67
Read.	2	L	1.44	----	1.52	1.76	1.62	1.60	1.38	1.32
Read.	1	R	2.40	2.34	2.40	2.60	2.34	1.80	2.16	2.06
Read.	2	R	----	----	1.32	1.48*	1.50	1.52	1.80	1.42

*Estimated data

TABLE 10

INDIVIDUAL VARIANCE DATA FOR SUBJECT NUMBER THREE

Treat-ment	Trial	Ear	Variance of Component in Microvolts							
			N3a	P3b	N4a	P4b	P5a	P5b	N6a	Silent
Quiet	1	L	----	1.85	2.16	1.82	2.00	2.40	1.70	2.10
Quiet	2	L	----	1.36	1.56	1.08	1.08	1.92	1.80*	1.22
Quiet	1	R	----	2.12	2.94	1.91*	2.40	1.44	1.92	2.23
Quiet	2	R	----	1.17	1.92	1.32	1.56	1.18	1.84	1.41
Count.	1	L	1.92	1.68	1.80	1.50*	2.60	3.16	2.46	2.25
Count.	2	L	----	1.36	1.56	1.20	1.14	1.50	1.96	1.18
Count.	1	R	----	1.98*	2.08	2.16	2.76	1.72	2.40	2.30
Count.	2	R	1.28	1.39	1.46	1.56	1.84	1.20	1.74	1.08
Discrim.	1	L	1.50	1.40	0.96	1.02*	1.12*	1.02	1.06*	1.42
Discrim.	2	L	1.08	1.00	1.20	1.23	0.96	1.27	1.18*	1.08
Discrim.	1	R	----	1.46	1.56	1.91*	1.70	1.80	2.04	1.46
Discrim.	2	R	----	1.76	1.58	1.70*	1.28	1.53*	1.80*	1.09
Read.	1	L	0.96	1.06	1.32	1.32	1.08	1.24	1.20	1.90
Read.	2	L	1.82	1.28	2.20	2.00	1.75*	0.50*	1.60*	1.82
Read.	1	R	----	1.80	1.80	1.80	1.44	1.64	1.80	1.90
Read.	2	R	1.84	1.84	1.42	1.72	2.00	2.04	2.08	1.89

*Estimated data

TABLE 11

INDIVIDUAL VARIANCE DATA FOR SUBJECT NUMBER FOUR

Treatment	Trial	Ear	Variance of Component in Microvolts							
			N3a	P3b	N4a	P4b	P5a	P5b	N6a	Silent
Quiet	1	L	1.92	1.90	1.92	1.81	1.92	1.70	1.84	1.88
Quiet	2	L	2.28	2.40	2.58	2.58	2.58	2.42	1.92	2.02
Quiet	1	R	2.24	1.86*	2.12	1.98	2.11	1.92	1.72	1.92
Quiet	2	R	2.40	2.18	2.16	1.94	2.16	1.92	2.04	2.48
Count.	1	L	1.64	1.32	1.60	1.50	1.32	1.17	1.68	1.62
Count.	2	L	1.80	1.80	1.75	1.62	2.00	1.92	2.00	1.85
Count.	1	R	1.50	1.42	1.40	1.31*	1.56	1.18	1.58	1.50
Count.	2	R	2.02	2.16	2.00	2.28	1.80	1.70*	1.72	1.80
Discrim.	1	L	1.00	1.56	1.62	1.58*	1.40	1.72	1.40	1.66
Discrim.	2	L	1.62	1.64	2.10	1.88	1.56	1.80	2.08	1.84
Discrim.	1	R	1.64	1.58	1.94	1.92	1.50	1.68*	1.58	1.66
Discrim.	2	R	1.44	1.68	2.36	2.10	1.60	1.32	1.70	1.73
Read.	1	L	1.12	0.98	1.56	1.54	1.47	1.47	1.28	1.12
Read.	2	L	1.62	1.77	1.62	1.28	1.38	1.82	1.28	1.68
Read.	1	R	1.32	1.00	1.20	1.08	1.29	0.96	1.38	1.22
Read.	2	R	2.10	1.64	2.14	1.92	1.70	1.74	1.62	1.67

*Estimated data

TABLE 12

INDIVIDUAL VARIANCE DATA FOR SUBJECT NUMBER FIVE

Treatment	Trial	Ear	Variance of Component in Microvolts							
			N3a	P3b	N4a	P4b	P5a	P5b	N6a	Silent
Quiet	1	L	2.40	2.40	1.92	1.93	1.94	2.25*	----	2.19
Quiet	2	L	3.40	3.84	4.62	3.80	3.84	3.84	----	3.21
Quiet	1	R	2.59	----	3.00	2.12	1.62	----	----	2.69
Quiet	2	R	4.08	3.84	3.46	----	3.20	3.60	----	3.89
Count.	1	L	2.76	2.40	2.52	2.78	2.10	----	----	2.43
Count.	2	L	5.28	4.85	5.28	4.18	3.36	----	----	4.36
Count.	1	R	2.74	2.76	2.21	2.40	2.88	2.55	2.45	2.42
Count.	2	R	4.68	4.70	4.20	----	3.52	----	----	4.10
Discrim.	1	L	1.80	1.84	2.28	----	2.90	3.96	----	2.24
Discrim.	2	L	3.60	4.80	6.84	----	3.08	3.14	----	3.52
Discrim.	1	R	2.00	2.34	3.48	----	2.88	3.40	2.16	2.38
Discrim.	2	R	3.36	4.32	3.65	----	2.28	3.06	----	3.81
Read.	1	L	2.00	----	2.36	----	1.92	----	----	1.80
Read.	2	L	3.60	----	3.60	----	3.38	----	4.56	3.60
Read.	1	R	----	----	----	1.63	1.80	1.94	2.25	2.06
Read.	2	R	3.80	4.10	2.96	----	3.74	3.64	3.65	4.09

*Estimated data

TABLE 13

INDIVIDUAL VARIANCE DATA FOR SUBJECT NUMBER SIX

Treatment	Trial	Ear	Variance of Component in Microvolts							
			N3a	P3b	N4a	P4b	P5a	P5b	N6a	Silent
Quiet	1	L	2.24	2.08	2.12	2.24	2.10	1.80	----	2.00
Quiet	2	L	2.28	2.16	2.31	2.30	2.10	----	----	2.16
Quiet	1	R	1.80	1.74	1.92	2.22	2.72	2.20	2.28	2.37
Quiet	2	R	1.56	1.80	1.78	1.72	1.80	----	----	2.18
Count.	1	L	1.68	1.68	1.64	1.56	1.56	1.68	----	1.84
Count.	2	L	1.32	1.38	1.35	1.38	1.27	1.62	----	1.54
Count.	1	R	1.92	1.62	1.60	1.48	1.46	2.00	2.28	1.83
Count.	2	R	1.80	1.52	1.95	2.16	2.25	2.14	2.16	1.86
Discrim.	1	L	1.77	1.60	1.71	1.68	1.54	2.09	2.00	1.77
Discrim.	2	L	1.82	1.44	1.68	1.48	1.54	1.40	1.70	2.32
Discrim.	1	R	2.00	1.86	2.40	2.40	----	1.58	----	1.69
Discrim.	2	R	2.04	2.00	1.76	1.62	1.86	2.16	2.32	1.99
Read.	1	L	1.36	1.56	1.62	1.30	----	1.62	----	1.22
Read.	2	L	0.84	0.84	0.92	1.00	----	0.78	----	1.06
Read.	1	R	1.32	1.17	1.01	1.32	1.92	1.40	1.20	1.67
Read.	2	R	1.23	1.30	1.66	1.38	1.54	----	1.08	1.15

TABLE 14

INDIVIDUAL VARIANCE DATA FOR SUBJECT NUMBER SEVEN

Treatment	Trial	Ear	Variance of Component in Microvolts							
			N3a	P3b	N4a	P4b	P5a	P5b	N6a	Silent
Quiet	1	L	2.20	2.64	1.80	2.50	1.75	2.22	2.64	2.47
Quiet	2	L	1.50*	0.92	1.26	----	1.26	1.00	0.84	1.67
Quiet	1	R	1.56	1.58	1.86	----	2.72	2.40	2.72	2.26
Quiet	2	R	1.19*	1.62	1.52	----	1.98	----	1.56	1.37
Count.	1	L	1.64	1.92	1.60	1.56	1.40	1.50	1.77	1.92
Count.	2	L	1.61*	1.15*	1.08	1.08	1.00	1.02	1.23	1.33
Count.	1	R	2.03*	1.48	1.32	----	1.50	1.00	1.26	1.40
Count.	2	R	1.08	0.96	0.80	0.86	0.74	1.02	1.24	1.42
Discrim.	1	L	1.62	1.88	1.24	1.72	1.64	1.98	1.44	1.52
Discrim.	2	L	1.10*	0.78	1.26	1.10	0.82	0.78	1.00	1.19
Discrim.	1	R	1.54	1.59*	2.08	----	1.56	1.56	2.12	1.56
Discrim.	2	R	1.23*	0.92*	0.80	0.72	0.86	0.98	0.90	1.11
Read.	1	L	1.32	1.48	0.96	1.50	0.84	1.08	1.16	1.22
Read.	2	L	1.20	1.12*	0.90	1.08	1.20	0.98	1.14	1.31
Read.	1	R	1.16	1.22*	0.84	1.08	1.40	----	0.87	1.32
Read.	2	R	1.02	0.95	1.06	1.24	0.94	----	0.87	1.40

*Estimated data

TABLE 15

INDIVIDUAL VARIANCE DATA FOR SUBJECT NUMBER EIGHT

Treatment	Trial	Ear	Variance of Component in Microvolts							
			N3a	P3b	N4a	P4b	P5a	P5b	N6a	Silent
Quiet	1	L	1.84	1.56	2.44	2.44	2.11	2.28	1.88	2.26
Quiet	2	L	2.16	1.92	1.86	1.84	1.70	2.44	----	2.03
Quiet	1	R	2.28	1.72	1.60	2.40	2.16	2.26	----	2.31
Quiet	2	R	----	1.84	2.25	----	1.52	2.32	----	2.62
Count.	1	L	----	----	2.76	2.93	----	2.24	2.40	1.86
Count.	2	L	2.18	----	2.28	3.04	2.36	2.70	2.32	2.14
Count.	1	R	----	----	1.44	----	----	2.68	2.16	2.44
Count.	2	R	1.70	----	1.32	1.64	1.98	1.68	1.80	1.96
Discrim.	1	L	----	2.30	1.80	----	----	2.68	2.40	1.90
Discrim.	2	L	2.16	2.52	2.36	----	1.62	2.35	2.28	2.02
Discrim.	1	R	2.06	----	2.52	1.82	2.40	2.48	2.22	1.82
Discrim.	2	R	1.62	1.80	2.72	2.80	2.32	2.34	3.00	1.95
Read.	1	L	0.84	1.00	0.88	1.16	----	1.08	1.02	1.11
Read.	2	L	1.48	1.40	1.18	1.20	----	1.02	1.36	1.24
Read.	1	R	0.92	0.70	1.20	1.17	1.08	1.00	0.96	1.06
Read.	2	R	1.05	1.20	1.16	1.16	1.27	1.00	1.08	1.24

TABLE 16

INDIVIDUAL LATENCY DATA FOR SUBJECT NUMBER ONE

Treatment	Trial	Ear	Latency of Component in Milliseconds						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	45	66	92	130	160	198	253
Quiet	2	L	45	55	96	128	157	180	253
Quiet	1	R	42	56	90	122*	148	178	237
Quiet	2	R	45	55	95	126	162	176	234
Counting	1	L	44	58	81	122	157	182	227
Counting	2	L	43	63	90	117	150	168	243
Counting	1	R	40	53	88	126	155	179	260
Counting	2	R	42	52	78	135	153	183	248
Discrim.	1	L	40	57	102	128	162	180	232
Discrim.	2	L	42	51	97	132	157	193	252
Discrim.	1	R	42	60	85	137	162	176	267
Discrim.	2	R	42	55	87	145	176	200	265
Reading	1	L	42	62	102	137	158	170	237
Reading	2	L	45	55	97	140	163	170	272
Reading	1	R	43	57	90	120	154	173	195
Reading	2	R	45	57	100	125	164	175	255

*Estimated data

TABLE 17

INDIVIDUAL LATENCY DATA FOR SUBJECT NUMBER TWO

Treatment	Trial	Ear	Latency of Component in Milliseconds						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	36	50	90	---	160	218*	257
Quiet	2	L	45	--	86	---	137	192	255
Quiet	1	R	--	--	90	125	165	185*	248
Quiet	2	R	40	57	95	---	162	174*	257
Counting	1	L	--	55	100	140	172	192	235
Counting	2	L	--	60	105	158	188	210	270
Counting	1	R	--	--	104	---	155	177	220
Counting	2	R	40	--	95	---	150	167	200
Discrim.	1	L	47	67	125	---	172	200	234*
Discrim.	2	L	--	--	90	127	163	197	247
Discrim.	1	R	--	--	122	163	185	205	263
Discrim.	2	R	47	--	125	165	178	197	252
Reading	1	L	40	--	115	155	180	210	260
Reading	2	L	42	--	103	147	180	212	242
Reading	1	R	43	65	95	133	152	173	236
Reading	2	R	--	--	93	---	150	178	235

*Estimated data

TABLE 18

INDIVIDUAL LATENCY DATA FOR SUBJECT NUMBER THREE

Treatment	Trial	Ear	Latency of Component in Milliseconds						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	--	58	102	125	150	170	222
Quiet	2	L	--	58	100	128	150	190	---
Quiet	1	R	--	50	110	127*	155	190	248
Quiet	2	R	--	52	99	137	155	175	217
Counting	1	L	46	52	102	124*	157	175	213
Counting	2	L	--	50	100	126	150	174	225
Counting	1	R	--	49*	101	132	146	170	218
Counting	2	R	47	52	95	123	145	172	215
Discrim.	1	L	42	50	93	135*	---	186	---
Discrim.	2	L	42	48	100	145	175	200	---
Discrim.	1	R	--	53	120	134*	182	240	340
Discrim.	2	R	--	50	135	148*	195	191*	333
Reading	1	L	45	56	105	140	162	207	293
Reading	2	L	40	58	107	135	---	171*	---
Reading	1	R	--	65	100	122	175	203	262
Reading	2	R	42	50	103	145	168	200	252

*Estimated data

TABLE 19

INDIVIDUAL LATENCY DATA FOR SUBJECT NUMBER FOUR

Treatment	Trial	Ear	Latency of Component in Milliseconds						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	40	60	93	124	155	183	220
Quiet	2	L	43	54	92	135	170	188	246
Quiet	1	R	43	45*	97	125	150	193	228
Quiet	2	R	42	50	95	120	154	185	228
Counting	1	L	40	55	97	125	170	195	233
Counting	2	L	42	57	90	127	167	212	282
Counting	1	R	40	55	90	132*	138	175	230
Counting	2	R	45	53	93	125	153	---	206
Discrim.	1	L	45	55	98	134*	150	183	296
Discrim.	2	L	46	55	100	127	168	197	275
Discrim.	1	R	42	50	98	124	164	---	243
Discrim.	2	R	40	55	93	127	160	182	245
Reading	1	L	43	57	101	137	165	215	308
Reading	2	L	44	55	101	132	147	180	237
Reading	1	R	40	60	100	135	160	172	218
Reading	2	R	45	60	100	135	168	197	255

*Estimated data

TABLE 20

INDIVIDUAL LATENCY DATA FOR SUBJECT NUMBER FIVE

Treatment	Trial	Ear	Latency of Component in Milliseconds						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	40	52	100	128	153	---	---
Quiet	2	L	40	50	100	126	147	175	---
Quiet	1	R	40	--	90	125	144	---	---
Quiet	2	R	40	50	95	---	150	170	---
Counting	1	L	38	55	93	137	157	---	---
Counting	2	L	40	55	92	127	158	---	---
Counting	1	R	40	52	95	130	150	170	273
Counting	2	R	43	60	95	---	148	---	---
Discrim.	1	L	38	56	110	---	175	197	---
Discrim.	2	L	47	53	115	---	174	205	---
Discrim.	1	R	40	50	110	---	158	218	270
Discrim.	2	R	42	53	107	---	165	202	---
Reading	1	L	55	--	82	---	137	---	---
Reading	2	L	50	--	90	---	137	---	193
Reading	1	R	--	--	95	121	140	165	---
Reading	2	R	40	50	81	---	137	155	182

TABLE 21

INDIVIDUAL LATENCY DATA FOR SUBJECT NUMBER SIX

Treatment	Trial	Ear	Latency of Component in Milliseconds						
			N3a	P3b	N3a	P4b	P5a	P5b	N6a
Quiet	1	L	43	55	92	126	167	192	---
Quiet	2	L	40	55	83	110	148	---	---
Quiet	1	R	45	52	90	115	142	172	218
Quiet	2	R	45	50	95	112	137	---	---
Counting	1	L	44	55	88	120	142	185	---
Counting	2	L	45	55	88	117	155	184	---
Counting	1	R	43	55	90	110	138	217	270
Counting	2	R	44	53	93	110	143	200	240
Discrim.	1	L	40	50	85	135	167	197	275
Discrim.	2	L	42	54	87	135	162	196	265
Discrim.	1	R	41	48	89	137	---	190	---
Discrim.	2	R	43	50	92	140	150	190	283
Reading	1	L	43	55	100	135	---	200	---
Reading	2	L	45	53	105	127	---	188	---
Reading	1	R	40	56	87	130	145	187	238
Reading	2	R	45	52	86	128	155	---	187

TABLE 22

INDIVIDUAL LATENCY DATA FOR SUBJECT NUMBER SEVEN

Treatment	Trial	Ear	Latency of Component in Milliseconds						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	43	54	100	139	160	188	233
Quiet	2	L	34*	60	100	---	145	167	225
Quiet	1	R	47	52	110	---	150	173	248
Quiet	2	R	41*	57	102	---	155	---	210
Counting	1	L	40	48	117	127	145	185	220
Counting	2	L	36*	--	115	132	154	182	224
Counting	1	R	45*	--	105	---	145	173	225
Counting	2	R	40	50	114	131	143	172	220
Discrim.	1	L	43	51	100	140	155	180	226
Discrim.	2	L	39*	52	112	125	163	185	213
Discrim.	1	R	40	--	115	---	150	180	222
Discrim.	2	R	38*	--	107	137	155	175	220
Reading	1	L	46	52	105	137	165	183	232
Reading	2	L	39	--	110	130	145	170	218
Reading	1	R	49	--	106	142	172	---	217*
Reading	2	R	40	50	123	133	173	---	253*

*Estimated data

TABLE 23

INDIVIDUAL LATENCY DATA FOR SUBJECT NUMBER EIGHT

Treatment	Trial	Ear	Latency of Component in Milliseconds						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	37	60	100	147	171	218	285
Quiet	2	L	38	57	95	155	180	213	---
Quiet	1	R	41	57	107	132	158	183	---
Quiet	2	R	--	52	112	---	148	183	---
Counting	1	L	--	--	100	133	---	193	247
Counting	2	L	38	--	82	130	157	190	244
Counting	1	R	--	--	85	---	---	197	233
Counting	2	R	44	--	93	135	157	188	225
Discrim.	1	L	--	66	112	---	---	187	285
Discrim.	2	L	37	62	120	---	165	197	292
Discrim.	1	R	45	--	110	157	182	213	276
Discrim.	2	R	38	52	123	153	172	188	248
Reading	1	L	36	64	102	140	---	205	298
Reading	2	L	38	60	101	147	---	193	290
Reading	1	R	35	60	90	136	160	190	260
Reading	2	R	38	56	94	133	150	180	265

TABLE 24

INDIVIDUAL AMPLITUDE DATA FOR SUBJECT NUMBER ONE

Treatment	Trial	Ear	Amplitude of Component in Microvolts						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	-1.1	0.4	-1.6	2.1	3.3	2.7	-0.4
Quiet	2	L	-1.2	0.2	-2.2	1.8	3.4	2.1	-1.2
Quiet	1	R	-1.2	0.4	-0.8	3.2*	5.2	4.0	-1.8
Quiet	2	R	-0.2	0.8	-0.8	1.2	3.4	3.0	0.5
Counting	1	L	-1.3	0.1	-1.4	2.0	3.9	3.4	-1.3
Counting	2	L	-0.6	0.2	-0.8	0.9	3.8	3.5	-2.0
Counting	1	R	-1.6	0.2	-1.8	2.3	4.0	3.1	-1.8
Counting	2	R	-0.4	0.8	-0.4	3.1	4.3	3.2	-0.8
Discrim.	1	L	-0.2	0.9	-0.6	3.1	5.0	4.1	1.1
Discrim.	2	L	-1.0	-0.1	-2.9	1.1	2.8	2.4	-1.0
Discrim.	1	R	-2.1	-0.6	-2.5	2.8	4.4	3.9	0.1
Discrim.	2	R	-1.5	-0.4	-2.0	2.4	3.3	2.2	-1.2
Reading	1	L	-0.2	1.4	-1.3	2.2	3.2	2.9	-0.4
Reading	2	L	0.0	0.4	-2.7	1.4	2.6	2.9	-1.9
Reading	1	R	-1.4	0.9	-0.8	1.1	2.7	2.3	0.7
Reading	2	R	-0.5	1.5	-0.4	0.9	2.3	2.2	-1.5

*Estimated data

TABLE 25

INDIVIDUAL AMPLITUDE DATA FOR SUBJECT NUMBER TWO

Treatment	Trial	Ear	Amplitude of Component in Microvolts						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	0.3	0.8	-1.2	---	3.0	1.0*	-2.4
Quiet	2	L	-0.6	---	-2.0	---	0.7	1.7	-0.2
Quiet	1	R	---	---	-1.2	0.5	5.3	2.7*	-2.4
Quiet	2	R	-1.2	-0.7	-2.8	---	1.8	2.9*	-1.3
Counting	1	L	---	0.3	-2.2	0.6	4.8	3.7	-0.7
Counting	2	L	---	0.8	-1.2	2.4	3.7	2.5	-0.4
Counting	1	R	---	---	-2.4	---	3.5	5.5	-0.1
Counting	2	R	1.7	---	-2.7	---	2.0	2.8	1.5
Discrim.	1	L	0.2	1.7	-1.5	---	1.9	2.4	-1.2*
Discrim.	2	L	---	---	-2.5	-1.7	-0.2	2.5	0.6
Discrim.	1	R	---	---	-2.8	-0.2	0.0	0.8	-0.6
Discrim.	2	R	-2.6	---	-3.6	1.1	2.6	2.9	-0.2
Reading	1	L	-1.5	---	-0.8	0.6	2.2	2.4	-3.1
Reading	2	L	-1.0	---	-1.2	1.0	1.8	1.0	-0.6
Reading	1	R	-1.3	-0.6	-2.8	-1.1	1.4	2.5	-2.1
Reading	2	R	---	---	-2.2	---	1.4	0.4	-1.2

*Estimated data

TABLE 26

INDIVIDUAL AMPLITUDE DATA FOR SUBJECT NUMBER THREE

Treatment	Trial	Ear	Amplitude of Component in Microvolts						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	---	-0.2	-3.3	-1.2	0.6	1.0	-0.9
Quiet	2	L	---	0.8	-2.0	1.2	2.7	1.6	---
Quiet	1	R	---	2.3	-2.8	-0.4*	2.2	4.6	1.8
Quiet	2	R	---	0.6	-2.0	1.1	2.0	2.7	0.0
Counting	1	L	-0.4	-0.3	-5.9	1.8*	3.2	3.9	0.1
Counting	2	L	---	1.2	-2.0	0.0	3.2	3.1	0.7
Counting	1	R	---	1.0*	-5.3	-1.0	0.6	4.8	0.6
Counting	2	R	0.7	1.0	-2.0	0.9	2.8	3.3	1.4
Discrim.	1	L	-0.5	0.1	-3.8	-0.8*	---	6.3	---
Discrim.	2	L	1.0	1.5	-3.2	-0.4	3.2	5.2	---
Discrim.	1	R	---	0.0	-5.6	0.1*	7.0	7.0	-5.0
Discrim.	2	R	---	0.7	-3.8	1.1*	6.8	4.9*	-4.8
Reading	1	L	-0.8	-0.1	-4.7	-0.8	-0.5	1.7	-2.3
Reading	2	L	-0.2	-0.2	-2.5	0.0	---	3.7*	---
Reading	1	R	---	-1.2	-7.0	-3.3	3.2	3.6	1.2
Reading	2	R	1.7	2.4	-3.2	0.8	2.5	3.9	-0.2

*Estimated data

TABLE 27

INDIVIDUAL AMPLITUDE DATA FOR SUBJECT NUMBER FOUR

Treatment	Trial	Ear	Amplitude of Component in Microvolts						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	-1.8	0.0	-3.9	1.1	3.5	3.4	0.4
Quiet	2	L	-0.9	-0.5	-4.2	3.1	3.0	2.7	-0.5
Quiet	1	R	-2.5	1.6*	-2.3	2.4	2.4	2.2	-0.3
Quiet	2	R	-1.3	-0.8	-1.6	1.6	3.8	1.9	-0.6
Counting	1	L	-1.5	-0.4	-3.8	1.3	4.7	3.9	1.0
Counting	2	L	-3.1	-1.6	-3.0	3.5	4.4	2.6	2.1
Counting	1	R	-1.0	0.4	-3.2	2.6*	4.3	3.4	-0.5
Counting	2	R	-1.2	-0.5	-3.1	3.2	5.0	---	0.3
Discrim.	1	L	-2.1	-1.5	-6.0	-0.7*	3.8	7.0	-2.3
Discrim.	2	L	-1.6	-1.4	-4.1	2.2	4.6	3.7	-2.0
Discrim.	1	R	-1.3	-0.6	-3.2	2.2	5.4	---	0.8
Discrim.	2	R	-2.2	-1.5	-3.1	2.2	4.1	4.0	-0.4
Reading	1	L	-2.0	-0.3	-3.8	2.6	3.9	2.3	-2.6
Reading	2	L	-0.4	0.2	-4.3	1.8	3.3	5.1	-1.0
Reading	1	R	-2.4	-0.2	-4.0	2.5	2.4	2.1	-1.9
Reading	2	R	-1.2	-0.2	-2.4	2.4	4.3	2.0	-3.0

*Estimated data

TABLE 28

INDIVIDUAL AMPLITUDE DATA FOR SUBJECT NUMBER FIVE

Treatment	Trial	Ear	Amplitude of Component in Microvolts						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	-2.2	-1.5	-2.7	0.2	2.6	---	---
Quiet	2	L	-2.3	-1.0	-1.6	3.8	6.8	2.7	---
Quiet	1	R	-2.9	---	-2.2	1.9	2.5	---	---
Quiet	2	R	-1.9	-1.2	-4.7	---	5.0	3.5	---
Counting	1	L	-4.3	-0.8	-0.5	5.2	7.2	---	---
Counting	2	L	-3.7	-1.0	-0.8	7.3	11.0	---	---
Counting	1	R	-1.4	0.5	-0.8	4.5	6.3	7.1	0.2
Counting	2	R	-3.6	-0.6	-2.2	---	7.4	---	---
Discrim.	1	L	-3.1	-0.1	-5.0	---	10.1	10.8	---
Discrim.	2	L	-6.0	-2.6	-8.0	---	12.5	12.4	---
Discrim.	1	R	-3.2	0.0	-10.2	---	9.6	9.8	6.0
Discrim.	2	R	-4.0	1.1	-8.8	---	15.1	14.0	---
Reading	1	L	-2.3	---	-1.5	---	1.2	---	---
Reading	2	L	-1.7	---	-1.6	---	2.2	---	-2.5
Reading	1	R	---	---	-3.4	0.4	1.7	0.6	---
Reading	2	R	-3.6	-2.4	-2.8	---	4.5	2.8	-2.4

TABLE 29

INDIVIDUAL AMPLITUDE DATA FOR SUBJECT NUMBER SIX

Treatment	Trial	Ear	Amplitude of Component in Microvolts						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	-0.8	0.5	-2.0	1.0	1.4	2.5	---
Quiet	2	L	-1.6	-0.7	-3.2	-0.8	2.7	---	---
Quiet	1	R	-1.0	-0.6	-4.0	-1.3	1.7	1.9	-0.2
Quiet	2	R	-1.4	-0.6	-4.4	-2.0	1.1	---	---
Counting	1	L	-2.4	-1.0	-4.8	-1.7	0.5	2.8	---
Counting	2	L	-1.9	-0.5	-3.7	-0.9	1.6	2.6	---
Counting	1	R	-2.9	-1.2	-5.2	-2.5	0.3	2.3	-1.6
Counting	2	R	-1.6	-0.2	-4.3	-1.2	1.7	2.8	0.2
Discrim.	1	L	-2.1	-0.9	-4.9	-1.0	0.1	2.2	-0.4
Discrim.	2	L	-0.6	0.9	-2.4	0.8	1.3	2.0	0.1
Discrim.	1	R	-0.9	-0.6	-4.0	1.2	---	1.8	---
Discrim.	2	R	0.0	0.7	-3.4	0.5	0.8	3.0	-0.3
Reading	1	L	-2.5	-0.4	-1.9	0.5	---	1.1	---
Reading	2	L	-1.9	-0.7	-0.9	0.8	---	0.8	---
Reading	1	R	-0.4	0.9	-3.4	0.4	0.8	1.4	-0.1
Reading	2	R	-1.8	-1.1	-3.0	0.1	0.9	---	-0.8

TABLE 30

INDIVIDUAL AMPLITUDE DATA FOR SUBJECT NUMBER SEVEN

Treatment	Trial	Ear	Amplitude of Component in Microvolts						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	-1.0	-0.4	-3.6	0.8	2.6	4.6	-0.4
Quiet	2	L	-4.7*	0.4	-1.6	---	1.5	1.6	-0.8
Quiet	1	R	-2.3	-1.8	-4.2	---	2.1	4.1	0.5
Quiet	2	R	-1.1*	0.4	-1.6	---	1.8	---	1.1
Counting	1	L	-0.1	0.6	-1.6	-0.4	1.3	1.8	-0.9
Counting	2	L	-3.9*	---	-1.3	0.1	1.2	1.8	0.2
Counting	1	R	-2.5*	---	4.1	---	1.9	3.3	-0.5
Counting	2	R	-1.4	0.1	-1.9	-1.2	-0.1	1.9	0.4
Discrim.	1	L	-0.6	0.0	-3.7	-0.8	0.8	2.6	0.5
Discrim.	1	L	-2.0*	-0.1	-2.7	-1.8	0.7	1.4	-0.3
Discrim.	1	R	-2.1	---	-5.9	---	0.6	2.7	-0.2
Discrim.	2	R	-0.9*	---	-2.2	-0.2	1.3	1.6	-0.8
Reading	1	L	-0.7	-0.3	-2.8	-0.4	1.2	1.2	-0.4
Reading	2	L	0.3	---	-1.3	0.1	1.3	1.5	-0.2
Reading	1	R	-1.2	---	-3.3	-0.8	0.8	---	-1.2*
Reading	2	R	0.1	0.7	-1.5	0.6	1.7	---	-2.6*

*Estimated data

TABLE 31

INDIVIDUAL AMPLITUDE DATA FOR SUBJECT NUMBER EIGHT

Treatment	Trial	Ear	Amplitude of Component in Microvolts						
			N3a	P3b	N4a	P4b	P5a	P5b	N6a
Quiet	1	L	-0.5	-0.9	-3.5	2.6	4.3	3.3	2.4
Quiet	2	L	-0.7	-0.2	-3.4	4.3	5.4	3.7	---
Quiet	1	R	0.8	1.6	-4.1	-0.2	1.5	2.8	---
Quiet	2	R	---	1.2	-1.9	---	1.5	3.4	---
Counting	1	L	---	---	-2.1	0.4	---	7.7	1.8
Counting	2	L	-0.2	---	-3.9	2.2	3.5	5.0	0.4
Counting	1	R	---	---	-4.0	---	---	4.3	0.2
Counting	2	R	-0.6	---	-2.3	2.4	3.8	5.0	2.0
Discrim.	1	L	---	-0.5	-8.3	---	---	4.5	1.9
Discrim.	2	L	-1.0	0.9	-6.8	---	3.8	6.9	-1.2
Discrim.	1	R	-1.0	---	-5.1	0.1	3.2	4.9	0.6
Discrim.	2	R	-0.5	0.0	-3.7	0.8	5.3	7.3	1.0
Reading	1	L	-0.6	-0.1	-4.2	-0.5	---	3.2	-0.1
Reading	2	L	0.6	-0.2	-5.6	1.9	---	3.4	-1.7
Reading	1	R	0.1	0.5	-3.3	0.0	0.6	1.5	-0.6
Reading	2	R	-0.6	0.5	-4.2	1.0	2.0	3.6	-1.7

APPENDIX C

ANALYSIS OF VARIANCE TABLES

TABLE 32

SUMMARY OF ANALYSIS OF VARIANCE FOR VARIANCE OF THE N3a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.00620	0.00620	<1
Ear x Subject	3	0.29315	0.09771	
Trial	1	0.06063	0.06063	<1
Trial x Subject	3	1.18876	0.39625	
Ear x Trial	1	0.10807	0.10807	<1
Ear x Trial x Subject	3	0.49377	0.16459	
Treatment	3	1.72030	0.57343	4.27*
Ear x Treatment	3	0.45782	0.15260	1.14
Trial x Treatment	3	0.20314	0.06771	<1
Run	3	0.89655	0.29885	2.23
Ear x Run	3	0.19942	0.06647	<1
Trial x Run	3	0.03494	0.01164	<1
Error	18	2.41530	0.13418	

*Significant at the five per cent level of confidence.

TABLE 33

SUMMARY OF ANALYSIS OF VARIANCE FOR VARIANCE OF THE P3b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.23401	0.23401	3.12
Ear x Subject	3	0.22490	0.07496	
Trial	1	0.00097	0.00097	<1
Trial x Subject	3	1.22284	0.40761	
Ear x Trial	1	0.28756	0.28756	1.82
Ear x Trial x Subject	3	0.47392	0.15797	
Treatment	3	1.13159	0.37719	3.88*
Ear x Treatment	3	0.40209	0.13403	1.38
Trial x Treatment	3	0.13327	0.04442	<1
Run	3	1.26105	0.42050	4.33*
Ear x Run	3	0.06302	0.02100	<1
Trial x Run	3	0.07424	0.02474	<1
Error	22	2.13644	0.09711	

*Significant at the five per cent level of confidence.

TABLE 34

SUMMARY OF ANALYSIS OF VARIANCE FOR VARIANCE OF THE N4a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.03850	0.03850	<1
Ear x Subject	7	1.87274	0.26753	
Trial	1	0.73205	0.73205	<1
Trial x Subject	7	16.10067	2.30009	
Ear x Trial	1	0.54601	0.54601	<1
Ear x Trial x Subject	7	3.89241	0.55605	
Treatment	3	6.30361	2.10120	11.22*
Ear x Treatment	3	0.46904	0.15634	<1
Trial x Treatment	3	0.27323	0.09107	<1
Run	3	2.09001	0.69667	3.72*
Ear x Run	3	0.40447	0.13482	<1
Trial x Run	3	1.11983	0.37327	1.99
Error	72	13.48033	0.18723	

*Significant at the five per cent level of confidence.

TABLE 35

SUMMARY OF ANALYSIS OF VARIANCE FOR VARIANCE OF THE P4b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.14726	0.14726	<1
Ear x Subject	3	0.77311	0.25770	
Trial	1	0.00007	0.00007	<1
Trial x Subject	3	0.77052	0.25684	
Ear x Trial	1	0.08628	0.08628	<1
Ear x Trial x Subject	3	0.51061	0.17020	
Treatment	3	0.67027	0.22342	2.69
Ear x Treatment	3	0.78180	0.26060	3.13
Trial x Treatment	3	0.22734	0.07578	<1
Run	3	1.71824	0.57274	6.89*
Ear x Run	3	0.17814	0.05938	<1
Trial x Run	3	0.16657	0.05552	<1
Error	16	1.33065	0.08316	

*Significant at the five per cent level of confidence.

TABLE 36

SUMMARY OF ANALYSIS OF VARIANCE FOR VARIANCE OF THE P5a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.05347	0.05347	<1
Ear x Subject	3	0.16681	0.05560	
Trial	1	0.45393	0.45393	1.05
Trial x Subject	3	1.30185	0.43395	
Ear x Trial	1	0.00701	0.00701	<1
Ear x Trial x Subject	3	0.37872	0.12624	
Treatment	3	1.55532	0.51844	2.87
Ear x Treatment	3	0.03057	0.01019	<1
Trial x Treatment	3	0.61721	0.20573	1.14
Run	3	0.49434	0.16478	<1
Ear x Run	3	0.49586	0.16528	<1
Trial x Run	3	0.56712	0.18904	1.05
Error	24	4.34159	0.18089	

No significant levels obtained at the five per cent level.

TABLE 37

SUMMARY OF ANALYSIS OF VARIANCE FOR VARIANCE OF THE P5b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.24132	0.24132	12.20*
Ear x Subject	3	0.05936	0.01978	
Trial	1	0.96285	0.96285	7.31
Trial x Subject	3	0.39516	0.13172	
Ear x Trial	1	0.04462	0.04462	<1
Ear x Trial x Subject	3	0.57821	0.19273	
Treatment	3	4.60481	1.53493	6.61*
Ear x Treatment	3	0.65859	0.21953	<1
Trial x Treatment	3	0.03736	0.01245	<1
Run	3	2.00572	0.66857	2.88
Ear x Run	3	0.83670	0.27890	1.20
Trial x Run	3	0.47637	0.15879	<1
Error	19	4.41367	0.23229	-

*Significant at the five per cent level of confidence.

TABLE 38

SUMMARY OF ANALYSIS OF VARIANCE FOR VARIANCE OF THE N6a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.11390	0.11390	<1
Ear x Subject	3	0.65396	0.21798	
Trial	1	0.64802	0.64802	<1
Trial x Subject	3	1.96732	0.65577	
Ear x Trial	1	0.01500	0.01500	<1
Ear x Trial x Subject	3	0.72286	0.24095	
Treatment	3	2.45722	0.81907	4.16*
Ear x Treatment	3	0.41191	0.13730	<1
Trial x Treatment	3	0.11667	0.03889	<1
Run	3	0.24667	0.08222	<1
Ear x Run	3	0.35916	0.11972	<1
Trial x Run	3	1.05842	0.35280	1.79
Error	21	4.13717	0.19700	

*Significant at the five per cent level of confidence.

TABLE 39

SUMMARY OF ANALYSIS OF VARIANCE FOR LATENCY OF THE N3a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	4.00000	4.00000	<1
Ear x Subject	3	24.12500	8.04166	
Trial	1	1.00000	1.00000	<1
Trial x Subject	3	163.12500	54.37500	
Ear x Trial	1	1.00000	1.00000	1.14
Ear x Trial x Subject	3	2.62500	0.87500	
Treatment	3	35.12500	11.70833	3.01
Ear x Treatment	3	17.87500	5.95833	1.53
Trial x Treatment	3	10.12500	3.37500	<1
Run	3	14.12500	4.70833	1.21
Ear x Run	3	26.37500	8.79166	2.26
Trial x Run	3	16.87500	5.62500	1.45
Error	18	70.00000	3.88888	

No significant levels obtained at the five per cent level.

TABLE 40

SUMMARY OF ANALYSIS OF VARIANCE FOR LATENCY OF THE P3b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	70.14062	70.14062	37.09*
Ear x Subject	3	6.17187	2.05729	
Trial	1	28.89062	28.89062	3.02
Trial x Subject	3	28.67187	9.55729	
Ear x Trial	1	0.76562	0.76562	<1
Ear x Trial x Subject	3	32.04687	10.68229	
Treatment	3	142.67187	47.55729	5.02*
Ear x Treatment	3	127.79687	42.59895	4.50*
Trial x Treatment	3	32.79687	10.93229	1.26
Run	3	60.92187	20.30729	2.14
Ear x Run	3	39.04687	13.01562	1.37
Trial x Run	3	15.04687	5.01562	<1
Error	22	208.37507	9.47159	

*Significant at the five per cent level of confidence.

TABLE 41

SUMMARY OF ANALYSIS OF VARIANCE FOR LATENCY OF THE N4a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	1.53125	1.53125	<1
Ear x Subject	7	353.34375	50.47767	
Trial	1	0.12500	0.12500	<1
Trial x Subject	7	220.00000	31.42857	
Ear x Trial	1	42.78125	42.78125	1.18
Ear x Trial x Subject	7	253.84375	36.26339	
Treatment	3	1781.93750	593.97916	7.19*
Ear x Treatment	3	478.15625	159.38541	1.93
Trial x Treatment	3	34.56250	11.52083	<1
Run	3	60.75000	20.25000	<1
Ear x Run	3	347.84375	115.94791	1.40
Trial x Run	3	203.12500	67.70833	<1
Error	72	5950.12500	82.64063	

*Significant at the five per cent level of confidence.

TABLE 42

SUMMARY OF ANALYSIS OF VARIANCE FOR LATENCY OF THE P4b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	13.14062	13.14062	<1
Ear x Subject	3	46.67187	15.55729	
Trial	1	21.39062	21.39062	<1
Trial x Subject	3	221.42187	73.80729	
Ear x Trial	1	74.39062	74.39062	1.20
Ear x Trial x Subject	3	62.17187	20.72395	
Treatment	3	1711.67187	570.55729	9.50*
Ear x Treatment	3	174.29687	58.09895	<1
Trial x Treatment	3	70.54687	23.51526	<1
Run	3	185.04687	61.68229	<1
Ear x Run	3	168.92187	56.30729	<1
Trial x Run	3	11.42187	3.80729	<1
Error	16	1037.62508	64.85156	

*Significant at the five per cent level of confidence.

TABLE 43

SUMMARY OF ANALYSIS OF VARIANCE FOR LATENCY OF THE P5a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	97.51562	97.51562	1.26
Ear x Subject	3	231.92187	77.30729	
Trial	1	5.64062	5.64062	<1
Trial x Subject	3	186.29687	62.09895	
Ear x Trial	1	107.64062	107.64062	4.15
Ear x Trial x Subject	3	77.79687	25.93229	
Treatment	3	852.54687	284.18229	2.49
Ear x Treatment	3	779.04687	259.68229	2.27
Trial x Treatment	3	65.17187	21.72395	<1
Run	3	17.42187	5.80729	<1
Ear x Run	3	898.67187	299.55729	2.62
Trial x Run	3	177.54687	59.18229	<1
Error	24	2740.37508	114.18229	

No significant levels obtained at the five per cent level.

TABLE 44

SUMMARY OF ANALYSIS OF VARIANCE FOR LATENCY OF THE P5b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	517.56250	517.56250	<1
Ear x Subject	3	2027.81250	675.93750	
Trial	1	333.06250	333.06250	4.73
Trial x Subject	3	211.31250	70.43750	
Ear x Trial	1	64.00000	64.00000	<1
Ear x Trial x Subject	3	400.62500	133.54166	
Treatment	3	1719.87500	573.29166	3.01
Ear x Treatment	3	1362.56250	454.18750	2.38
Trial x Treatment	3	85.56250	28.52083	<1
Run	3	283.00000	94.33333	<1
Ear x Run	3	197.18750	65.72916	<1
Trial x Run	3	698.18750	232.72916	1.22
Error	19	3622.75000	190.67105	

No significant levels obtained at the five per cent level.

TABLE 45

SUMMARY OF ANALYSIS OF VARIANCE FOR LATENCY OF THE N6a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	1570.14062	1570.14062	1.75
Ear x Subject	3	2685.92187	895.30729	
Trial	1	92.64062	92.64062	<1
Trial x Subject	3	821.42187	273.80729	
Ear x Trial	1	9.76562	9.76562	<1
Ear x Trial x Subject	3	296.04687	98.68229	
Treatment	3	1410.92187	470.30729	1.29
Ear x Treatment	3	835.92187	278.64062	<1
Trial x Treatment	3	307.17187	102.39062	<1
Run	3	1554.17187	518.05729	1.42
Ear x Run	3	1038.92187	346.30729	<1
Trial x Run	3	965.67187	321.89062	<1
Error	21	7655.62509	364.55357	

No significant levels obtained at the five per cent level.

TABLE 46

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO THE N4a COMPONENT FOR THE N3a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	2.68140	2.68140	1.09
Ear x Subject	3	7.35796	2.45265	
Trial	1	0.66051	0.66051	<1
Trial x Subject	3	20.02171	6.67390	
Ear x Trial	1	0.34515	0.34515	<1
Ear x Trial x Subject	3	4.53671	1.51223	
Treatment	3	2.27796	0.75932	<1
Ear x Treatment	3	18.21796	6.07265	1.55
Trial x Treatment	3	11.50921	3.83640	<1
Run	3	10.61046	3.53682	<1
Ear x Run	3	11.43296	3.81098	<1
Trial x Run	3	24.05421	8.01807	2.05
Error	18	70.44851	3.91380	

No significant levels obtained at the five per cent level.

TABLE 47

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO THE N4a COMPONENT FOR THE P3b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.47265	0.47265	<1
Ear x Subject	3	2.00046	0.66682	
Trial	1	4.35765	4.35765	2.31
Trial x Subject	3	5.66796	1.88932	
Ear x Trial	1	0.00140	0.00140	<1
Ear x Trial x Subject	3	3.21921	1.07307	
Treatment	3	3.43421	1.14473	<1
Ear x Treatment	3	8.75671	2.91890	<1
Trial x Treatment	3	22.10671	7.36890	2.20
Run	3	30.39421	10.12140	3.02
Ear x Run	3	7.03921	2.34640	<1
Trial x Run	3	42.36921	14.12307	4.21*
Error	22	73.74137	3.35188	

*Significant at the five per cent level of confidence.

TABLE 48

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO THE N4a COMPONENT FOR THE P4b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.19140	0.19140	<1
Ear x Subject	3	3.62671	1.20890	
Trial	1	1.72265	1.72265	<1
Trial x Subject	3	27.25546	9.08515	
Ear x Trial	1	0.02640	0.02640	<1
Ear x Trial x Subject	3	8.27421	2.75807	
Treatment	3	7.37671	2.45890	<1
Ear x Treatment	3	11.49171	3.83057	<1
Trial x Treatment	3	18.30796	6.10265	1.52
Run	3	6.76046	2.25348	<1
Ear x Run	3	18.28296	6.09432	1.51
Trial x Run	3	21.99171	7.33057	1.82
Error	16	64.38637	4.02414	

No significant levels obtained at the five per cent level.

TABLE 49

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO THE N4a COMPONENT FOR THE P5a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.76562	0.76562	<1
Ear x Subject	3	10.12187	3.37395	
Trial	1	10.08062	10.08062	1.92
Trial x Subject	3	15.76687	5.25562	
Ear x Trial	1	0.14062	0.14062	<1
Ear x Trial x Subject	3	9.45687	3.15229	
Treatment	3	10.17312	3.39104	1.63
Ear x Treatment	3	8.57062	2.85687	1.38
Trial x Treatment	3	10.91562	3.63854	1.75
Run	3	9.78062	3.26020	1.57
Ear x Run	3	11.28062	3.76020	1.81
Trial x Run	3	5.45812	1.81937	<1
Error	24	49.84507	2.07687	

No significant levels obtained at the five per cent level.

TABLE 50

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO THE N4a COMPONENT FOR THE P5b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.66015	0.66015	<1
Ear x Subject	3	35.93421	11.97807	
Trial	1	9.37890	9.37890	<1
Trial x Subject	3	30.92796	10.30932	
Ear x Trial	1	0.08265	0.08265	<1
Ear x Trial x Subject	3	5.95921	1.98640	
Treatment	3	100.44546	33.48182	8.50*
Ear x Treatment	3	9.25296	3.08432	<1
Trial x Treatment	3	18.35421	6.11807	1.55
Run	3	38.19796	12.73265	3.23*
Ear x Run	3	1.73046	0.57682	<1
Trial x Run	3	11.90421	3.96807	1.01
Error	19	74.88136	3.94112	

*Significant at the five per cent level of confidence.

TABLE 51

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO THE N4a COMPONENT FOR THE N6a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.42249	0.42249	<1
Ear x Subject	3	13.98374	4.66124	
Trial	1	0.05062	0.05062	<1
Trial x Subject	3	31.75812	10.58640	
Ear x Trial	1	4.95062	4.95062	5.45
Ear x Trial x Subject	3	2.72562	0.90854	
Treatment	3	19.52125	6.50708	2.20
Ear x Treatment	3	4.98875	1.66291	<1
Trial x Treatment	3	1.56312	0.52104	<1
Run	3	17.39249	5.79749	1.96
Ear x Run	3	0.72749	0.24249	<1
Trial x Run	3	4.93687	1.64562	<1
Error	21	61.99257	2.95202	

No significant levels obtained at the five per cent level.

TABLE 52

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO ZERO BASELINE FOR THE N3a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.06890	0.06890	<1
Ear x Subject	3	1.62546	0.54182	
Trial	1	0.47265	0.47265	<1
Trial x Subject	3	1.74171	0.58057	
Ear x Trial	1	3.46890	3.46890	1.37
Ear x Trial x Subject	3	7.59046	2.53015	
Treatment	3	4.22796	1.40932	1.56
Ear x Treatment	3	0.67171	0.22390	<1
Trial x Treatment	3	1.58046	0.52682	<1
Run	3	3.56671	1.18890	1.32
Ear x Run	3	1.32046	0.44015	<1
Trial x Run	3	3.98671	1.32890	1.47
Error	18	16.22387	0.90132	

No significant levels obtained at the five per cent level.

TABLE 53

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO ZERO BASELINE FOR THE P3b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.97515	0.97515	3.32
Ear x Subject	3	0.88171	0.29390	
Trial	1	0.05640	0.05640	<1
Trial x Subject	3	4.34046	1.44682	
Ear x Trial	1	0.01265	0.01265	<1
Ear x Trial x Subject	3	1.64421	0.54807	
Treatment	3	2.07546	0.69182	1.32
Ear x Treatment	3	1.22546	0.40848	<1
Trial x Treatment	3	2.34171	0.78057	1.49
Run	3	4.41171	1.47057	2.81
Ear x Run	3	0.57171	0.19057	<1
Trial x Run	3	3.68296	1.22765	2.35
Error	22	11.50137	0.52278	

No significant levels obtained at the five per cent level.

TABLE 54

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO ZERO BASELINE FOR THE N4a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	2.05031	2.05031	<1
Ear x Subject	7	33.24718	4.74959	
Trial	1	10.12499	10.12499	2.46
Trial x Subject	7	28.81249	4.11607	
Ear x Trial	1	2.36531	2.36531	3.67
Ear x Trial x Subject	7	4.51468	0.64495	
Treatment	3	60.66312	20.22104	1.00
Ear x Treatment	3	1.10781	0.36927	<1
Trial x Treatment	3	1.63937	0.54645	<1
Run	3	5.45437	1.81812	<1
Ear x Run	3	3.98156	1.32718	<1
Trial x Run	3	1.14187	0.38062	<1
Error	72	144.97132	20.13491	

No significant levels obtained at the five per cent level.

TABLE 55

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO ZERO BASELINE FOR THE P4b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.10562	0.10562	<1
Ear x Subject	3	2.13812	0.71270	
Trial	1	1.89062	1.89062	<1
Trial x Subject	3	9.27062	3.09020	
Ear x Trial	1	0.01562	0.01562	<1
Ear x Trial x Subject	3	6.01312	2.00437	
Treatment	3	0.41312	0.13770	<1
Ear x Treatment	3	5.31312	1.77104	<1
Trial x Treatment	3	0.62062	0.20687	<1
Run	3	7.83062	2.61020	1.45
Ear x Run	3	0.65562	0.21854	<1
Trial x Run	3	0.95562	0.31854	<1
Error	16	28.76507	1.79781	

No significant levels obtained at the five per cent level.

TABLE 56

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO ZERO BASELINE FOR THE P5a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.04515	0.04515	<1
Ear x Subject	3	0.14671	0.04890	
Trial	1	3.01890	3.01890	2.33
Trial x Subject	3	3.89046	1.29682	
Ear x Trial	1	0.78765	0.78765	3.49
Ear x Trial x Subject	3	0.67671	0.22557	
Treatment	3	9.24296	3.08098	3.30*
Ear x Treatment	3	2.64171	0.88057	<1
Trial x Treatment	3	2.53796	0.84598	<1
Run	3	12.66546	4.22182	4.52*
Ear x Run	3	1.82671	0.60890	<1
Trial x Run	3	0.52796	0.17598	<1
Error	24	22.43387	0.93474	

*Significant at the five per cent level of confidence.

TABLE 57

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO ZERO BASELINE FOR THE P5b COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.68062	0.68062	<1
Ear x Subject	3	5.80687	1.92562	
Trial	1	0.90250	0.90250	<1
Trial x Subject	3	5.34500	1.78166	
Ear x Trial	1	0.03999	0.03999	<1
Ear x Trial x Subject	3	4.07249	1.35949	
Treatment	3	37.39187	12.46395	8.54*
Ear x Treatment	3	4.52187	1.50729	<1
Trial x Treatment	3	3.23249	1.07749	<1
Run	3	7.92187	2.64062	1.81
Ear x Run	3	1.96187	0.65395	<1
Trial x Run	3	12.40250	4.13416	2.83
Error	19	27.72758	1.45934	

*Significant at the five per cent level of confidence.

TABLE 58

SUMMARY OF ANALYSIS OF VARIANCE FOR AMPLITUDE RELATIVE TO ZERO BASELINE FOR THE N6a COMPONENT

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Square	F
Ear	1	0.02249	0.02249	<1
Ear x Subject	3	0.33624	0.11208	
Trial	1	0.45562	0.45562	<1
Trial x Subject	3	8.60562	2.86854	
Ear x Trial	1	0.08999	0.08999	<1
Ear x Trial x Subject	3	2.87375	0.95791	
Treatment	3	13.55062	4.51687	3.01
Ear x Treatment	3	0.78125	0.26041	<1
Trial x Treatment	3	3.60062	1.20020	<1
Run	3	9.54687	3.18229	2.12
Ear x Run	3	0.24000	0.08000	<1
Trial x Run	3	0.38187	0.12729	<1
Error	21	31.55757	1.50274	

No significant levels obtained at the five per cent level.