OPERANT CONDITIONING OF THE LATENCY OF A VISUAL EVOKED POTENTIAL: INDEPENDENCE OF EARLY AND LATE COMPONENTS

by

Verner Stuart Westerberg

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VERNER STUART WESTERBERG, 1977
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Supervisor: Dr. David Walker [redacted]

ABSTRACT

Two hooded rats were operantly conditioned for both decreasing (Phase 1), and increasing (Phase 2) the latency of a late component in a photically evoked cortical potential. Reinforcement was under on-line control of a small digital computer with software designed to recognize a specified potential at a specified latency of occurrence. Both animals exhibited learning specific to the late component in Phase 1 as compared to Baseline. Phase 2 conditioning showed that one animal did acquire the reversal task; the second animal showed only marginal learning.

On the basis of the results in conditioning, three evoked potential components, one early and two later, were analyzed according to latency, amplitude, and occurrence. All components were shown to be independent of one another in terms of latency and amplitude, and to some degree, in occurrence. It was also shown that the latency and amplitude of the conditioned component were independent of one another.

The conclusion reached was that the latency of a component can be shown to be of adaptive significance by itself, and that the process of conditioning changes the significance of the conditioned potential. Thus, each parameter of each potential of the evoked potential has the capability of discrete encoding of behavior which is dependent on the conditioned state.

Committee Members:

Dr. Louis Sutker [redacted]

Dr. Alan Drengson [redacted]

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Dedication

This thesis is dedicated to all laboratory animals, especially twenty hooded rats who helped me through three years of research in this field.

Introduction

The purpose of the study of behavior is to understand how the organism functions, on all levels from molecular processes to complex behavior, and to unify the results of these studies. Electrophysiologists have taken the view that the most viable means for understanding behavior lies in that system which is directly responsible for the transmission, storage, retrieval, and interpretation of information -- the nervous system.

Studies of the nervous system may be concerned with the conduction of impulses in isolated neurons or with gross brain responses involving millions of neurons in freely moving animals. While there is no question that a total understanding of the nervous system, indeed of all complex behavior, cannot be complete without knowledge of the structure and function of the components of the nervous system, it is also true that a study of the integrative action of the nervous system must proceed in parallel. This statement follows from the fact that as systems become more complex, properties may emerge which cannot be predicted from the constituants of the system.

This paper will take the position that the study of the electrical processes of adapting systems may take place quite independently of the knowledge of the constituants of the processes. That is, the significance of an electrical event may be demonstrated without necessarily being able to specify all of the conditions which give rise to the event.

The studies of bioelectric events in the central nervous system(CNS) have followed two lines of research, pure physiological (conduction, excitation, synaptic transmission), and what Sherrington (1906) called special physiology (integration and adaptation). The conclusions which

arose from the early doctrines of Cajal and the electrophysiological research of workers such as Adrian, Gasser and Erlanger led to the belief that the nervous system could be regarded as a binary or pulse-coding device (Bullock, 1959), with the neuron as the basic unit and the nerve impulse as the unit of information transfer. This strict view of the nervous system has undergone revision with discoveries which have indicated that there is more to nervous activity and neural transmission than the axon spike (Bullock, 1967; Grundfest, 1967; Purpura, 1967; Purpura, Girado and Grundfest, 1969; Schmitt, Dev and Smith, 1976).

Most work in electrophysiology has been concerned with projection neurons, those that have long axons, and have used axon spikes as an important part of the transmission of the neural signal. However, in 1899 Ramon y Cajal had stated: "The functional superiority of the human brain is ultimately linked up with the prodigious abundance and unaccustomed wealth of forms of the so-called neurons with short axons." (p. 301). Schmitt et al. (1976) have stated, that based on experimental evidence, that graded electrotonic potentials, rather than the classic axon spike, may be the language of a large part of the central nervous system.

That the axon spike is not the only means of neural transmission is exemplified by the local circuit neuron (LCN). LCNs are those neurons which have short axons or no axons, such as the horizontal and amacrine cells of the retina (Dowling, 1975), and the granule cells in the olfactory system (Rall, 1975). These neurons are capable of integrating and processing information at a local level without the use of the axon spike (Racik, 1975); in fact, Dowling asserts that in the axon type horizontal cell the axon may actually isolate the dendritic area from the axon terminals. Dowling also reports that action potentials are sometimes superimposed

on the slow potentials of the amacrine cell, but that the amacrine cell does not code information by spike trains.

It is likely that psychologists and physiologists would both be in agreement with the statement that a single neuron cannot carry enough information to account for the behavioral abilities of an animal. Rapoport (1965) asserts that the mapping of synaptic events to gross behavior would not produce a linear relationship. Thus, he concludes that trying to describe all gross behaviors in terms of sequences of synaptic events would be an impossible task. In spite of this agreement, the physiological and psychological approach to the studies of the CNS are basically from two different points of view. Physiologists assert that complex behavior may be understood in terms of the isolated behavior of small groups of neurons (Bullock, 1959; Pribram, 1972). Psychologists, on the other hand, are not willing to accept that behavior can be reduced to merely the sum of its basic parts. Some years ago Boring (1932) described this difference:

> The physiologist holds to the faith that the brain, being made up of neurons, is capable only of that excitation which is the sum of the excitations of many neurons, and that these central neurons obey the same laws and are excited under the same limitations as apply to the peripheral neurons which have been experimentally studied. To this article of faith the psychologist sometimes opposes another belief, that the organization of the cerebral excitation corresponds to the organization of phenomenal experience. (p. 32)

Uttal (1967) further emphasizes this idea:

> A reduction analysis of behavior to the level of the individual actions of neurons and synapses, on a strictly deterministic basis, may be just as difficult as describing the behavior of a box of gas on the dynamics of the individual molecules constituting the gas. (p. 631)

Physiological studies have traditionally used powerful stimulus-response designs, with the intent to formulate laws governing coding of stimuli and the interaction of neural structure and function. This design is powerful in terms of relating a discrete stimulus to a neural behavior,

however, the adaptation of the organism is usually not affected by the stimulus nor dependent upon the neural events studied (Walker and Long, 1976). That is, the experimental environment is not challenging to the organism under study; the behavior is evoked rather than emitted. For psychology, the area of interest is in adaptive behaviors, the behaviors of whole organisms in a situation where behavior is allowed to be emitted. Pursuant to this, research in psychology has been more interested in variables affected by the history of the animal in adaptive situations -- such as perception, learning, memory, attention, and motivation.

Neural Substrate of Slow Waves

Bullock (1967) lists several forms of bioelectric events recognized as important as signals in the CNS. These include: passive electrotonic potentials, active electrogenic potentials, interneuronal transmitters, and electrical signs in masses of neurons. While physiological psychology has dealt with single units and complex behavior, the bulk of the research has been with the last group listed, the electrical signs in masses of neurons. It is these slow wave (SW) phenomena, which include the electroencephalogram(EEG), and the stimulus-bound evoked potential (EP) which have been of primary interest in psychology. Since the body of this is concerned with slow waves, an attempt will be made to review their connection with the neural substrate.

Early research indicated that SWs were envelopes of spike discharges (Adrian and Matthews, 1934). Extensive review of the subject (Landau, 1967; Purpura, 1967) has since provided ample evidence that the slow wave phenomena are more the result of dendritic activity than soma-axon hillock spikes or the .5 msec axon spike.

Bartley and Bishop (1933), recording from rabbit cortex, calculated that there could not be enough fibers in a given mass of cortical tissue for an axonal model to account for slow waves. They concluded that the cortex operates differently from the lower centers and peripheral nerves, and that the concepts founded on studies of lower centers could not be applied to cortex. Sherrington (1906) made much this same point when he stated that the spinal reflex showed a complexity of behavior which could not be accounted for on the basis of knowledge of the action of the nerve trunks alone.

Li and Jasper (1953), simultaneously recording single unit and EEG activity, found that they could still record EEG when no single units were firing. Some 13 years prior to this, Renshaw, Forbes and Morison (1940) recorded single unit potentials and SWs and concluded that there was no interdependency of spikes and SWs.

However, in a series of two experiments, Creutzfeldt, Watnabe and Lux (1966 a, b) have asserted that EEG phenomena (including evoked responses) and cellular responses could be related. They concluded that cortical evoked potentials were composed of several intracortical events which included transients near the cell soma, slow dendritic potentials, and fiber activity. This is in accord with findings by Eccles (1951), Purpura (1967), and Purpura et al. (1959).

Purpura (1967), in a study utilizing direct cortical stimulation, stated that the changes produced by weak polarizing currents occur as a consequence of alterations in post synaptic potentials (PSPs) initiated in dendrites which are remote from the generation site of excitatory PSPs (EPSPs) associated with the soma. That is, the PSPs generated in dendrites make the major contribution to epicortical EPs, and that the dendrites, at

least in cortical pyramidal neurons, do not give rise to conducted spikes. Purpura, Girado and Grunfest (1960) stated that in the cortex the effects of PSPs would be expected to predominate over those of spikes or of graded responses. This indicates that the recorded activity would then be primarily the result of PSPs initiated in dendrites.

The research which separated out axon spikes as a source of SWs also came to indicate a poor relation between spike and wave (Fox, 1970). Although the findings are that SWs are not dependent on spikes, there does appear to be a definite relation between SWs and some single unit phenomena. Creutzfeldt et al. (1966) reported that after stimulation of the ventrolateral nucleus of the thalamus, the afferent volley, primary EPSP, and the early stages of the IPSP coincided with the primary positivity (15-50 msec) of the surface evoked potential, while the remainder of the IPSP coincided with a surface negativity. Freeman(1967) concluded the same; surface negativity is correlated with excitation of pyramidal cells, and positivity with inhibition of these cells.

Gerstein and Kiang (1964) have reported that there is no congruence between SWs and spike potentials in auditory cortex. However, other workers have reported good correlations between the probability of spike discharge and the EEG (Fox and Norman, 1968; Krekule and Walker, 1971) and the EP (Fox and O'Brien, 1965; Verzeano, Dill, Vallecalle, Groves and Thomas, 1968).

Recording both single cell activity and EPs with the same electrode in response to somatic and photic stimulation reveals that the frequency distribution of the firing of any particular cell corresponds closely to the average evoked waveform (Fox and O'Brien, 1965). The conclusion is, therefore, that whatever potentials contribute to the evoked potential,

they are related on a probabilistic basis to the firing of single cells. A distinguishing feature of this study when contrasted with the negative results of Gerstein and Kiang is the large number of single unit responses obtained. Fox and O'Brien correlated the spike firings to 3,150 presentations of the stimulus with the average of 150 gross evoked sweeps, and the spike firings to 4,918 stimulus presentations with 200 averaged evoked sweeps. Gerstein and Kiang, on the other hand, used small samples on the order of 250-300 stimulus presentations.

Fox (1970) reports that the correlation between spontaneous firing of single cells with EEG voltage is not a constant, but may vary as much as 28% from sample to sample. He further reports that spike-wave congruence in half the cells studied increased when photically driven and the other half decreased. Mountcastle (1969), commenting on this, states that driving the cell population would tend to stabilize an otherwise shifting relation between spike and SW.

It thus seems that while axon spikes in particular cells are not necessary for SW generation, there is a definite connection between SWs and an underlying cell population, and that the largest factor contributing to the EP is dendritic activity. It has also been shown that there is a relation between SWs and spike activity if a large enough sample is used so as to offset the enormous variability in single unit behavior.

Variability: The Problem of Neural Correlates

In the study of adaptive behavior one approach is to attempt to correlate a bioelectric change with a change in behavior. To this end, researchers have studied single cells (Jasper, Ricci, and Doane, 1960; Bures and Buresova, 1965; O'Brien and Fox, 1969a,b), groups of single cells with microelectrode arrays (John, 1972; John and Morgades, 1969), and

SWs (Shucard and Horn, 1972; Gardiner, 1969; Smith, Donchin, Cohen and Starr, 1969) in an effort to relate them to complex behavior.

The correlative approach assumes the existance of stable connections between stimulus input and behavioral output, or at least stable neural circuits which are capable of carrying only certain types of information. While there has been some success in correlating certain motor behaviors with neural signals (Rosenfeld and Fox, 1972),learning with single cell activity (Jasper et al., 1960; O'Brien and Fox, 1969a,b), and sensory input to single cell activity (Mountcastle, 1957, 1967, 1975), these approaches have not yielded invariant data. It is a truism that correlative research cannot demonstrate a causal effect, and this is a serious problem in the neural correlates paradigm. Uttal (1967) asks: "at what level of correlation, if not perfect, should we accept an action of the brain to be identical with a state of behavior?" (p. 627). Without this one-to-one relationship of bioelectric event to behavior, anything we can try to say about brain-behavior relationships is very tentative (and possibly misleading).

The nervous system is complex and highly integrated; as the number of units increase so does the functional complexity (Grundfest, 1959). This complexity is compounded by the fact that the CNS produces the highest integrated behaviors when functioning as a whole rather than in a non-integrated fashion (Sherrington, 1947). Grundfest further states that the interplay of different kinds of electrophysiological processes leads to complications in overall behavior. Thus, the behavioral or neural output of brain processing could potentially involve every neuron in the brain. As one investigates more complex behaviors, both the behavior and any concommitant electrical activity should become correspondingly more complex.

The complexity of the nervous system at basic levels is well illustrated

in a study by Gerstein and Kiang (1964). They report, in studies of units in the cat's auditory cortex, that although a particular unit may respond with a statistically reliable pattern to a particular stimulus, there is variability in response to single stimulus presentations. Burns and Smith (1962) note that all cortical units monitored were in constant activity, and that the firing pattern of these units could change as a result of the presentation of any stimulus. Fox (1970) states that a small percentage of primary cortical cells are responsive to stimuli in the relevant modality, but may respond to stimuli in other modalities. O'Brien and Fox (1969a,b) report that approximately 80% of the cells which they studied in the motor cortex responded to stimuli presentations in other sensory modalities.

John and Morgades (1969), in training cats to press one bar to a 2 Hz light flicker, and another bar to an 8 Hz light flicker, noticed that the electrical response in the lateral geniculate body differed for each learned response. They state, however, that single unit activity displays many different response patterns to the same stimuli. This was in contrast to the responses of multiple units which display invarient types of discharge to the same stimulus. Thus they concluded that the "neural ensembles" are a more reliable source on which to base the differential behavioral response than are the firing patterns of single cells.

The variability in response of the CNS during complex behaviors was first demonstrated in ablation and learning experiments by Karl Lashley. Lashley's Laws of Mass Action and Equipotentiality state that the loss in a learned function is not the result of the location of brain tissue within a functional area removed, but its volume. This has been demonstrated in much more recent studies of the effects of lesions. Galambos, Norton

and Frommer (1967) made multiple lesions in the visual system designed to interrupt the retinotopic projection representation in visual cortex, and obtained little disruption of pattern vision. They concluded that they were unable to reconcile their findings with others (Hubel, 1963; Hubel and Wiesel, 1963, 1965) which suggest that very orderly connections are necessary for pattern vision. Further, research by Doty (1958) indicates that after extensive lesions in visual cortex were performed, little disturbance in pattern vision was noticed.

Since responses of single cells are so variable, and since single cells provide only a small sample of the populations that are functional under behavioral conditions (O'Brien and Fox, 1969a,b; John, 1972; John and Morgades, 1969), some workers attempted to relate SWs to behavior. Since SWs appeared to reflect the synaptic activity from the underlying neural population, it seemed reasonable that they could be correlated with behavior. However, SW correlation with behavior has not yielded the data necessary for firm statements of brain-behavior relationships either. For example, hippocampal theta has been related to orienting (Green and Arduini, 1954; Leaton, 1965); learning and memory processes (Elazar and Adey, 1967); movement (Dalton and Black, 1968; Vanderwolf, 1969); and tonic immobility (Harper, 1971). Habituation, on the other hand, has been related to increased (Thompson and Shaw, 1965; Sharpless and Jasper, 1956), and unchanged (Worden and Marsh, 1963) EP amplitude. Interestingly, attention has been related to increased (Jane, Smirnov and Jasper, 1962; Horn, 1960) and decreased (Shaw and Thompson, 1964) EP amplitude.

Once again, these studies indicate the "problem" of variability. To further overcome this problem, gross data reduction techniques such as signal averaging have been used. However, this type of data reduction

underestimates the possible significance of variability. Signal averaging is misleading (Walker and Long, 1976) and may be destroying a large part of the information the CNS utilizes.

By way of illustration, Walker and Long provide data on 140 visual EPs from alert hooded rats. If these 140 sweeps are averaged it is apparent that the late positive component occurs at about 240 msec post stimulus time (PST). However, a PST histogram shows that the latencies of the components were essentially randomly distributed between 200 and 260 msec. If two separate averages are formed, one average of those sweeps with a late component latency less than 240 msec, and another average with a component latency of greater than 240 msec, some interesting data emerge. These separate averages show gross changes throughout the post-stimulus period. Besides generally shorter latencies for all components in the "before 240 msec" averages, a new positive component emerges for the "after 240 msec" averages. Thus, a single average of all 140 EPs necessarily obscures data and misrepresents some neural process.

Further, Walker (1974) reports that digital positive fast potentials (PFPs) in the rat visual cortex are not well represented by signal averaging. By the averaging criterion, then, these potentials are not of importance. Yet, at least some of these potentials are of adaptive significance since they can be conditioned to a stable occurrence to gain reinforcement. Thus, it seems as though such techniques may not only suppress data, but that they can be misleading and misrepresentative of CNS function.

Variability in responses of the CNS is not the only problem with the neural correlates design; there is also the problem of measurement. We cannot specify which neural parameter to measure because the neural

concommitants of a behavior cannot be specified. Also, we are not capable of specifying the relevant aspects of behavior being used as a correlate for neural events (Fox, 1970). A further problem is that our measurements of behavior are not on a time base fast enough to correlate them with a neural event.

One last problem of neuro-behavioral designs in the adapting organism is that the stimulus which evokes the neural signal changes in its significance over time (Fox, 1970). Research on habituation of EPs indicates that if a stimulus is presented repeatedly with no behavioral consequence for the animal the EP will diminish in amplitude (Morrell, 1961; Galambos et al., 1956).

The inability of correlative designs to deal with the variability of the CNS, the problem of what and where to measure, and the changing significance of brain events, seriously limits the use of correlative designs for the study of neural events. However, this leaves us in a precarious position. Still desiring to work with adaptive behavior, yet unable to gain useful information from correlative designs, we are forced to find a procedure which still gives a possibility for understanding brain and brain events in an adaptive situation while avoiding the problems of correlative research.

Operant Control of Neural Events

The operant control of neural events (OCNE; Fox and Rudell, 1968) is a procedure which escapes the limitations of the correlative paradigm, yet still permits the study of adaptive behavior. The OCNE approach provides for a study of bioelectric response parameters that encode learned behavior (Fox, 1970). The study and quantification of these response parameters

are necessary before any laws of adaptive neural function can be specified. It is not possible to correlate activity in the brain with behavior without these neural laws. The OCNE approach can provide data on rate of acquisition, the conditionability of wave components, and the return of the component to baseline after cessation of the reinforcement contingency. This information can help lead to the formulation of laws of adaptive function.

One stable source of information in bioelectric events is not in the behavior but in the neural signal itself. The signal can be understood in terms of amplitude, frequency, latency, and in some cases, occurrence and non-occurrence. These parameters can be manipulated and quantified.

An underlying assumption in the OCNE paradigm is not that the SW is being conditioned, but that conditioning illustrates the relationship between the SW and a certain state of the SW generators. The conditioned state then can be shown to be reliable enough to allow specification of the most adaptive state of the generators in terms of a particular SW parameter (Walker, 1974).

In the OCNE paradigm, the bioelectric signal (EEG or EP) is no longer the dependent variable as in the neural correlates paradigm; it becomes both a dependent and an independent variable. The animal is placed in a situation in which it is allowed a free range of behavior. A reward which has previously been demonstrated as important to the animal is given if the animal brings about a specified change in the pattern of the bioelectric signal. Thus, it is the brain wave which is manipulated and the object of study. Using this procedure puts one in a position to study brain "independently" of molar behavior; independently in the sense that the behavior becomes almost irrelevant to the reinforcement.

Neural Conditioning: General

Much research has been conducted in the conditioning of bioelectric phenomena; these include rate of hypothalamic unit discharge (Olds, 1965); rate of single unit discharge in motor cortex (Fetz, 1969; Fetz and Finocchio, 1971); and hippocampal rhythmic slow activity (RSA; Black, 1971b; Black et al., 1970). Other conditioning research has involved high and low voltage EEG (Carmona, 1967) and alpha waves (Peper and Mulholland, 1970; Nowles and Kamiya, 1970). In addition, there is a large volume of literature on the operant conditioning of epicortical EPs (Fetz, 1969; Fox and Rudell, 1968; Rosenfeld and Fox, 1970,1971; Rosenfeld and Owen, 1972; Rudell, 1970; Rudell and Fox, 1970; Walker, 1974; Walker and Shaver, 1972a,b).

The first question which may arise about the OCNE paradigm, or neural conditioning in general, is if the change in the bioelectric response is the result of trivial mediation. That is, if the change results from such things as a specific posture or orientation to the stimulus, then conditioning of neural events has not so much conditioned the event as it has conditioned the feedback from skeletal muscle.

Rudell (1970) noticed that while being conditioned to increase amplitude of a component of the photic EP, rats assumed a posture which oriented them towards the light source. This may, of course, mean that the neural change was brought about by a skeletal response or by retinal absorption of light. Rudell noticed that the task to increase amplitude was not affected by administration of atropine, varying the light intensity, or utilization of various postures. The rats' assumed postures, he concluded, were all examples of superstitious behavior.

Rudell and Fox (1972) had also ruled out timelocking to the stimulus

as the reason for neural conditioning by choosing a 20 msec PST component to condition. The latency of this component is less than the reaction time of the animal; therefore timelocking had to be ruled out.

Rat Evoked Potentials

The rat has a rather reliable evoked potential with little variability of most components. The components which occur reliably are named Type I potentials (Walker, 1974). Walker has provided much normative data on rat evoked potentials; the potentials, their variability, and names are all after Walker.

The first response to photic stimulation is a relatively fast positive potential (P1) occurring at approximately 25 msec post stimulus time (PST). While this potential is not always seen in all rats, when it occurs its latency varies little as evident in sweep averages. Following P1 is a negative potential (N1) which peaks at about 25-35 msec PST. This is usually the most negative potential in the EP. The next component is a positive potential (P2) occurring reliably at 50-60msec PST. P2 is usually the most positive peak in the EP; the standard deviation of this component is about 2 msec. Following P2 is a long slow negative sweep (NS) which is easily seen in averages. The duration of NS is ended by a late positive (PL) component occurring in the range of 175-228 msec. While there is considerable latency variation in PL, it is reliably present. Since the latencies of the initial components display little variability the duration of NS will be affected by the latency of PL.

Superimposed on NS is variable activity termed Type II activity. These deflections are termed positive fast potentials (PFPs). They are characterized by variations in amplitude (1-2μV to 100μV), frequency

(30-80 Hz), and whether or not they occur (Walker and Long, 1974). These potentials display such variability that unlike P1, N1, and P2 they cannot be adequately described by signal averaging. Walker and Long hypothesize from the digital nature and independent occurrence of these PFPs that the temporal pattern is of possible informational significance.

Significance of the Late Component

It is generally accepted that the primary components of the EP (P1, N1, P2) occurring in the range of 10 - 50 msec PST, are related to stimulus input (Creutzfeldt, Rosina, Ito, and Probst, 1969; Purpura et al., 1960). Fehrmi, Adkins, and Lindsley (1969) have demonstrated that the initial components (about 12 msec PST) of the monkey EP convey all the necessary sensory information to allow the animal to perform correctly in a visual discrimination task. Creutzfeldt et al. (1969) found that the initial component of the first positive wave (15 - 50 msec) of the EP correlated with the discharge of the on-center afferent fibers in the striate cortex of the awake, paralyzed cat.

The later components in the evoked potential are seen to be the most susceptible to changes induced by conditioning (Morrell, 1961). John (1969) argues that the late component is "endogenous", that is, released by the organism as a function of its state, and is triggered by the stimulus. Sutton, Tueting, Zubin and John (1967) set up an experiment in which four types of auditory stimuli were used: (1) a single, soft stimulus; (2) a single, loud stimulus; (3) a double, soft stimulus; and (4) a double, loud stimulus. The subject was instructed to predict either loud versus soft or single versus double. In the single versus double situation a late positive component appeared at about 225-300 msec PST. This component

appeared whether or not the second stimulus occurred. John (1969) stated that variables such as subject uncertainty about the appearance of a stimulus, what will appear, and the time interval between stimuli all exert influence on the later components of the human EP.

That the occurrence of late components may be related to attentional factors in animals is reported by Kimura (1962) and Pickenhain and Klingberg (1965). Kimura asserts that late positive discharges in the rat EP, which he termed multiple responses, occur only after the animal is accustomed to the experimental situation, and that their occurrence is correlated with a low level of activity in the brain stem activating system. These responses recur at 160 - 240 msec intervals after the initial evoked response at 30 msec. These responses occur in the region of those potentials which in this study are termed PL and all later positive components.

Pickenhain and Klingberg report that these same discharges, which they termed after-discharges (AD) do not appear during orienting and searching behavior in the rat, and that they increased during the time the animal was falling asleep but not in sleep. Further, AD disappeared totally after an application of shock, but tended to reappear if the animal learned an escape task following presentation of shock. They further report that they believe a connection exists between AD and an inhibitory system whose "neurophysiological mechanism is unknown."

It appears, from the research cited above, that the later components of the EP are more susceptible to changes in arousal or attention and are more a reflection of the organism's state than are earlier components. Since this is the case it would seem that the late components are most likely to be significant in terms of adaptive behavior and conditioning should focus on the later components rather than the earlier components

which seem to be reflections of the classical visual pathways.

Most conditioning studies, whether correlative (Haider, Spong and Lindsley, 1964; Davis, 1964; Hernandez-Peon, Scherrer and Jovet, 1956; Galambos, Sheatz, and Vernier, 1956; John and Killam, 1959) or using the OCNE approach (Fox and Rudell, 1968, 1970; Rosenfeld, 1975; Rosenfeld and Owen, 1972; Rudell, 1970) have taken as the point of focus the amplitude of the potential. Perhaps this is because amplitude reflects the coherence of currents which are responsible for the genesis of the SW. This conclusion results from the model of the genesis of SWs which are thought to be a summation of voltage drops in the extracellular resistance (Purpura, 1967; Landau, 1967). Presumably then, one may think that it is easiest to relate the amplitude to either behavior or the generators of the SWs. On the other hand, little research has been done on latency. Vaughan, Costa, and Gilden (1966) reported good relations between latency and stimulus intensity. Shucard and Horn (1971) have reported that latency variations may give information about the state of the nervous system when the stimulus is delivered. However, the prevailing view seems to be one of skepticism that latency can be of functional significance. Mountcastle (1969) asserted that latency is not likely to be a measure of intensity because no time reference for the subject is available. This position is also held by Burns and Pritchard (1964). Thus, it seems that if latency cannot even qualify as a measure of stimulus intensity it is very unlikely to qualify as a measure of any more complex behavior.

The functional significance of a component can be assessed by whether or not it can be conditioned (Fox, 1970; Fox and Rudell, 1968). If a component can be manipulated by the animal for adaptive purposes (e.g. gaining food reinforcement when hungry) then this component must be meaningful to the animal. This is not to say that the change is

related to a particular behavior, but only that this component can be manipulated by the animal to gain something from the environment.

Assuming that the ability to condition a component demonstrates its functional significance, then several other questions logically follow. First, are the changes in one component independent of changes in other components? That is, can the occurrence, amplitude, and latency of other components be shown not to covary with changes in the conditioned component? Fox and Rudell (1970) argue that components of the EP are capable of independent encoding of behavior. But is it the whole component or simply one aspect of the component? Are the changes in various parameters of the conditioned component independent of one another?

With these questions in mind this study seeks to demonstrate several things. First, using the OCNE paradigm, an attempt will be made to operantly condition the latency of a late positive potential in rats. If this attempt is successful, then functional significance of this component shall have been demonstrated. Thus, the first part of the study will be a replication of previous experiments, except that here latency will be conditioned instead of occurrence or amplitude. Latency was chosen for one specific reason. Latency does not seem to be an important factor as far as functional significance is concerned. However, if the latency of this component can be conditioned then its functional significance will have been demonstrated. This leads to the second part of the study. A change in the latency creates opportunities to compare changes in the whole EP and aspects of several selected components with preconditioning waves. It is expected that changes in the late component will be independent of the other EP components studied. But what of the other aspects of PL? If the latency and amplitude are found to covary then it might be concluded that the component operates as a unit in encoding a behavior

or set of behaviors. However, if latency is found to change independently of other parameters of PL then it must be assumed that latency, by itself, is of functional or adaptive significance. That is, if changes in latency are what the criterion for reinforcement is, and these changes occur independently of other parameters of the component, then latency must be the factor of adaptive significance. It is the only important factor in the study.

Method

Subjects. Subjects were two adult male hooded rats weighing 289 grams and 295 grams before food deprivation. Both chronically implanted animals were reduced 85% body weight by food deprivation before baseline recording started.

Surgical Procedure. Both animals were chronically implanted bilaterally with twisted pair bipolar electrodes made of teflon coated 0.007 gauge stainless steel wire. The deepest tip extended 1.5 mm past the other; recording was done over the cross-sectional area. The animals were implanted over Area 17 of visual cortex (Bregma - 7.0 mm, lateral - 3.0 mm; Skinner, 1970). The shorter electrode of each pair rested on dura while the other was implanted 1 1/2 mm below the cortical surface.

The proximal ends of the electrode pair were joined by dental cement (Hygenic Co.) and inserted into a four connector nylon plug (Plastic Products Co.) which was then fastened to the animal's head with dental cement. Stainless steel screws (0.080 guage) were used to butress the dental acrylic plug. Since there was one electrode pair from each hemisphere, the possibility existed of being able to record potentials in each hemisphere and between hemispheres. In practice, however, recording was done only from the right hemisphere.

Equipment and recording. Training and recording sessions were carried out in a Skinner box in electrical shielding provided by a modified refrigerator shell. The inside of the shell was painted white and dimly lit by a small dc bulb. A hole had been cut in the top of the shell for insertion of the strobe head of a Grass PS22 photostimulator, which was set at 16, the highest setting. Another hole was cut in the front of the shell for observation. Both holes were covered with copper

screen. A small circulating fan, which ran throughout the experiment, was fixed to one side of the shell.

The Skinner box was made of clear plexiglas and measured 28 cm long x 24 cm wide x 33 cm high with an alleyway inside 8 cm wide and 12 cm long. Two sets of photocells were fixed just in front of the food cup at the end of the alleyway. These photocells had to be broken before food reinforcement would be delivered. One end of a connecting cable was screwed to the nylon plug on the animal's head and the other end was secured to a wood block resting on top of the Skinner box. From this end another cable led to input/output connectors fixed in the side of the refrigerator. Cable from the output connectors led to two Tektronix 26A2 differential amplifiers with filters set at 10 and 100 Hz, and the gain set at 2K. A "T" connector simultaneously passed the signal to a Tektronix 502A Dual Beam oscilloscope, and to a PDP8e (Digital Equipment Corp.) digital computer for on-line reinforcement and data collection.

All experimental procedure was under on-line control of the PDP8e. Standard inputs to the computer included a teletype, A to D converter, and a paper tape reader. Signals from the animal were fed, via a patch panel, into the computer, and the number of waves collected and reinforcements delivered were counted on two digital counters. Each wave collected was digitized in 256 points with a time duration of 512 msecs. The collected waves were stored on an Iodisk disk pack with each wave taking up one storage block. A total of 3,146 blocks were available for data storage on each disk.

To determine the latency of PL in an on-line computer controlled operant conditioning situation, a program was developed which would search for PL in a specified latency zone (window). A component occurring in

this time zone was then tested against a stencil which could recognize PL X msec after its real-time occurrence. This stencil obtained the amplitude differences between a reference point (c) and points earlier and later in time (c - x msec and c + y msec; where y may equal x). The two amplitude values obtained by subtracting the amplitude (m) at c (M_c) from the amplitude at c - x (M_{c-x}) and c + x (M_{c+x}) were obtained. These values could be referred to amplitude values in core memory and if pre-specified criteria (C) for M_{c+x} and M_{c-x} were exceeded PL was then tested against a latency criterion. If PL exceeded the amplitude criteria and satisfied the latency criterion, one of 1023 (decimal) digital output patterns could occur to deliver reinforcement or identify waves for later reference in disk memory. The delay and duration of reinforcement were variable within the constraints of the Inter-Trial Interval which, along with all other parameters, was a keyboard variable entered for each animal. The program specifying all parameters for each animal's conditioning task was written separately (Ratstn, Westerberg, 1976) and stored as subroutines in a larger program (Debbie, Shaver, 1975) which controlled the broad constraints of presenting stimuli and throughputting coded data to disk for later retrieval by purely off-line programs. These programs could recognize a disk-stored digitized representation of the analog brain signal by either its class membership, coded by one of the 1023 different possible combinations of 10 ID bits, or by the number of its order in the daily presentation of stimuli. Advanced off-line programs could compute x-y and y-t histograms or DCPs (Distribution of Criterion Potentials, Walker and Shaver, 1974) whose variety was limited only by the imagination of the experimenter. These histograms could be subjected to mathematical computations (+, -, ÷, x, differentiate, integrate). Any wave could be

recalled for xy display on an oscilloscope or for phone line transmission to an IBM 360 computer which was used as an input device to a calcomp plotter.

Procedure. Each animal was connected and placed in the Skinner box, which allowed as free a range of behavior as permitted by the space. When the animal approached the foodcup, it broke the first set of photocells which, via the computer, triggered a flash of the photostimulator. The resultant EP was fed through the amplifiers and into the computer and tested against the reinforcement parameters in Ratstn. If the component was a criterion response as determined by the window, stencil, and latency criterion, a tone sounded and a pellet of food was delivered.

Each animal was run in the following series:

(1) Baseline condition - Each animal was given five to six days of baseline training and allowed to learn how to trip the photocells for food. During this time no reward contingency was in effect, so the reward was delivered each time the photocells were tripped. After the stimulus was delivered, a tone sounded, and a pellet of food rolled into the foodcup. If the animal responded maximally it could obtain one reinforcement every 10 seconds. The 10 second delay was instituted to avoid interference by chewing artifact. For Animal 2 the task was learned quite easily. By the second day it was pressing the bar fairly regularly for food. Animal 1 had to be placed in the Skinner box for two days before it responded over 10 times. The third day was the first day of actual data collection for this animal.

The data from the baseline recordings were then visually scanned and processed by off-line computer programs to yield a latency criterion point for the conditioning part of the study. Obtaining the parameters for determining what was to be a criterion response for PL were based on two

factors. First, PL had to exceed a certain amplitude. A stencil was utilized which specified minimal M_{c+x} and M_{c-x} parameters such that PL was just large enough so it could be distinguished from other activity on the EP. A test of this stencil revealed that it reliably picked up components P2, PL and P3 when they were visually distinguishable from lower amplitude activity on the EP. Second, if PL was present, as specified by the stencil, it had to occur a low percentage of the time at a particular latency. This latency was determined by constructing y - t histograms which were then integrated to find a latency which occurred 20% to 25% of the time. Both amplitude and latency criteria differed slightly for each animal because of differences in the amplitude and latency of PL in Baseline.

(2) Conditioning of PL - Conditioning was divided into two parts, Phase 1 and Phase 2. The latency criteria determined in Baseline underwent constant revision throughout the course of the experiment. It had become obvious by the early days of conditioning that the latency parameters obtained during Baseline were no longer useful. Instead of starting at the latency criteria determined in Baseline the animals were shaped into decreasing the latency of PL over a period of days by narrowing the criterion by two msec per day. If the animal generated PL at that latency about 60% of the time it was decreased another two msec the next day. The amplitude criterion was kept at the same level which had been determined in Baseline. Conditioning proceeded in this fashion until enough days of conditioning had been run for the effect to have been demonstrated.

Phase 1 - Each animal was trained to decrease the latency of PL. After the parameters had been selected the animal was allowed to use whatever means possible to decrease the latency of PL to criterion, except for obvious behavioral manipulations (e.g. generating cable artifact by

shaking the head). Both animals were run for 300 trials per day in this phase until conditioning had been demonstrated. After 12 days for animal 1 and 15 days for animal 2, 100 trials with no reinforcement were run. This was used to signal the animal that a change in procedure had begun.

Phase 2 - This phase was used as a control phase. In this phase the animal was required to increase the latency of PL as far as possible back toward baseline. All parameters, except the latency criterion, were the same as in Phase 1. Phase 2 was run for 300 trials per day until the animals reached asymptote, which was 12 days for animal 1 and 11 days for animal 2.

Post-experimental analysis. Using the same off-line analysis programs described earlier, several methods of analyzing the results were used. First, using a special off-line program, averages were constructed for all trials for each animal each day. Second, daily latency and amplitude histograms for all trials for three components, P2, PL, and P3, for each animal were constructed. The so-constructed histograms were then integrated and visually scanned to find the median latency of each distribution. These median values were used as the data in all subsequent statistical analysis. Latency values in A to D units which were calculated from the integrated y - t histograms, were converted into msec PST. Amplitude histograms were constructed by subtracting the low point of the component found by a stencil from the previous high point. Thus amplitude was really a difference score. Amplitude values were not converted from A to D units to uV because the precision of A to D units would have been altered. All median values were then graphed to show the trend over days of conditioning.

The percent of criterion responses in Baseline, Phase1, and Phase 2 were also graphed. Daily latency histograms for P2, PL, and P3, daily

averages of all waves, and selected single sweeps were then transferred to magnetic tape for plotting.

Results

The data analyzed in this study consist of bipolar records of EPs to visual stimuli recorded from the visual cortex (Area 17) of the hooded rat. Several representative EPs are shown for animal 1 in Figure 1 and animal 2 in Figure 2. The records in these two figures illustrate the sequential changes in potential referred to as P1, N1, P2, NS, PL, and P3. The component of interest with regard to conditioning was the variable PL component. The top three traces of Figures 1 and 2 illustrate the wide range of latencies and amplitudes demonstrated by this component, and also illustrate the relative stability of the latencies of the earlier P1, N1, and P2 components. A visual inspection of Figures 1 and 2 also illustrate that although the latency of P3 is more variable than P1, N1, or P2, it is not strongly correlated with the latency of PL, although there are occassions when a particularly late PL (Figure 1, trace 3 and Figure 2, trace 3) is associated with a later P3.

For the purpose of identifying PL in an operant conditioning paradigm, a computer stencil was developed to specify PL. These stencils are illustrated by the inserts in Figures 1 and 2 and the digital pulse output signifying a fit to the stencil for the third EP is shown in trace 4. These stencils were applied to each EP on-line and in real time to determine if PL had occurred and if it had occurred at a latency within the time zone considered appropriate for each animal in each phase of the experiment. The fifth and sixth trace in Figures 1 and 2 illustrate EPs in which PL, as defined by the stencil, failed to occur. An inspection of these EPs in which PL did not occur reveals that there are no remarkable changes in the earlier P1, N1, or P2 components. Trace 6 also illustrates that an absent PL component can be associated with the presence of P3 components.

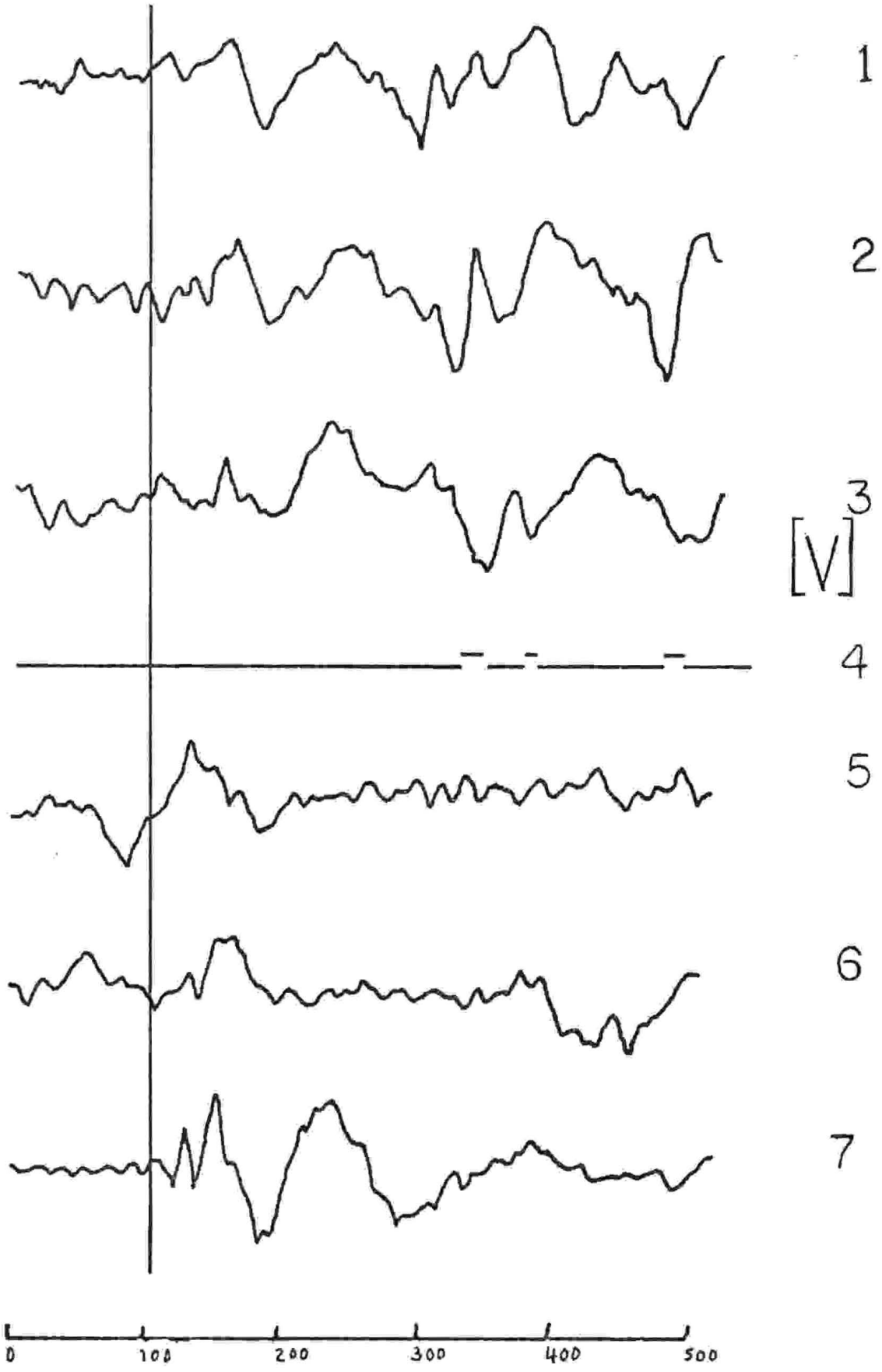

Figure 1

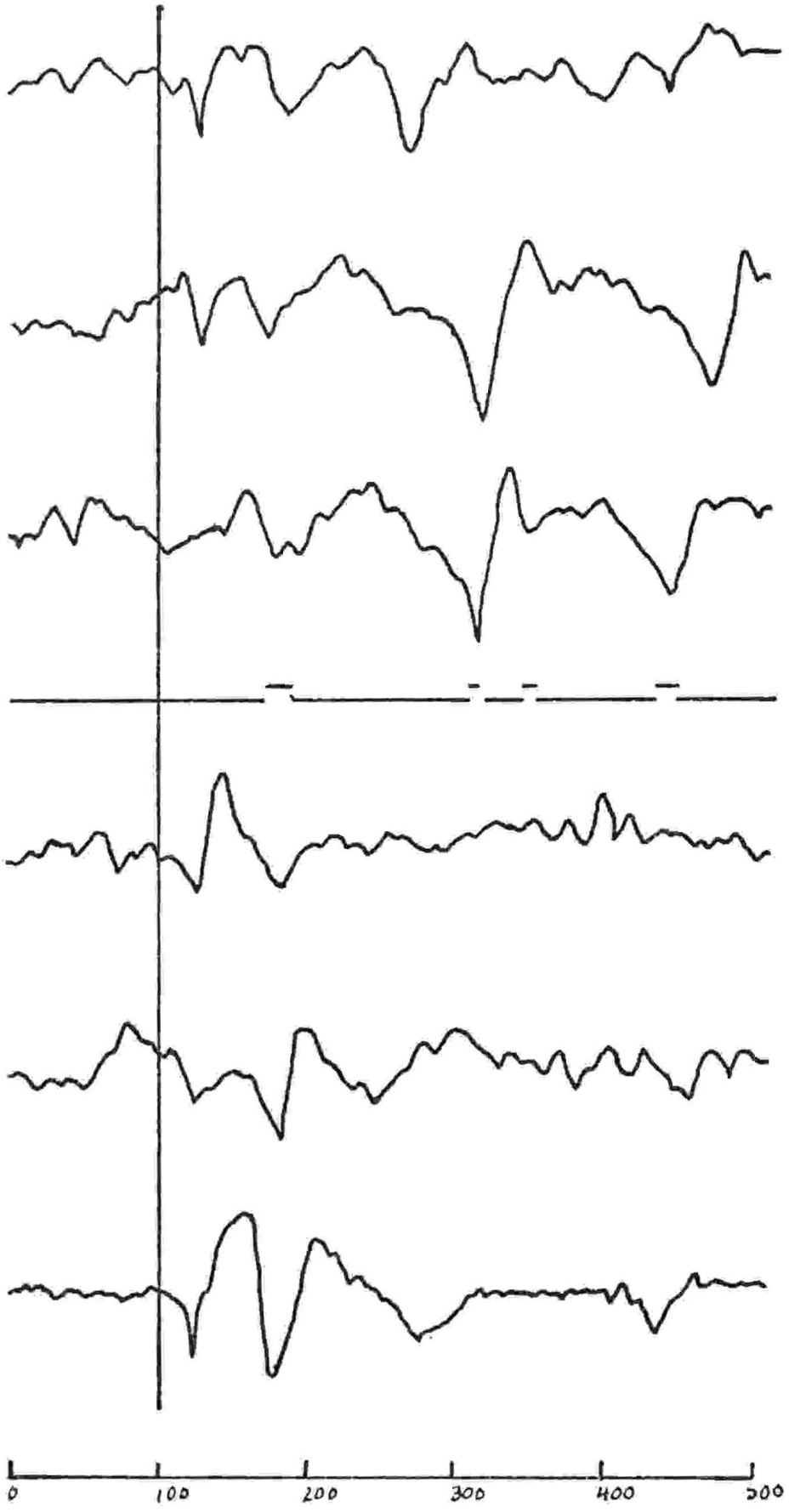

Figure 2

Trace 7 in both Figures 1 and 2 shown 300 averaged EPs. What is clear from these averages is that each component is shown occurring at one particular latency with a fixed amplitude, yet the single sweeps show that there can be obvious differences in the latency, occurrence, and amplitude of components, especially PL and P3. These differences may be understood more clearly by investigating the nature of the averaging process. The averaging process takes each single wave and separates it into 256 2-msec bins. For each bin the values of all 300 waves are averaged and thus become a point on the averaged wave at that particular latency. Thus, the latency of any component on the averaged wave describes the latency which occurs most often at that latency and omits other more variable latencies. Similarly, the amplitude of a component in the averaged wave is dependent on two things: (1) the amplitude of that component in each single sweep and, (2) the variability of each component in each single sweep. If a particular component occurs at quite variable latencies then each different latency will be summated in different bins which will decrease the amplitude and increase the frequency (duration) of that component. Thus, the more latency variability of a component the smaller the amplitude and the greater the frequency. What is seen then, in an averaged EP, is the non-variable activity; the variance is averaged out.

The principle data of interest in this study concerns the degree to which contingent reinforcement controlled the latency of PL. Closely related to the conditioning of PL latency is an investigation of the mechanisms involved in the production of components of a particular latency. Data relevant to this question is discussed after Baseline and conditioning data under the heading "Behavioral observations and behavior of P2, PL and P3 components."

Behavior of PL Under Baseline and Contingent Reinforcement Conditions

Figures 3 and 4 show daily averages of all EPs recorded each day in Baseline, Phase 1 and Phase 2 for animal 1 and animal 2 respectively. These averages are displayed in sequential order so that trace 1 represents the first day of Baseline and trace 7 in Figure 3 represents the first day of Phase 1 and so on. An inspection of both figures shows that PL is distinctly represented in the averages in the zone of 175 - 200 msec PST. The line at 100 msec marks the point at which the stimulus was delivered. It can be seen in both figures that the tip of PL in Phase 1 (Figure 3, traces 7 - 18; and Figure 4, traces 6 - 20) is at a slightly shorter latency than the tip of PL in Baseline (Figure 3, traces 1 - 6; Figure 4, traces 1 - 5). The tip of PL does not noticibly increase in latency during Phase 2 for animal 1 (Figure 3, traces 19 - 30). However, for animal 2 the tip of PL definitely shows increased latency during Phase 2 (Figure 4, traces 21 - 31).

The early components, P1, N1, and P2, are also clearly seen in all averages for both animals. PL occurs for both animals in a time zone of 20 - 30 msec PST. Of interest here is that P1 for animal 1 is actually a double component which continues from Baseline through Phase 2. For animal 2, however, P1 is a double component in Baseline which gradually becomes single in the time zone of 20 - 30 msec PST. N1 appears clearly in both figures as a upward deflection with a peak latency occurring in a time zone of 50 - 60 msec PST. It can be seen that in Figure 3 for animal 1 N1 occurs at an extremely stable latency of 50 msec PST, whereas for animal 2 (Figure 4) the peak of N1 is more variable with the variance increasing over conditioning. P2 appears as a downward deflection occurring in a time zone of 80 to 90 msec PST for both animals. Following P2 is

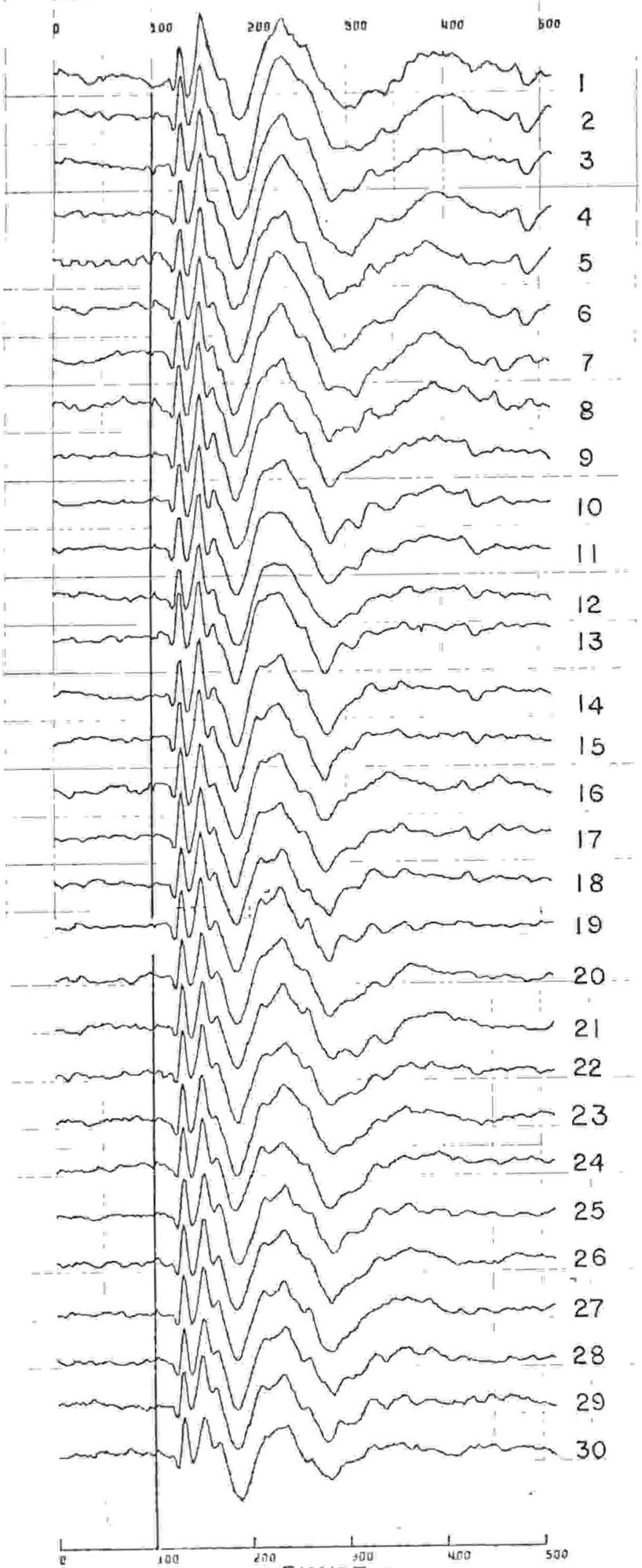
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FIGURE 3

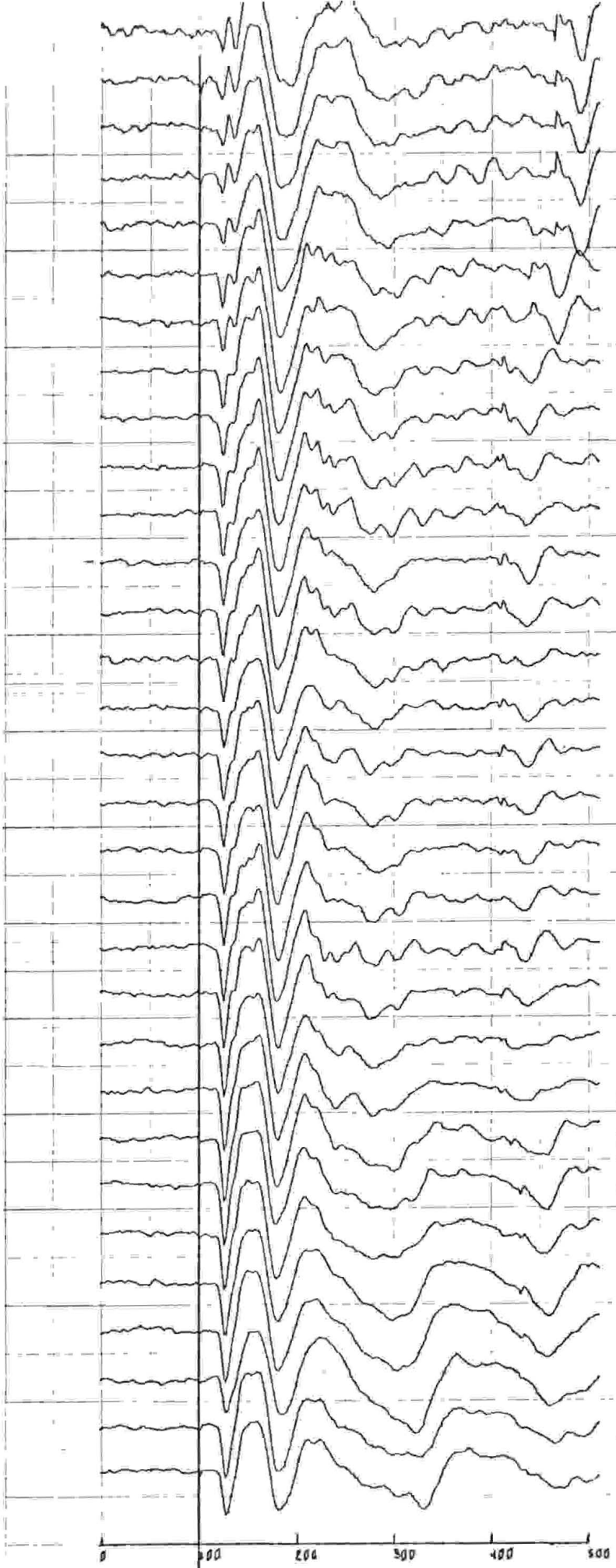
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FIGURE 4

a large upward deflection labeled NS. As can be seen from Figures 3 and 4 the latency and duration of NS varies with the latency of PL. This is quite clear in Figure 4 comparing the first 5 traces (Baseline) to the next 15 traces (Phase 1). Through Phase 1 NS becomes more distinct and peaks at an earlier latency than in Baseline. The latest positive component, P3, which follows P1, is also quite clearly seen in Baseline for both animals. P3 is the largest downward deflection occurring in a time zone of 300 to 400 msec PST. It can be seen that the tip of P3, especially in animal 2, decreases in latency during Phase 1 and shows an increase in latency during Phase 2 for animal 2.

The most revealing aspect of these averages is not in the latency of the tip of PL as much as in the fall time from NS to the tip of PL and the duration of PL. Figure 3 illustrates that PL for animal 1 has a rather short fall time and short duration as compared to animal 2. These differences are especially noticible in Figure 4, traces 25 - 31, compared to Figure 3, traces 25 - 28. It is apparent in Figure 4, especially in traces 15 - 21, that a new depression occurs at approximately 130 to 150 msec PST. This depression indicates that enough single sweeps large enough to be reinforced occurred in this earlier time zone. Thus animal 2 not only shifted the tip of PL to shorter latencies, but generated a new depression in PL to meet the reinforcement criterion. Concommitant with the rise of this depression was a lessening of the number of waves bearing PL in the Baseline latency time zone. This is further corroborated by the change in PL during Phase 2. During this phase the new notch in PL disappeared and once again PL reverted to a single notch component. Animal 1 did not demonstrate this change in PL over conditioning. This change in the form of PL for animal 2 indicates that simply reinforcing the tip

of a component does not restrict the animal to a "simple" latency shift of that tip; various other, more exotic means may be chosen to gain reinforcement.

The differences in the averages between the two animals is clarified by the histograms for animal 1 in Figure 5 and for animal 2 in Figure 6. Each histogram, called a DCP, contains the distribution of PL for each day in Baseline, Phase 1, and Phase 2. Each DCP is shown in sequential order, as were the AEPs; thus, Figure 5, trace 6, for example, is a DCP of the same waves which were shown in the average in Figure 3, trace 6. Figure 5 shows that in Baseline (traces 1 - 6) for animal 1 the number of components occurring in the zone of PL increased over days. During Phase 1 conditioning (traces 7 - 18) the distributions became distinctly bimodal in form with an increasing number of PL components occurring earlier. In early Phase 2 the distribution still remained bimodal, but the predominant mode shifted back to increased latency, until on the last day the distribution was almost unimodal in form. The main feature of the DCPs for animal 1 is that the number of components in the predominant mode in Baseline decreased with a resultant increase in the number of components in the earlier mode. That is, the modes reflected a change in the direction of conditioning.

The DCPs for animal 2 are yet more interesting and again corroborate the results seen in the averages. It can be seen in Figure 6, traces 1 - 5, that the number of components generated in the criterion segment increased over Baseline as in animal 1. The main difference between the Baseline performance of animal 1 and animal 2 is that the distribution of PL for animal 2 became bimodal before conditioning started. This suggests that animal 2 was already generating two latency classes of PL potentials.

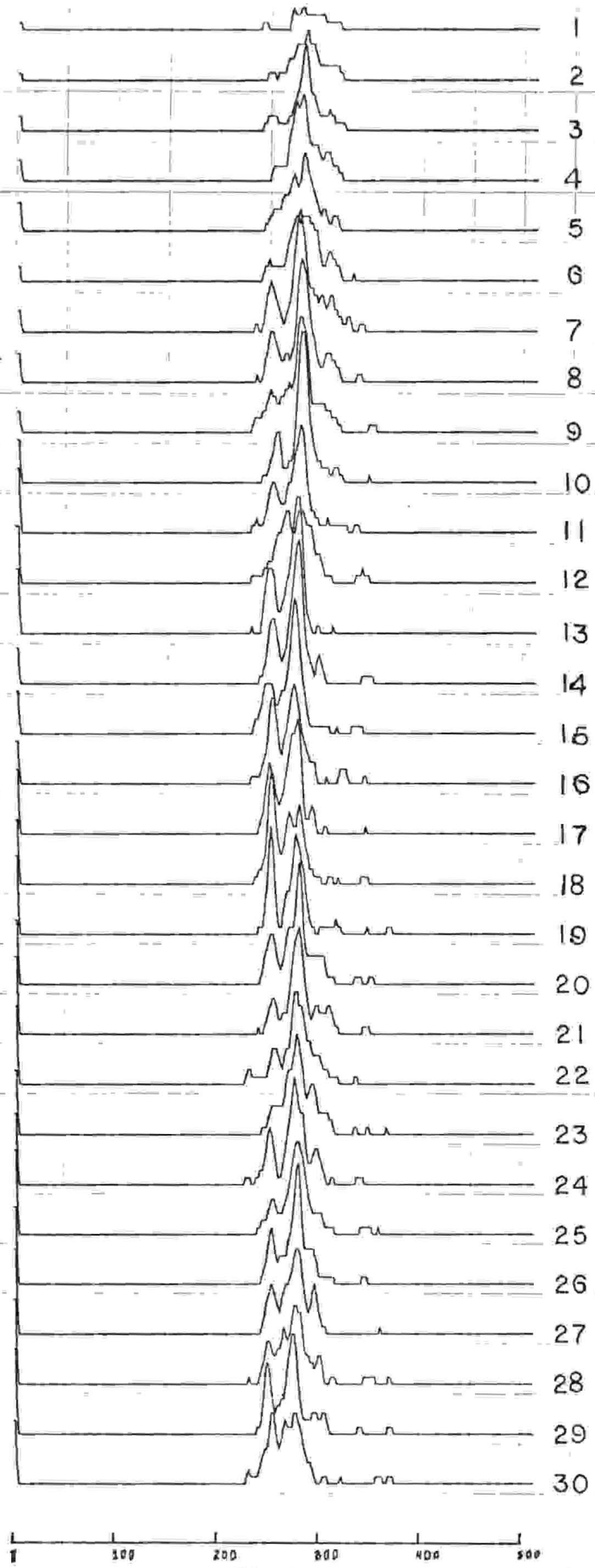
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FIGURE 5

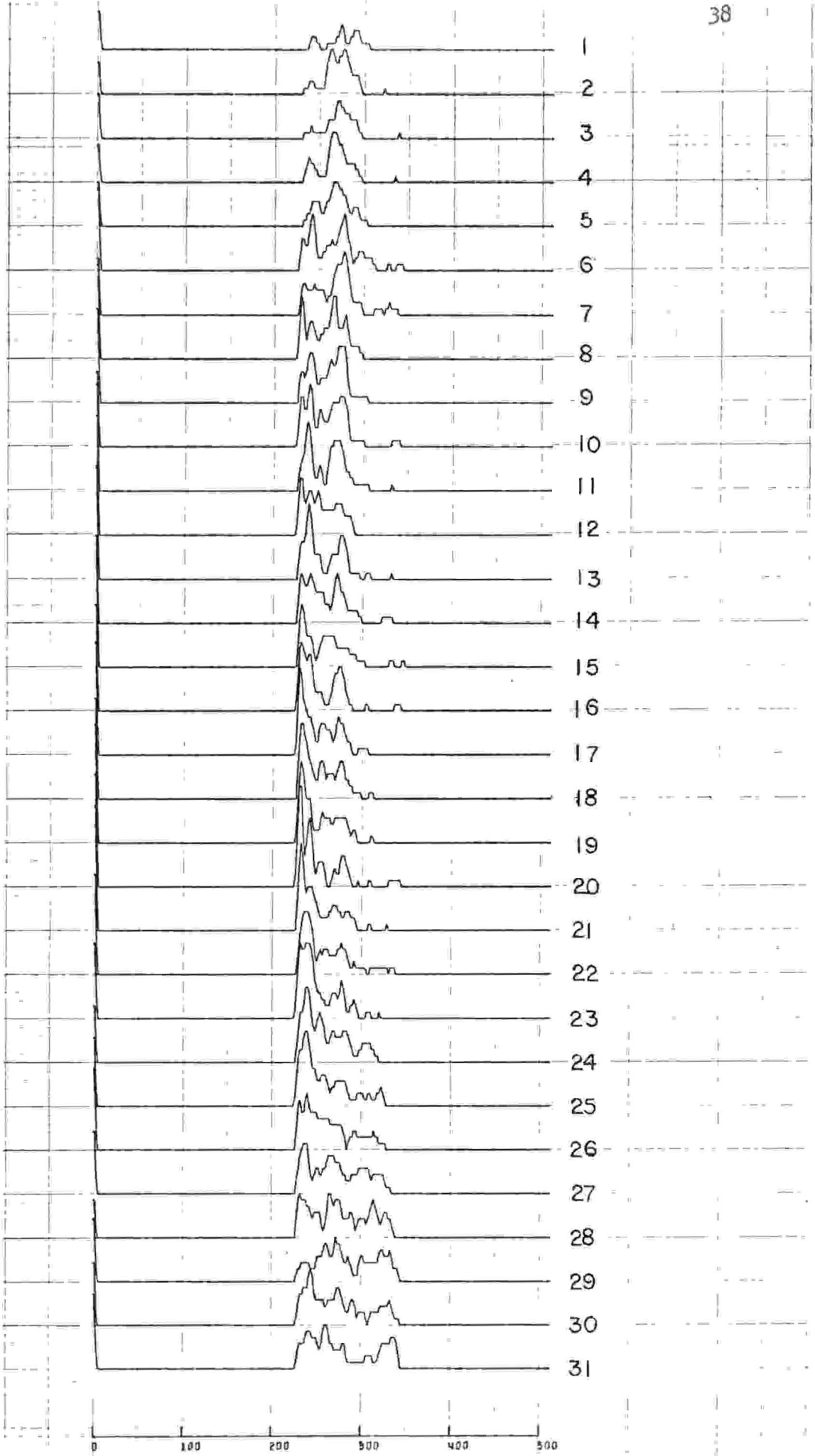
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FIGURE 6

During Phase 1 conditioning more potentials began to appear earlier which increased the earlier mode of the distribution, as did animal 1. But by day 12 of Phase 1 (trace 17), the distribution became tri-modal, indicating that the animal had begun to generate potentials in three separate latency classes with the earliest class containing an increasing number of potentials as Phase 1 conditioning progressed. Phase 2 histograms show a lessening of the number of potentials in the earliest latency mode with a return to essentially a bimodal form on the last three days of Phase 2 conditioning. Animal 2, then, also showed that the predominant mode changed in the direction of conditioning although instead of only two classes of PL latencies as seen with animal 1, animal 2 developed three classes.

In order to clarify the major changes in PL latency from Baseline through Phase 2 conditioning summated histograms were constructed for each animal for each condition. These histograms are seen in Figure 7 for animal 1 and Figure 8 for animal 2. A comparison of these figures across conditions illustrates the emerging bimodality from Baseline through Phase 1 for animal 1 and the trimodality for animal 2. It can be seen quite clearly here that the histograms as a whole reflect the changes seen in the daily DCPs. For animal 1, trace 1 illustrates that during Baseline the distribution of the medians of PL was unimodal. This distribution changed during Phase 1 (trace 2) to a bimodal distribution with the new mode appearing in an earlier time zone which corresponded to the latency decrease task in this phase. During Phase 2 for animal 1, no change in the summated DCP was apparent when compared to Phase 1 except for a lessening of the number of potentials in the whole distribution.

For animal 2 the summated DCPs in Figure 8 show that compared to Baseline (trace 1) the bimodality became more prominant in Phase 1 (trace

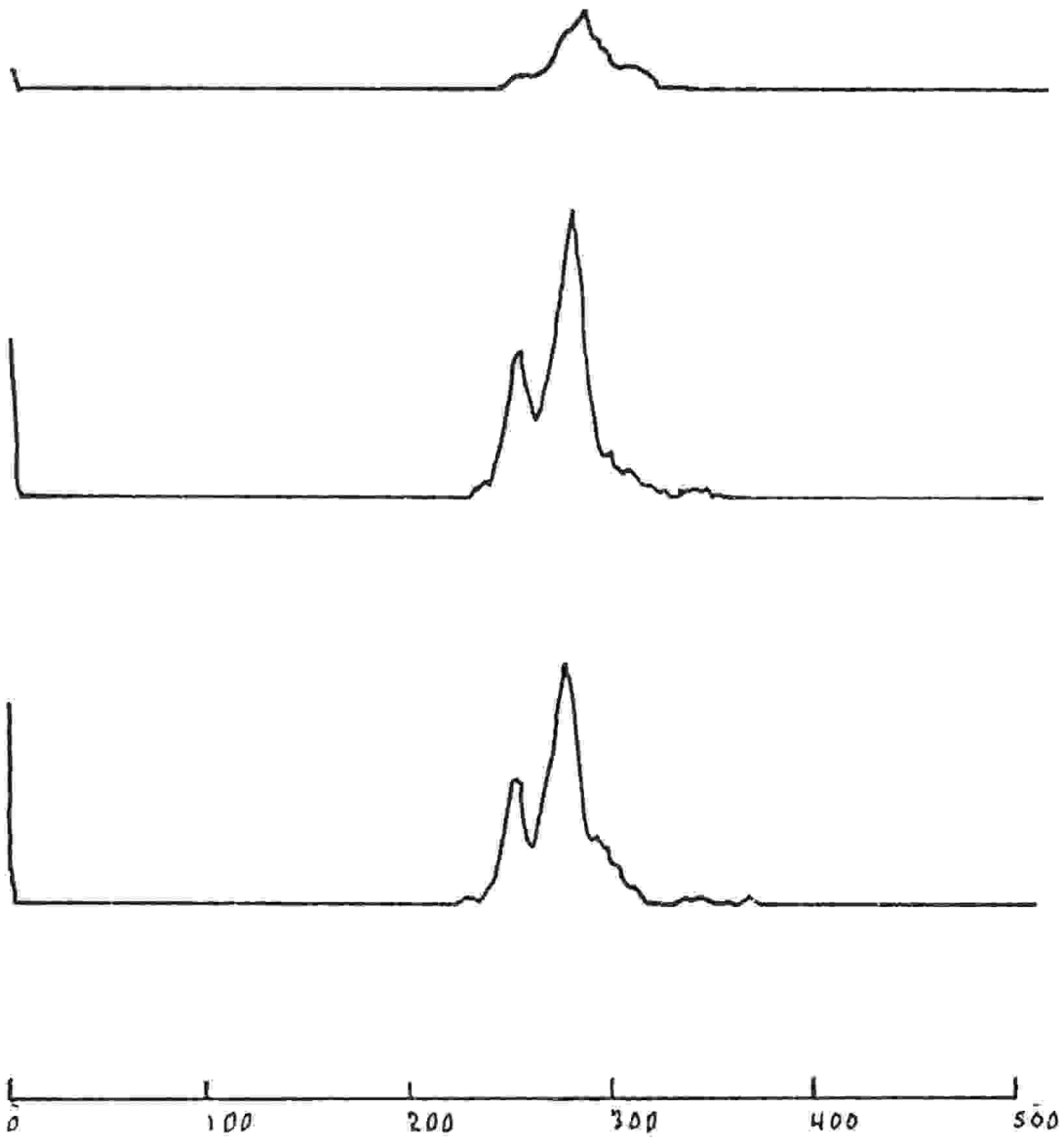

Figure 7

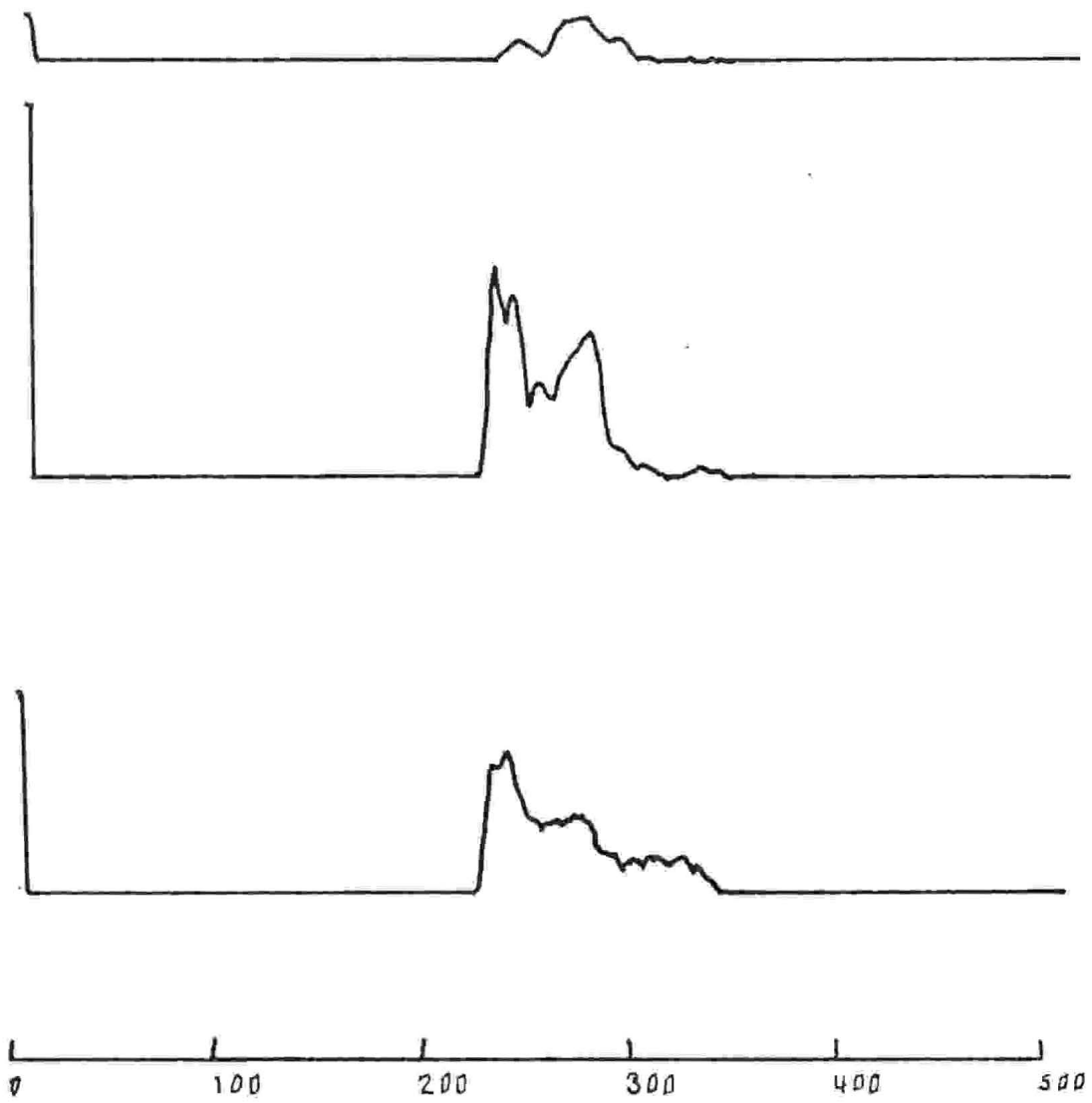

Figure 8

2) with an indication that a third mode might emerge with extended training in this phase. As with animal 1, the new mode emerged in the direction that conditioning specified in that phase. For Phase 2 (trace 3) it can be seen that the mode which emerged in Phase 1 was still evident, but that more potentials appeared at an increased latency which was the task specified in Phase 2. Thus, for this animal, even more so than animal 1, the summated DCPs reflected the direction of conditioning.

The differences in the DCPs in Figures 7 and 8 are corroborated by the data in Table 1 for both animals. Table 1 shows the first, second, and third quartile values for each of the three distributions in Figures 7 and 8. For animal 1, the table shows that there are changes in the distributions from Baseline to Phase 1 which reflect the changes seen in trace 1 and trace 2 of Figure 7. The quartile values also show a change in Phase 2 for animal 1, even though the change was not apparent from a visual inspection of Figure 7, and that these changes were in the direction specified by conditioning.

Of greatest interest in Table 1 for animal 1 are the changes in the first quartile from Baseline to Phase 1 and in the third quartile from Phase 1 to Phase 2. The first quartile change was 14.08, which corroborates the DCPs in Figure 7. It was seen from Figure 7 that the peak of the mode which occurred in Baseline still remained in Phase 1 at only a slightly decreased latency. However, a new mode arose earlier in time which would have been responsible for the major change seen in the first quartile. Interestingly, there was a 7.48 msec change in the third quartile from Phase 1 to Phase 2 which was not evident from the DCPs in Figure 7. This indicates that there was a change in not only the whole distribution, but that animal 1 did generate a later PL class in Phase 2.

Table 1

Quartile Values in Msecs for summated Histograms in Baseline, Phase 1, and Phase 2

Condition	Quartile	Animal 1	Animal 2
Baseline	1st	172.88	158.64
	2nd	180.42	168.66
	3rd	190.50	178.44
Phase 1	1st	158.80	137.16
	2nd	173.66	152.84
	3rd	180.94	171.32
Phase 2	1st	163.38	138.66
	2nd	174.92	157.00
	3rd	188.42	180.00

Animal 2 illustrated these same trends to a greater degree than did animal 1. From an inspection of Table 1 it can be seen that the quartile values accurately reflect the direction of conditioning. From Baseline to Phase 1, animal 2 clearly generated a new latency class of PL, which the first quartile change of 21.48 msec confirms. For Phase 2, the number of potentials in the earlier mode decreased with an increase of the number of potentials in a later mode, a fact confirmed by the change of 8.68 msec in the third quartile of Phase 1 compared to Phase 2. Thus, for animal 2 these quartile values show quite clearly that this animal did generate both an earlier latency class of PL in Phase 1 and a later PL latency class in Phase 2.

Tables 2 and 3 list the means and standard deviations of P2, PL, and P3 in Baseline, Phase 1 and Phase 2, for animal 1 and animal 2 respectively. The means were computed by taking the distribution (DCP) median for each day and averaging across all days in each condition. Table 2 shows the Baseline mean latency of PL for animal 1 to be 180.78 msec (range = 179.22 msec to 182.34 msec) with a low variability (S.D. = 1.27 msec). This low variability corroborates the graphic data seen in the first six traces of Figure 3. Figure 7 graphs the median of each distribution (DCP) over all conditions for animal 1. Animal 1 showed a gradually decreasing latency over days 2, 3, 4, and 5 of Baseline with a small increase on day 6. It was seen from the averages in Figure 4 that the variability of PL for animal 2 was greater than that of animal 1. The data to bear this out are listed in Table 3. The mean latency for animal 2 in Baseline was some 6 msec earlier than in animal 1 with a concommitant increase in variability (S.D. - 2.95 msec; range = 168.00 msec to 174.60 msec) as compared to animal 1. The median of each PL distribution for each day in Baseline, Phase 1, and Phase 2 for animal 2 is graphed in Figure 8.

Table 2

Means and Standard Deviations of the Median Latency and Amplitude of P2, PL, and P3 for Animal 1

Condition	Measure	Component P2	PL	P3
Baseline	X Latency	85.12	180.78	335.53
	S.D. Latency	1.18	1.27	3.52
	X Amplitude	78.83	85.08	95.22
	S.D. Amplitude	2.59	3.87	5.16
Phase 1	X Latency	83.64	174.32	333.52
	S.D. Latency	0.77	3.83	2.32
	X Amplitude	80.31	91.71	94.12
	S.D. Amplitude	3.85	2.75	2.87
Phase 2	X Latency	83.24	176.32	319.99
	S.D. Latency	0.83	2.66	5.83
	X Amplitude	70.24	100.91	95.95
	S.D. Amplitude	3.25	2.31	2.48

Table 3

Means and Standard Deviations of the Median Latency and Amplitude of P2, PL, and P3 for Animal 2

Condition	Measure	Component P2	PL	P3
Baseline	X Latency	82.90	171.32	345.36
	S.D. Latency	0.59	2.95	6.25
	X Amplitude	104.09	110.79	99.69
	S.D. Amplitude	8.25	2.93	3.76
Phase 1	X Latency	81.98	158.58	332.82
	S.D. Latency	0.94	7.23	4.94
	X Amplitude	117.12	109.14	97.17
	S.D. Amplitude	6.45	2.01	4.70
Phase 2	X Latency	81.82	161.98	339.24
	S.D. Latency	1.17	10.60	5.12
	X Amplitude	107.20	115.09	101.74
	S.D. Amplitude	5.24	4.99	3.52

Animal 2 showed latency decreases on days 2 and 4 and latency increases on days 3 and 5.

Phase 1 conditioning imposed rather marked effects on PL latency in both animals. The latency of PL decreased in animal 1 12.36 msec from the last day of Baseline to the last day of Phase 1 conditioning. This effect was yet more striking in animal 2 which showed a change from 168.76 msec on the last day of Baseline to 143.66 msec on the last day of Phase 1 - a 25 msec difference over Phase 1 training! From Baseline records a latency decrease of as much as 3.12 msec for animal 1 and 6.6 msec for animal 2 could be expected, but in both cases those changes were almost four times as great.

For animal 1, as can be seen from Figure 9, the longest latency in Phase 1 occurred on day 4 with the shortest latency on day 12, the last day of training in this phase. As seen in Figure 10, animal 2 showed the longest latency occurring on day 2 with the shortest latency occurring on day 15, also the last day of Phase 1 training. As is evident from the figures, the trend for both animals in Phase 1 showed a continual decline toward shorter latencies with no sign of asymptote in either animal as of the last day of Phase 1 conditioning, indicating that the latency of PL could have continued to decrease if Phase 1 had been continued.

Phase 2 (increase) conditioning exerted different effects on the two animals. Referring again to Figure 9, animal 1 showed a large initial increase on day 1 followed by increases on days 2 and 3, with a leveling on day 4 and 5, and then for the most part, a gradual decline over the remainder of Phase 2. Comparing the first day of Phase 2 conditioning with the last day of Phase 1 there was an increase of 6.02 msec. The difference between the last day of Phase 1 and the last day of Phase 2 was 2.20 msec increase. At no time did a value in Phase 2 for animal 1

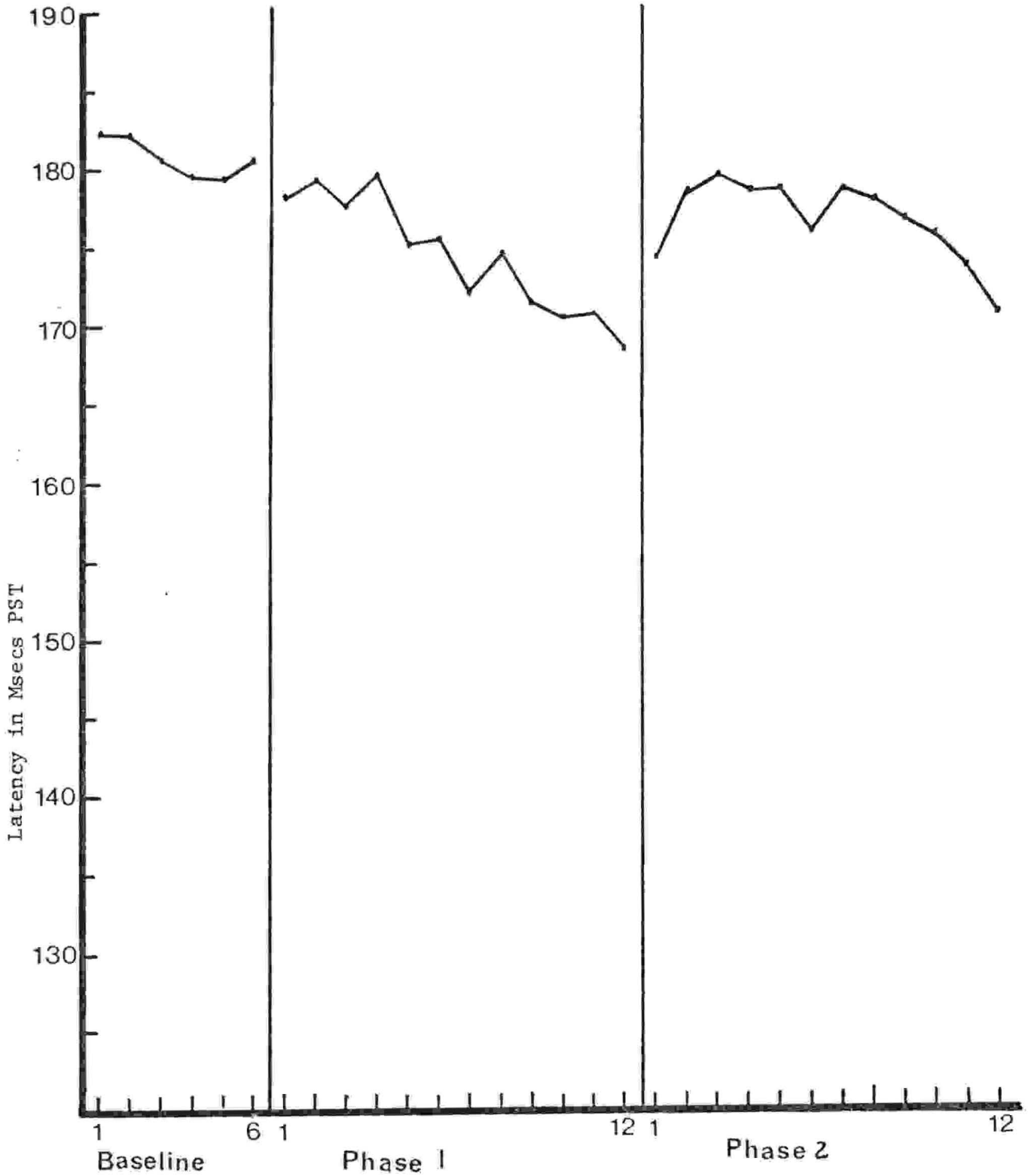
190
180
170
160
150
140
130
Latency in Msecs PST
1
6
1
12
1
12
Baseline
Phase I
Phase 2
Days

Figure 9

exceed either a value in Baseline or the mean of Baseline. Animal 2, however, showed a far more dramatic picture of Phase 2 conditioning. The initial increase from the last day of Phase 1 to the first day of Phase 2 was 4.54 msec. The latency increase from the last day of Phase 1 to the first day of Phase 2 was from 143.66 msec to 170.00 msec, or a change of 26.34 msec over Phase 2 training. This change is illustrated in Figure 10. One of the most striking aspects of Phase 2 conditioning for animal 2 was that after large fluctuations in the first three days, the latency increased to 177.76 msec which exceeded by 3.16 msec the longest latency in Baseline. As seen in animal 1, however, the mean of Phase 2 did not exceed the mean of Baseline.

The Baseline records in Figures 9 and 10 seem to indicate the form the latency decrease will take in Phase 1. Baseline changes in animal 1 tended to be gradual over days with no sharp increases or decreases. This course was followed in Phase 1 also where decreases were for the most part gradual, a trend that continued in Phase 2. Animal 2, on the other hand, showed abrupt latency increases and decreases in Baseline and continued this pattern in Phase 1 and Phase 2.

The variability in the latency of PL also changed considerably during training in both animals. Animal 1 displayed substantially greater variability in Phase 1 (S.D. = 3.83 msec) than in Baseline (S.D. = 1.27 msec). This was due to the fact that the latency in the last 7 days did not continue to increase. Animal 2 showed the same trend in Phase 1 as did animal 1. Variability increased considerably from Baseline (S.D. = 7.23 msec) but, unlike animal 1, rose yet higher in Phase 2 (S.D. = 10.60 msec). Again, the increase in Phase 2 variability is a reflection of the large latency increases in that phase.

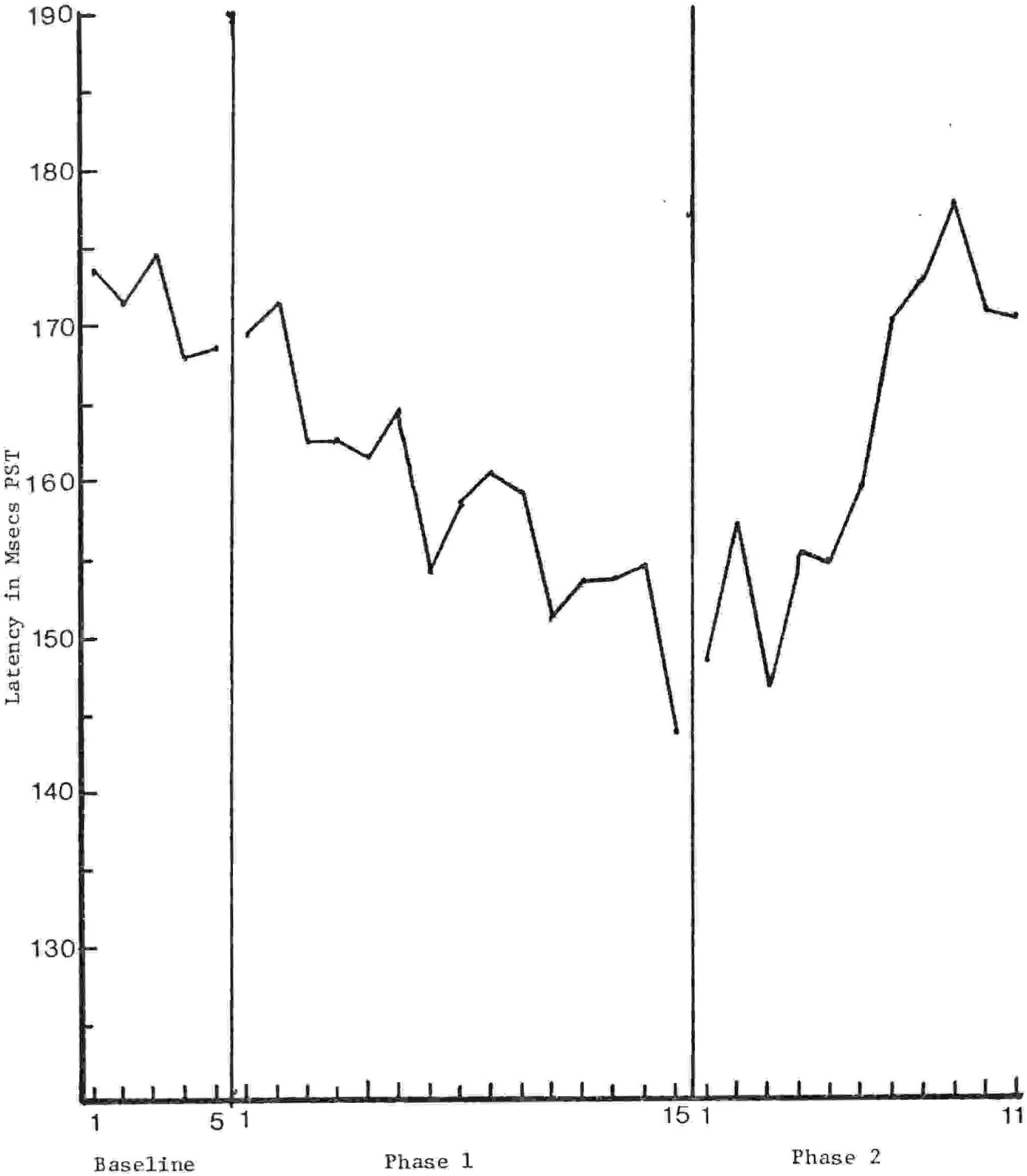
190
180
170
160
150
140
130
Latency in Msecs PST
1
5
1
15
1
11
Baseline
Phase 1
Phase 2
Days

Figure 10

Based on the changes in the latency of PL it should be expected that as conditioning takes place the percent of reinforcements should also rise. Figure 11 for animal 1 and Figure 12 for animal 2 show that this is what occurred in Phase 1. From Table 4 it can be seen that both animals generated about 24% criterion responses. By the end of Phase 1 the percent of trials on which the animals received reinforcement had risen to just over 53% in both animals. However, during Phase 2 animal 1 was able to generate only about 20% criterion responses, and animal 2 was able to generate only 34% criterion responses.

Even though Figures 11 and 12 reveal the same general falling, rising, and falling trend in Phase 1 these trends are not very much in evidence in Baseline or in Phase 2. What is revealing about the figures, however, is that the two animals showed quite large individual differences each day in the percent of criterion responses yet ended up with very nearly the same total percentage of criterion responses for both Baseline and Phase 1. Thus, a great degree of variability may exist across animals yet yield the same end result.

Behavioral Observations and Behavior of P2, PL and P3 Components

Behavioral observations. The success of conditioning raises the question of how the latency manipulations were accomplished. Behavioral observations were not recorded as dependent variables but observations throughout the course of the experiment yielded no surprising results. The animals remained in a posture which oriented them toward the light, remaining quite still. This posture was invarient throughout most of the study from late Baseline through both phases of conditioning.

Behavior of P2, PL and P3 during Baseline, Phase 1 and Phase 2. One might hope that the behavior of other aspects of the EP would provide clues

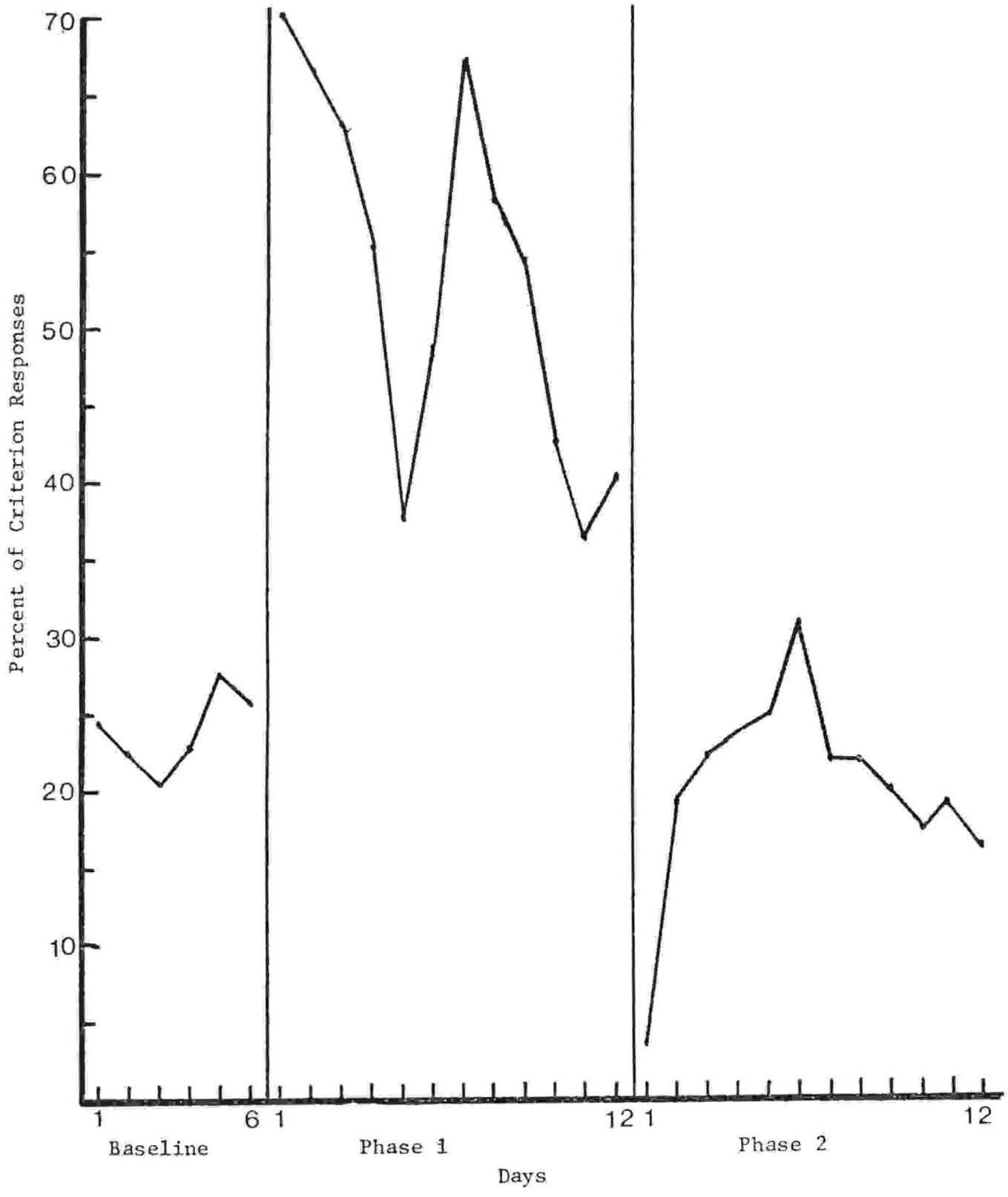
70
60
50
40
30
20
10
Percent of Criterion Responses
1
6
1
12
1
12
Baseline
Phase 1
Phase 2
Days

Figure 11

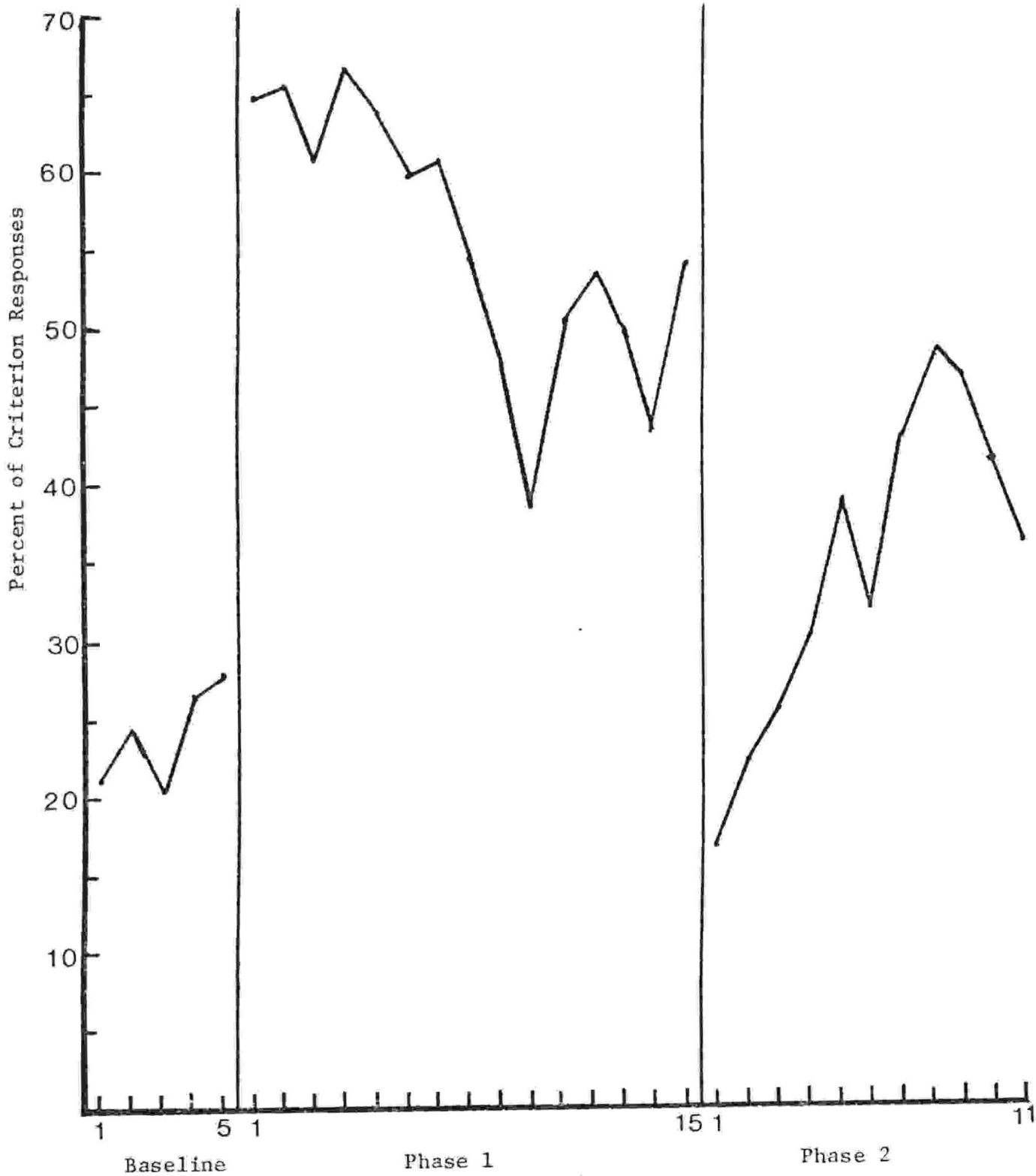
70
60
50
40
30
20
10
Percent of Criterion Responses
1
5
1
15
1
11
Baseline
Phase 1
Phase 2
Days

Figure 12

Table 4

Percent of Responses Reaching Criterion for Animals 1 and 2

	Animal	
Condition	Rat 1	Rat 2
Baseline	24.10	24.31
Phase 1	53.19	53.24
Phase 2	20.28	33.82

to the mechanisms of reinforcement changes in latency. Although there are some trends in the behavior of different components across conditions, the overall picture is that the manipulation of the latency of PL is relatively independent of changes in other aspects of response and that the mechanisms employed are likely to differ greatly between animals. For further analysis the amplitude and latency of components P2, PL and P3 were examined during Baseline, Phase 1 and Phase 2. The means and standard deviations of the basic data used in this analysis are shown in Table 2 for animal 1 and Table 3 for animal 2.

During Baseline the trend was for the variability of the latency of later components to be greater than that of early components. An inspection of Tables 2 and 3 shows the standard deviations of P2, PL and P3 to be 1.18 msec, 1.27 msec, and 3.52 msec for animal 1 and .59 msec, 2.95 msec, and 6.25 msec for animal 2. Conditioning tended to change this picture since in both conditioning phases for animal 2 and in Phase 1 for animal 2, the variability of PL became greater than that of P3. However, even over training the variability of P2 remained less than that of PL or P3, which can be seen in the averages in Figures 3 and 4 and in the graphs of the medians over training in Figures 13 and 14 for P2 and Figures 15 and 16 for P3. Figure 13 for animal 1 shows that there was essentially no change in the latency of P2 from Baseline through both phases of conditioning, although the variance decreased in both Phase 1 and Phase 2 compared to Baseline. Figure 14 also shows little variability of the latency of P2 for animal 2. For this animal the variability does increase somewhat from Baseline through Phase 1 and Phase 2 but is still quite low overall. Generally the variability of latency shows no trend for the three components across conditions. However, for both P2 and PL

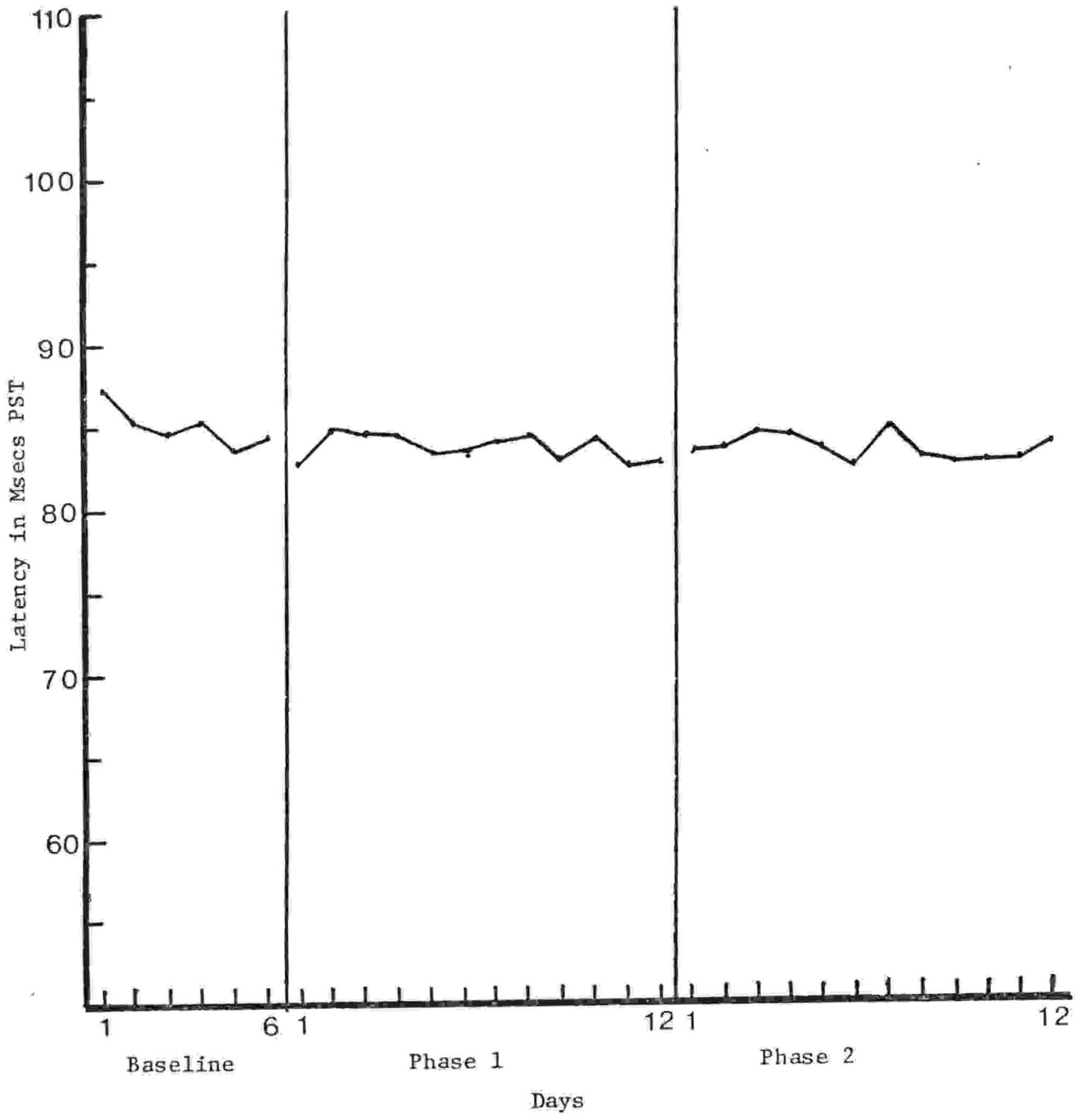
110
100
90
80
70
60
Latency in Msecs PST
1
6
1
12
1
12
Baseline
Phase 1
Phase 2
Days

Figure 13

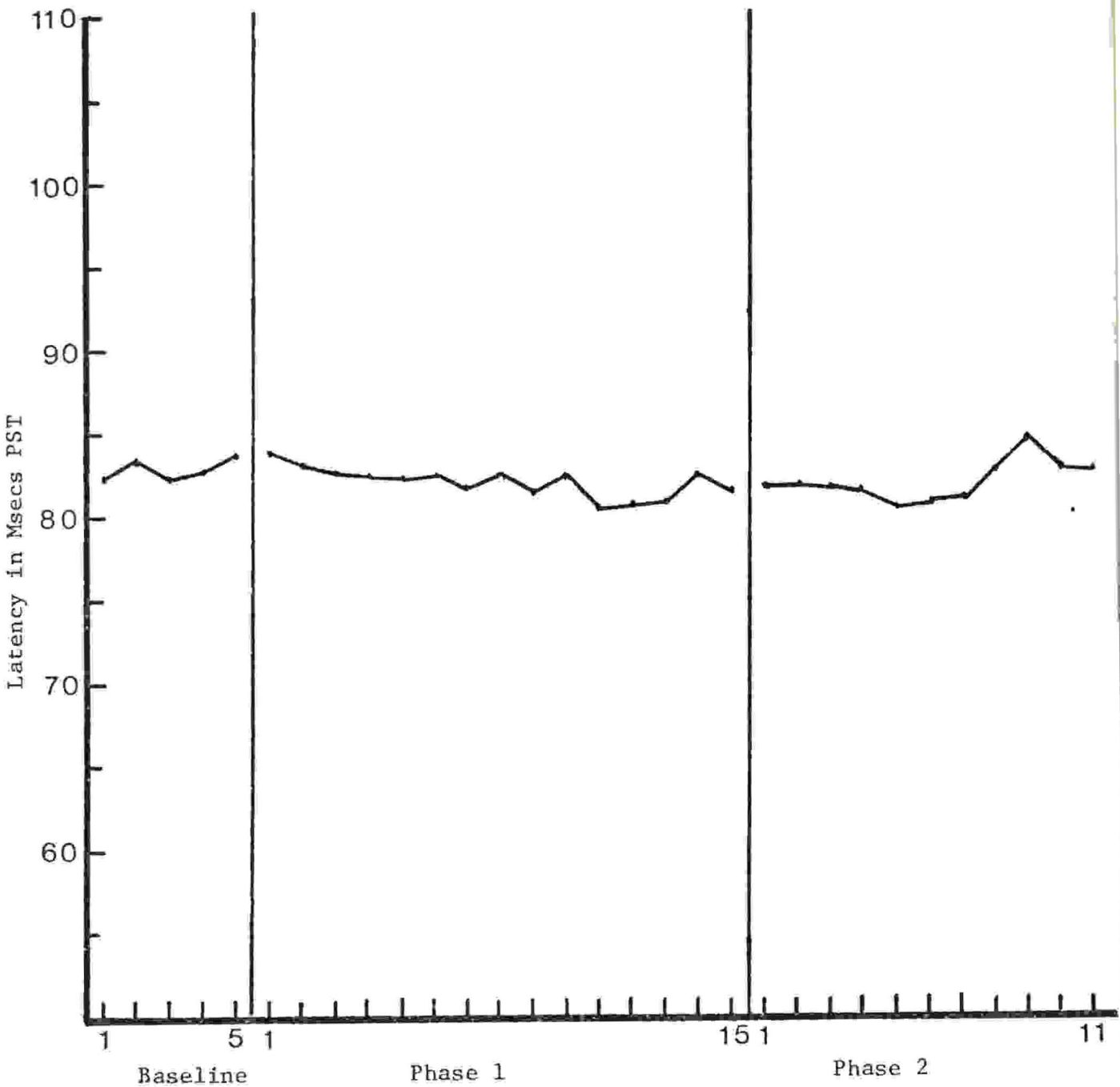
110
100
90
80
70
60
Latency in Msecs PST
1
5
1
15
1
11
Baseline
Phase 1
Phase 2
Days

Figure 14

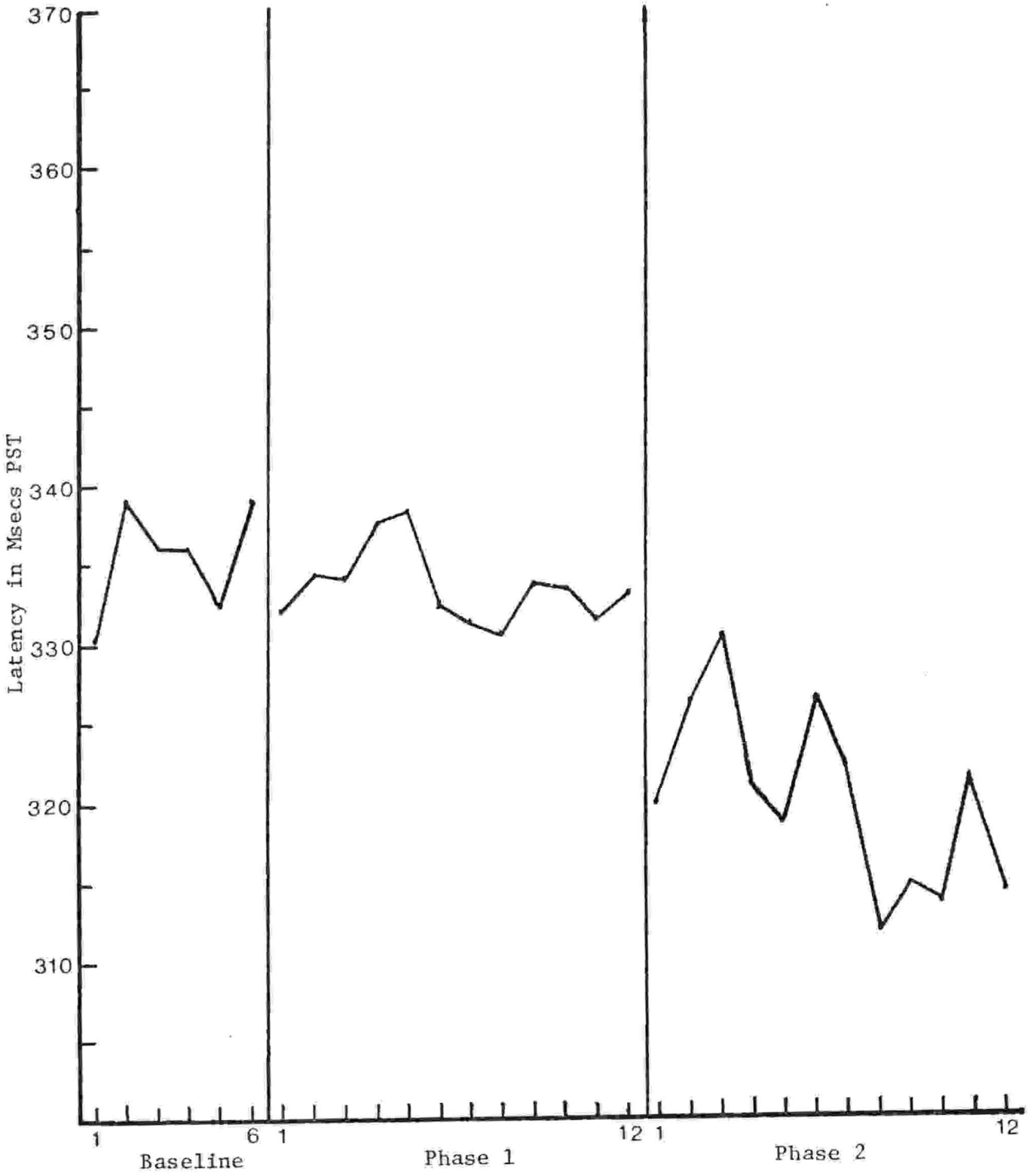
370
360
350
340
330
320
310
Latency in Msecs PST
1
6
1
12
1
12
Baseline
Phase 1
Phase 2
Days

Figure 15

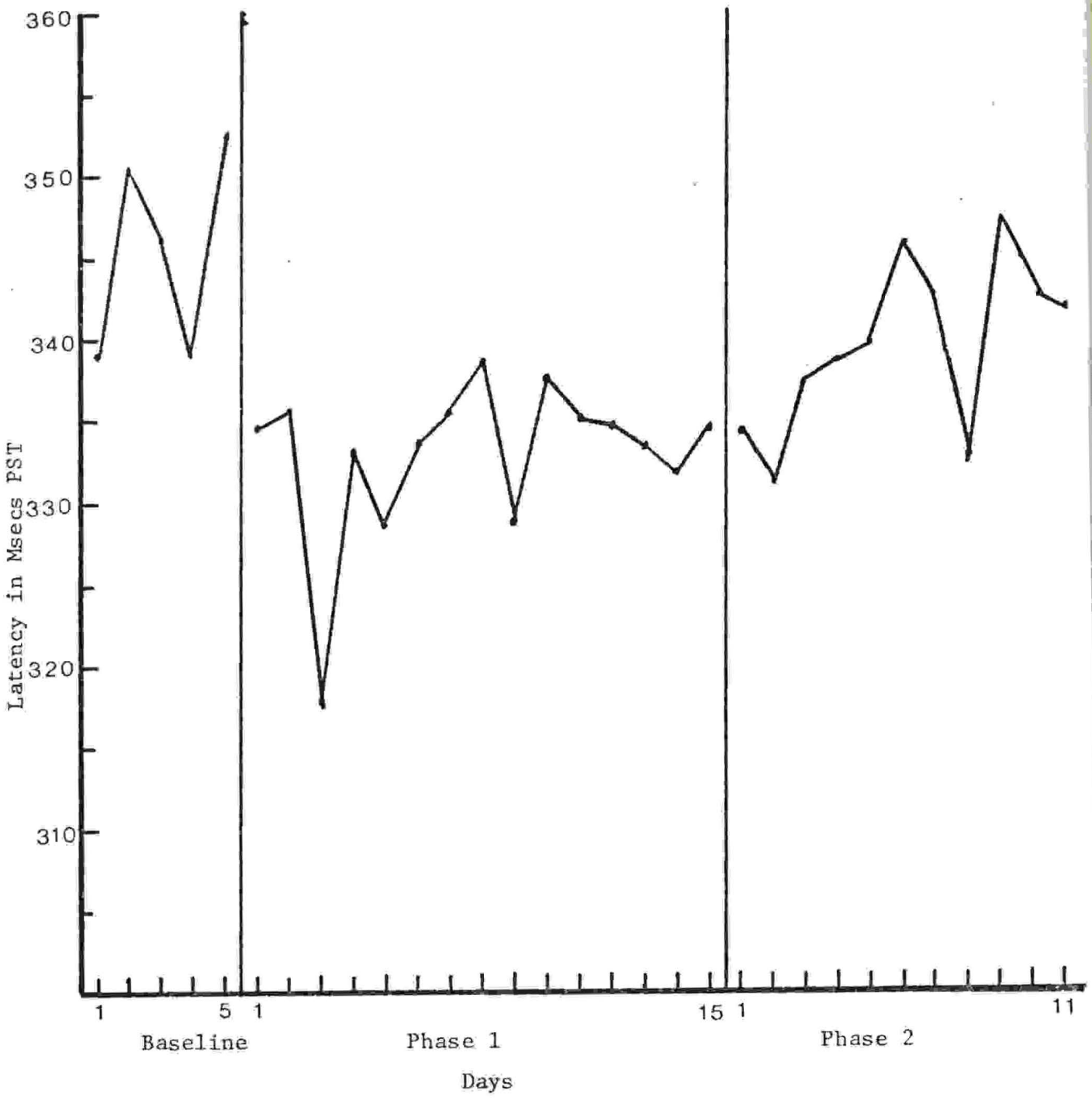
360
350
340
330
320
310
Latency in Msecs PST
1
5
1
15
1
11
Baseline
Phase 1
Phase 2
Days

Figure 16

in animal 2 variability increased throughout conditioning.

The variance of amplitude across or within components showed no consistent trends or opposite trends from one animal to another. The median amplitudes over days for the three components P2, PL, and P3 across all conditions are graphed in Figures 17, 19 and 21 for animal 1, and Figures 18, 20 and 22 for animal 2. Tables 2 and 3 also list the means and standard deviations for the amplitude of these same three components across Baseline, Phase 1, and Phase 2.

A visual inspection of both the tables and figures shows that the variance of amplitudes across components or within components across training showed no consistent trends or opposite trends from one animal to another. For example, for animal 1 the variance of the ampltiude of P2 increased from Baseline to Phase 1 and decreased from Phase 1 to Phase 2. For PL of animal 1, however, the variance decreased from Baseline to Phase 1 and decreased further in Phase 2. For animal 2 the variance of the amplitude of P2 decreased throughout conditioning, whereas for PL the variance decreased from Baseline to Phase 1 and increased in Phase 2. The only trends which occur are, that for animal 1 only, the variance of the amplitude of PL and P3 decreased from Baseline through Phase 2.

The overall picture emerging from the preceeding results is that there is a high degree of independence in the three EP components analyzed. Certainly there are no striking trends within animals and some rather pronounced differences between them. To further investigate dependencies between components, correlations were computed between latency and amplitude for P2, PL, and P3 for both animals. These correlation matrices are shown in Table 5 and Table 6 for animal 1 and animal 2 respectively.

An inspection of Tables 5 and 6 shows that there was little relation-

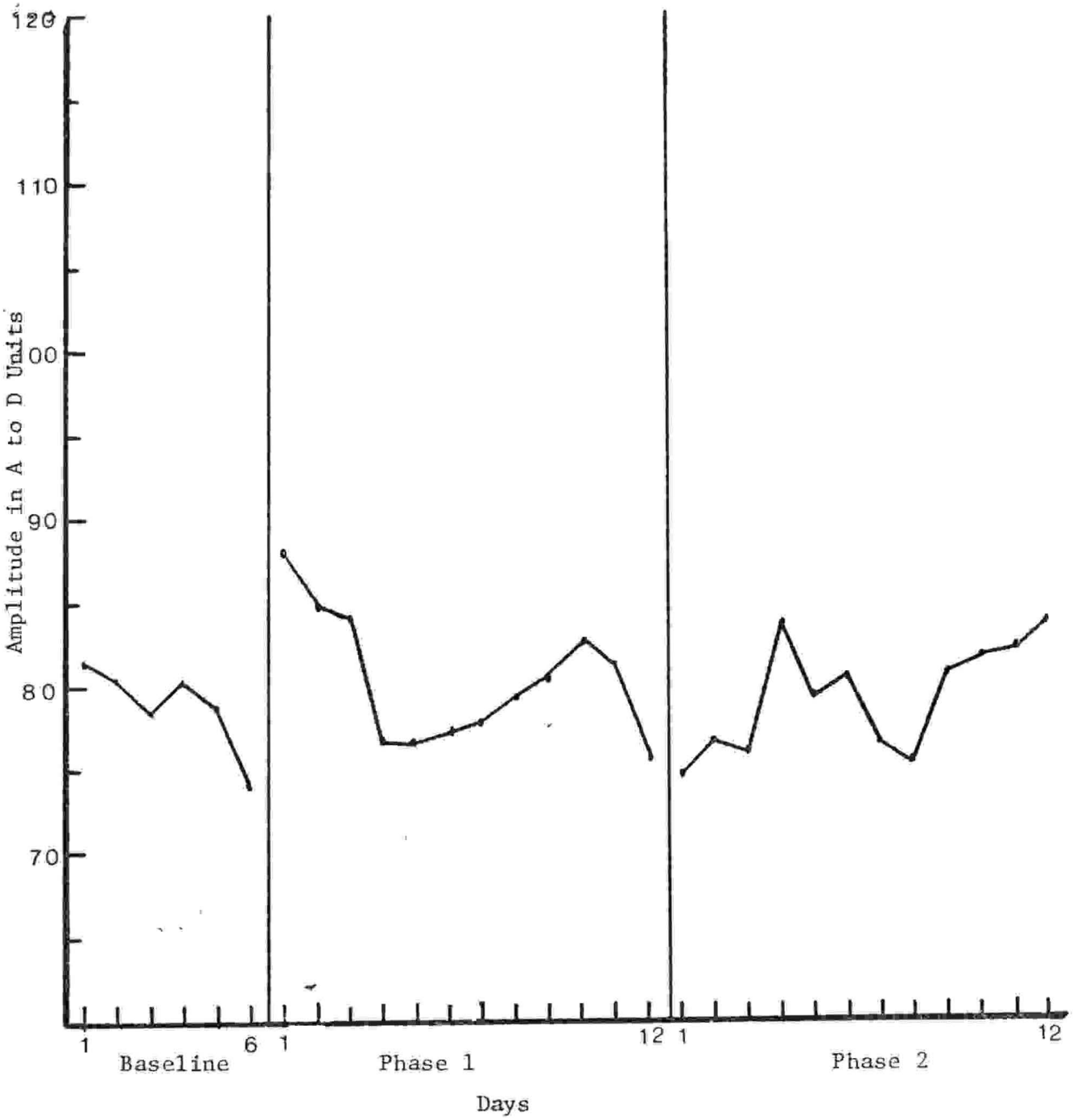
120
110
00
90
80
70
Amplitude in A to D Units
1
6
1
12
1
12
Baseline
Phase 1
Phase 2
Days

Figure 17

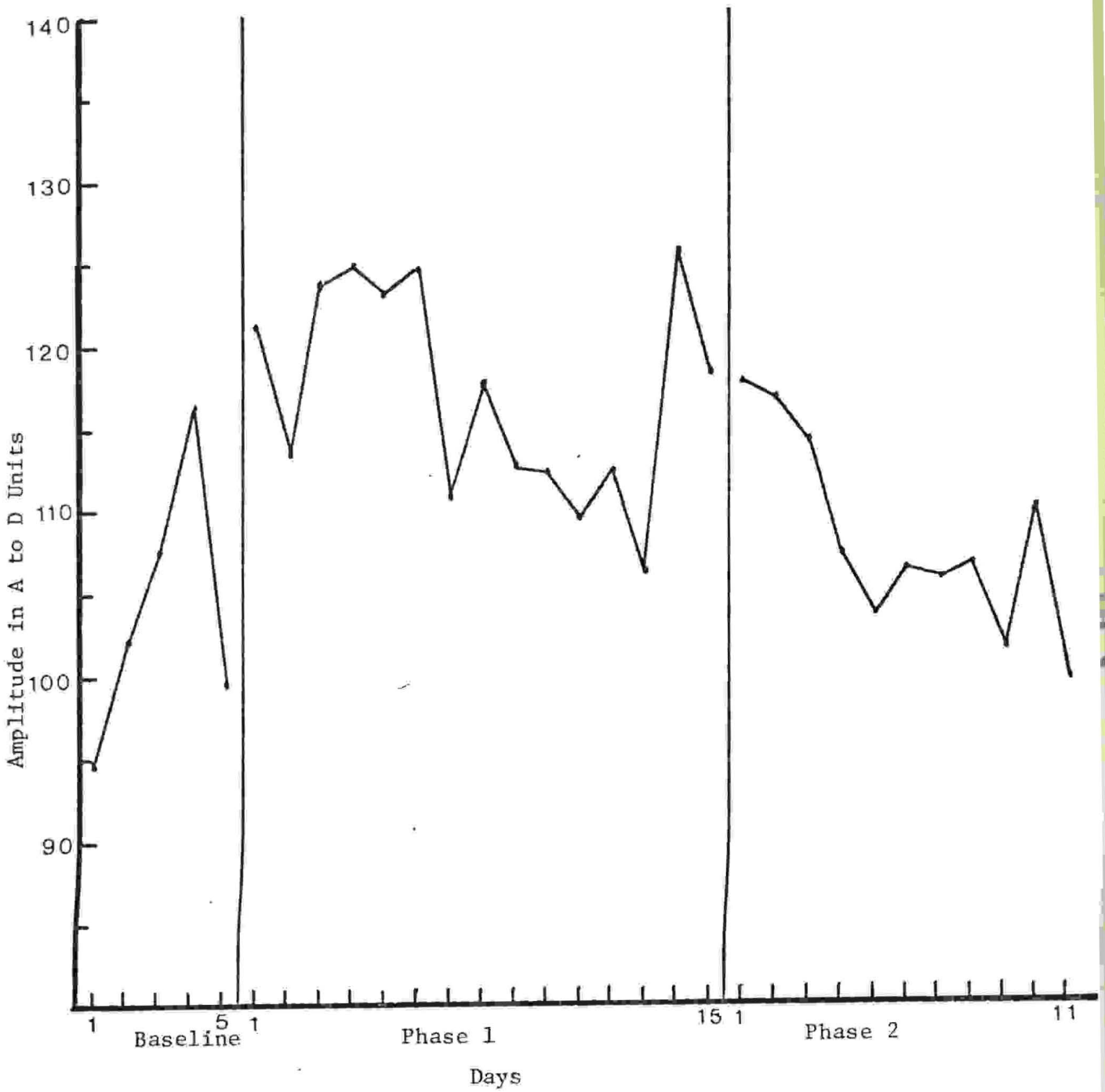
140
130
120
110
100
90
Amplitude in A to D Units
1
5
1
15
1
11
Baseline
Phase 1
Phase 2
Days

Figure 18

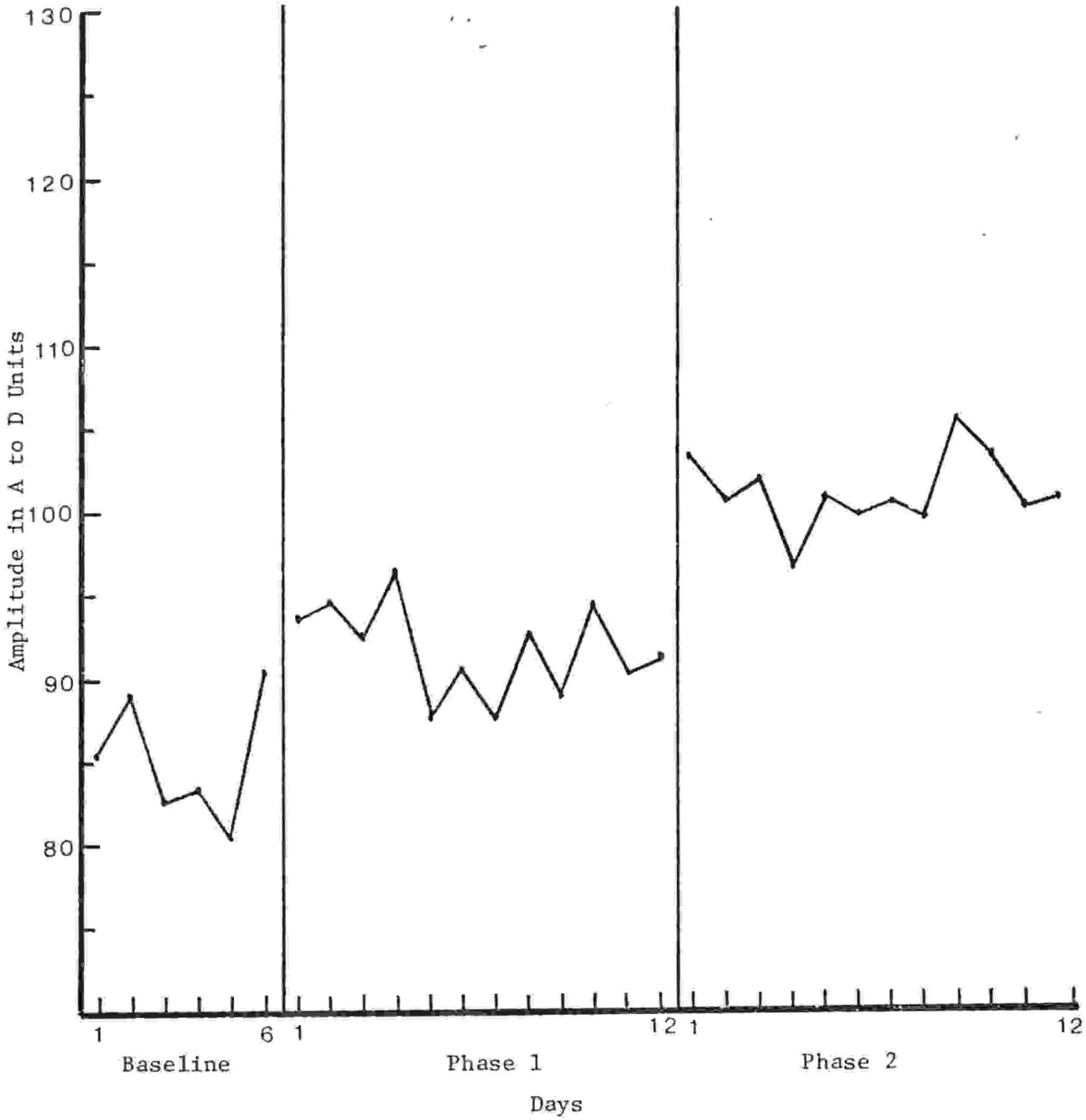
130
120
110
100
90
80
Amplitude in A to D Units
1
6
1
12
1
12
Baseline
Phase 1
Phase 2
Days

Figure 19

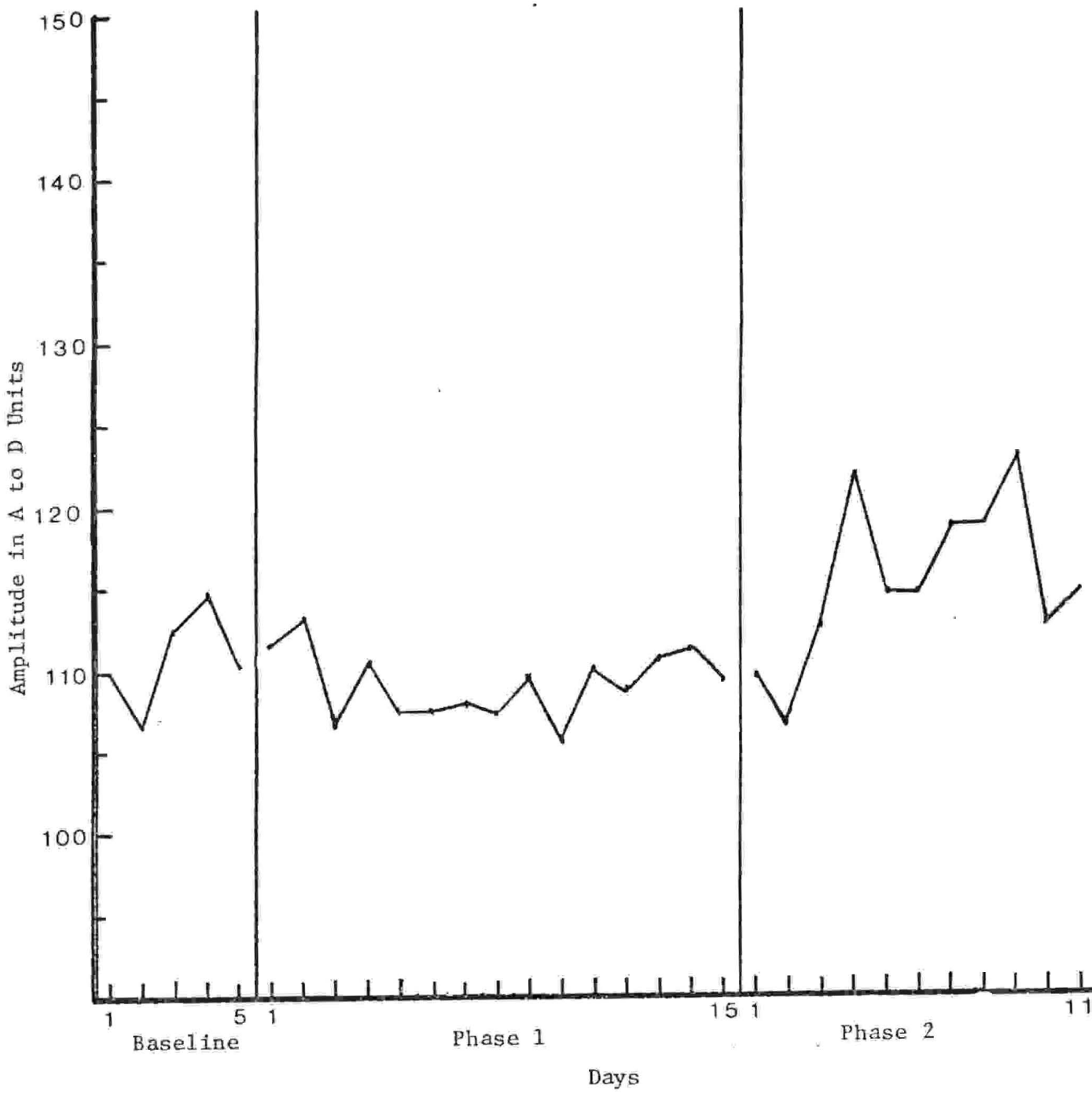
150
140
130
120
110
100
Amplitude in A to D Units
1
5
1
15
1
11
Baseline
Phase 1
Phase 2
Days

Figure 20

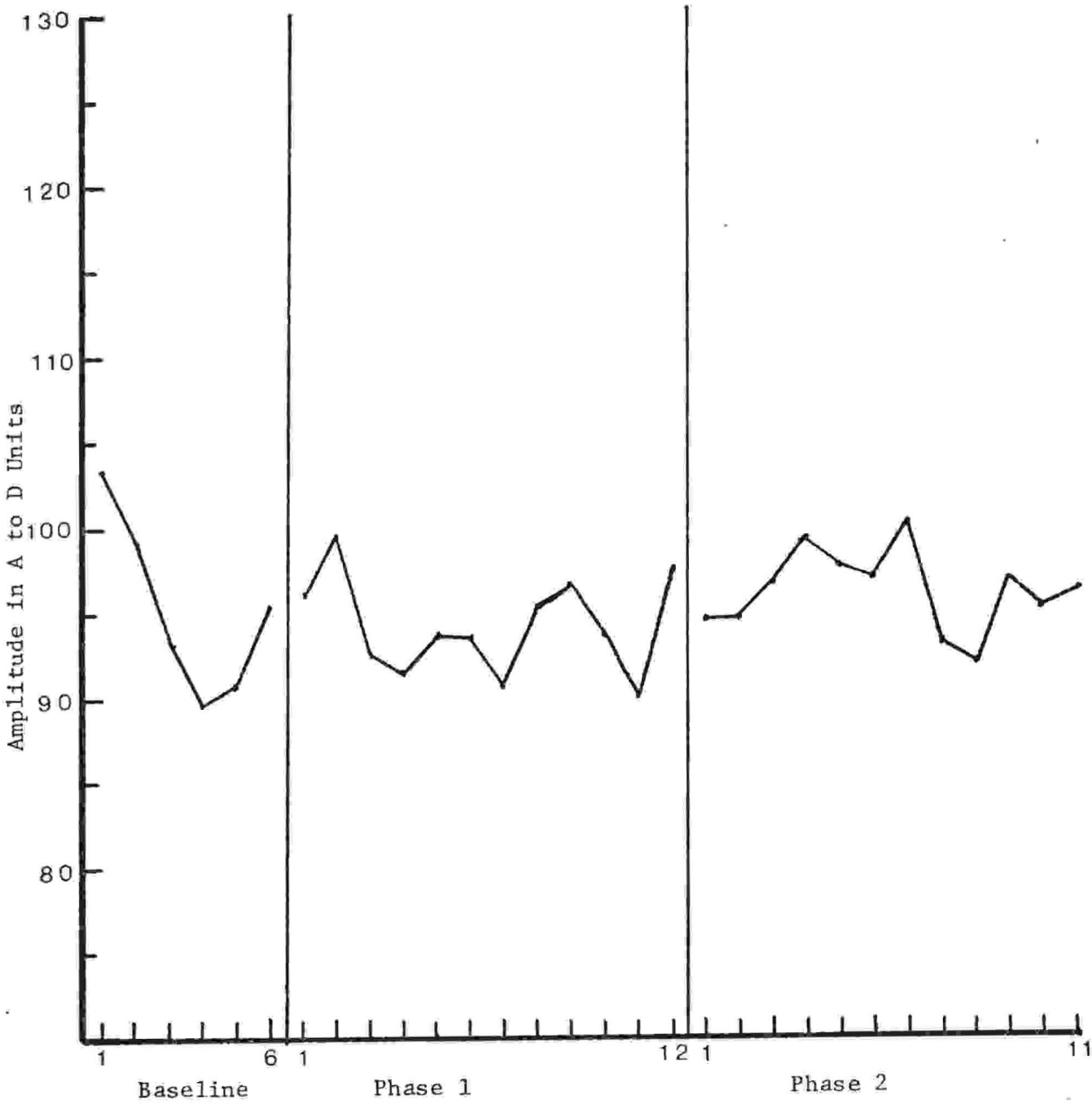
130
120
110
100
90
80
Amplitude in A to D Units
1
6
1
12
1
11
Baseline
Phase 1
Phase 2
Days

Figure 21

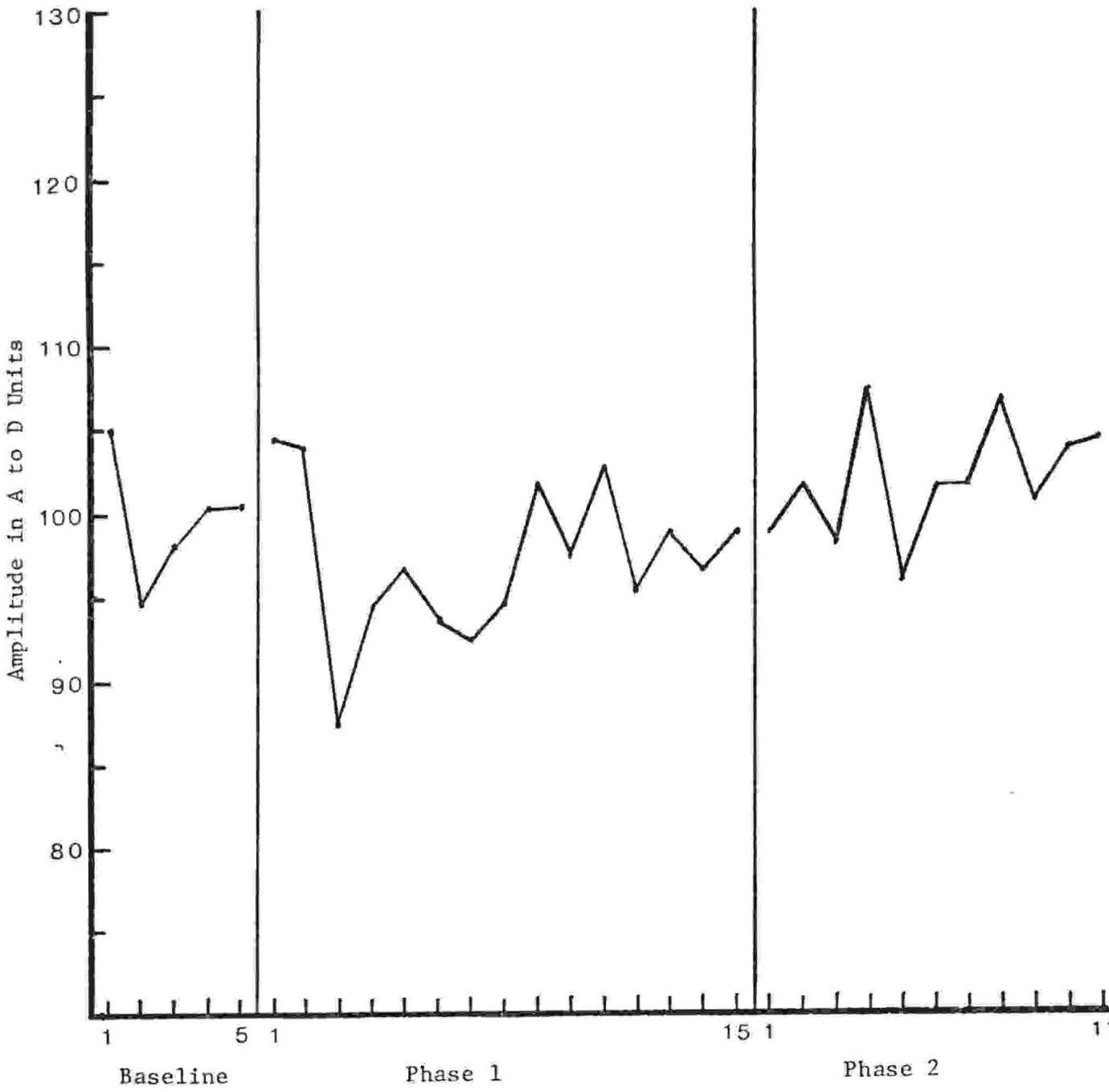
130
120
110
100
90
80
Amplitude in A to D Units
1
5
1
15
1
Baseline
Phase 1
Phase 2
Days

Figure 22

Table 5

Intercorrelations of All Components for Animal 1

Component	Condition	P2 Amplitude	PL Latency	PL Amplitude	P3 Latency	P3 Amplitude
P2 Latency	Baseline	.6556	.7523	.0890	-.5060	.7718
	Phase 1	.1491	.5718	.4881	.2648	.0796
	Phase 2	-.2754	.4101	-.3434	.4053	.6445
P2 Amplitude	Baseline		.3338	-.4080	-.5436	.2858
	Phase 1		.3917	.4195	-.2448	.2928
	Phase 2		-.4684	-.2499	-.2609	.2015
PL Latency	Baseline			.5154	-.0142	.9331
	Phase 1			.5277	.3776	.0751
	Phase 2			-.1536	.3649	.2204
PL Amplitude	Baseline				.6117	.5119
	Phase 1				.0939	.2122
	Phase 2				-.2166	-.5538
P3 Latency	Baseline					-.2263
	Phase 1					-.2237
	Phase 2					.3740

Table 6

Intercorrelations of All Components for Animal 2

Component	Condition	P2 Amplitude	PL Latency	PL Amplitude	P3 Latency	P3 Amplitude
P2 Latency	Baseline	-.0292	-.7780	-.3504	.6803	-.3791
	Phase 1	.6731	.7909	.1554	-.1249	-.0261
	Phase 2	-.1527	.6055	-.2916	.1522	.3172
P2 Amplitude	Baseline		-.4271	.7260	-.2673	-.3471
	Phase 1		.3748	-.0568	-.3814	-.3501
	Phase 2		-.6510	-.4533	-.4730	-.2288
PL Latency	Baseline			-.2571	-.1466	.0916
	Phase 1			.2220	-.1502	.1307
	Phase 2			-.5278	.4678	.4589
PL Amplitude	Baseline				-.5941	.3412
	Phase 1				.1852	.7038
	Phase 2				.5196	.3708
P3 Latency	Baseline					-.6257
	Phase 1					.4397
	Phase 2					-.0815

ship between latency and amplitude for any component in animal 1 or animal 2. Generally, the factors which determine latency account for little of the variance in amplitude. Correlations between the amplitude of one component and the latency of any other are generally of a low order. There are some exceptions to this and these exceptions occur only in animal 1. Relatively high correlations were obtained in Baseline between P2 latency and P3 amplitude (r = +.77, r^2 = .59) and between PL latency and P3 amplitude (r = +.93, r^2 = .86). However, in Phase 1 and Phase 2 these high Baseline correlations became too low to account for a significant portion of the variance.

Since there seem to be no consistent relationships between amplitude and latency of any components, a next logical step would be to investigate the possibility of relationships between the latencies of various components. From Tables 5 and 6 it can be seen that for both animals the latencies of both P2 and P3 are highly independent; correlationsbetween the latencies of these components tend to be of a low magnitude. The only exception to this was for animal 2 in Baseline where the correlation was +.68 which still accounted for only 46% of the variance. This relationship declined to almost zero in both phases of conditioning. A comparison of the changes in P2 latency across conditioning for animal 1 (Figure 13) and animal 2 (Figure 14) with the changes in P3 latency (Figure 15, animal 1; Figure 16, animal 2) again reflect this independence. It can be seen comparing Baseline for animal 2 in Figure 14 with Figure 16 that there is some correspondence between P2 and P3, but that this does not continue across training.

Some dependence is evident between the latency of P2 and the Latency of PL. A comparison of Figure 13 with Figure 9 for animal 1, and Figure

14 with Figure 10 for animal 2 does show some dependence in Baseline. For animal 1, the correlation between PL latency and P2 latency is substantially positive (r = +.75, r^2 = .56) accounting for 56% of the variance in Baseline. For animal 2 the relationship is somewhat stronger (r = .78, r^2 = .61) accounting for 61% of the variance. However, for animal 1 this relationship decreased over Phase 1 (r = +.57, r^2 = .33) and further decreased over Phase 2 (r = +.41, r^2 = .17). In animal 2 this relationship became stronger in Phase 1 (r = +.79, r^2 = .63) than in Baseline and then declined in Phase 2 (r = +.61, r^2 = .37).

Generally then, it can be said that there are no factors which seem to consistently affect the latency across components for both animals. The relationship which did occur in Baseline between P2 and PL declined substantially over training for animal 1 and declined in Phase 2 for animal 2, indicating no consistent dependencies between the latencies of components.

There do not seem to be any common factors affecting amplitude. Almost all correlations are so low so as to account for little of the variance. The only exception was the correlation between P2 amplitude and PL amplitude for animal 2 in Baseline. Here the relationship was quite substantial (r = +.73, r^2 = .53) and accounted for 53% of the variance. However, a comparison of Figure 18 for P2 amplitude and Figure 20 for PL amplitude reveal that this relationship declined during both phases of conditioning. This same correlation in Baseline for animal 1 was much lower (r = -.41, r^2 = .17) and remained low throughout conditioning as a comparison of Figures 17 and 19 show. Figures 21 and 22 for animals 1 and 2 respectively are included to illustrate the course of the amplitude changes for P3 over days in Baseline, Phase 1 and Phase 2.

Table 7 shows the percent of occurrence of P2, PL and P3 for both

Table 7

Mean Percent of Occurrence for Three Components in Three Conditions for Animals 1 and 2

		Animal	
Condition	Component	Rat 1	Rat 2
Baseline	P2	89.84	97.99
	PL	91.54	78.39
	P3	39.72	77.32
Phase 1	P2	87.97	99.02
	PL	87.58	69.99
	P3	40.22	59.54
Phase 2	P2	86.00	97.46
	PL	79.78	77.67
	P3	51.86	69.12

animals over all conditions. The table shows that there are no consistent trends across conditions for both animals. However, the table does show that for both animals the percent of occurrence for PL dropped in Phase 1 compared to Baseline. Apparently then, the task of conditioning was sufficient to decrease the occurrence of the component. For animal 1 the occurrence of PL continued to decrease in Phase 2 in which minimal conditioning took place, and increased in Phase 2 for animal 2 where conditioning occurred. It appears then that as the animal became more proficient at the task PL again occurred more frequently as in Baseline.

There are some similar trends between animals when the correlations between the occurrence of different components are considered. From an inspection of Table 8 it appears that as the latency of PL decreased the relationship between the occurrence of PL and P2 decreased for both animals. For Phase 2, the relationship between P2 and PL still decreased for animal 1, but increased slightly for animal 2, however the magnitude of the correlation was still less than in Baseline. This example of Baseline effects decreasing through training is again seen between the occurrence of P3 and P2 for animal 2 and to a very minor degree in animal 1. In Baseline the predictive power of P2 for P3 is substantial ($\underline{r}$ = +.75, $\underline{r}^2$ = .56) but drops dramatically in Phase 1 ($\underline{r}$ = -.16; $\underline{r}^2$ = .03) and rises slightly in Phase 2 ($\underline{r}$ = -.39; $\underline{r}^2$ = .15) to account for 15% of the variance. This decreasing trend is seen also in animal 1, except that the Baseline relationship accounts for only 11% of the variance ($\underline{r}$ = +.33, $\underline{r}^2$ = .11).

Correlations between the occurrence of P3 and PL showed quite similar effects across conditioning. Here, low Baseline relationships are increased as a result of Phase 1 conditioning, a result that was indicated by the

Table 8

Intercorrelations of Three Components Based on Occurrence

Condition	Component	Animal: Rat 1 PL	Rat 1 P3	Rat 2 PL	Rat 2 P3
Baseline	P2	+.69	+.33	+.70	+.75
Phase 1		+.38	+.31	+.18	-.16
Phase 2		+.03	+.05	-.27	-.39
Baseline	PL		+.35		+.44
Phase 1			+.84		+.62
Phase 2			+.44		+.82

averages in Figures 3 and 4. For animal 1 only 12% of the Baseline variance of PL was accounted for by P3, and for animal 2, 19% of the variance was accounted for. However Phase 1 conditioning of PL increased the percent of accountable variance to 71% for animal 1, and 38% for animal 2. In Phase 1 task acquisition for both animals was very much in evidence. For Phase 2, the percentage of accountable variation dropped to 19% for animal 1, but rose to 67% for animal 2. These results suggest that in the phases where conditioning was clearly evident the ability to predict the occurrence of P3 from the occurrence of PL clearly increased. The only case where this was not true was for animal 1 in Phase 2 where task acquisition was not very much in evidence.

Discussion

Conditioning

The results of this study can be seen as a replication and an extension of earlier work in the Operant Control of Neural Events (Fox and Rudell, 1968, 1970; Rosenfeld and Owen, 1972; Rudell, 1970; Walker, 1974). In the present study it has been shown that it is possible to explicitly condition the latency of the late positive component of the photic evoked potential. Previous work using the OCNE paradigm has been concerned primarily with the conditioning of amplitude. If latency was considered at all it was merely as a product of amplitude conditioning.

That conditioning did occur in this study is shown by the several lines of evidence presented. First, considering only Phase 1, and comparing it to Baseline, it is clear from the graphs that the latency of PL displays a steady downward trend during Phase 1 conditioning for both animals, a trend which showed no sign of asymptote in either animal. The mean changes in the latency of PL in Phase 1 also argue strongly for an effect of conditioning. When one considers that this mean difference between Baseline and Phase 1 reflects a change over Phase 1 of over 12 msec for animal 1 and 25 msec for animal 2 the results are all the more convincing.

The efficacy of conditioning varied to some extent between animals. It is obvious that animal 2 was able to exercise more control over the latency of PL than was animal 1. It might be argued that this results because animal 2 already showed in Baseline a tendency to decrease its latency. However, the change over five days of Baseline was only about 5 msec, whereas the change over the first five days of conditioning was nearly twice that. It is doubtful that a tendency to drop in Baseline is an adequate explanation for superior performance in animal 2. It is

also clear that the occurrence of PL has nothing to do with the ability to decrease latency. Animal 2 showed a less frequently occurring PL than did animal 1 in each phase of conditioning and in Baseline. Also, percentage of criterion responses as a reason for differing efficacy must be ruled out, at least for Phase 1. For both Baseline and Phase 1 both animals had nearly the same percentage of criterion responses; the only difference resulted in Phase 2 where animal 2 generated more criterion responses than animal 1 and showed more effect of conditioning.

The most likely reason for the differences between animals is an increase in the signal-to-noise ratio in animal 2. Such an increase could result from either greater occurrence of PL or from larger PL amplitudes. In this case it is clear that greater occurrence could not explain the difference; animal 1, in fact, generated more PL potentials than did animal 2. However, a comparison of Tables 1 and 2 showed that animal 2 did generate consistently higher PL amplitudes in Baseline, Phase 1 and Phase 2 than did animal 1. This alone could account for the difference in task acquisition; if a signal is stronger then presumably it is more noticible and should lend itself to change more readily.

Corroborating evidence of conditioning is furnished by the plots of averages and DCPs over days. Although the change illustrated by the averages is not great, it is because the average is not a very sensitive measure (Walker and Long, 1976). It can be seen from inspection of the daily DCPs in Figures 5 and 6 and the summated DCPs in Figures 7 and 8 that even a small change in the averages represents a great deal of change in the form of the underlying distribution. The summated DCPs illustrate that in Phase 1 a new mode arises which is in the direction of conditioning, that is, an increase in the mode corresponding to decreased

latency. The decrease of this mode in Phase 2, with a shift to modes corresponding to increased latency is evident for animal 2. However, for animal 1 the only noticeable change in the figure in Phase 2 is that there are fewer potentials in both modes.

These DC s show that there are different modes of responding in those conditions where task acquisition was evident, with the distributions reflecting the direction of conditioning.

Further evidence that conditioning has taken place is furnished by Phase 2 of the study in which the animals were required to increase the latency of PL. The large increase in latency between the last day of Phase 1 and the first day of Phase 2 is further evidence for experimental control of the phenomenon. This increase is graphically illustrated in Figure 9 for animal 1, and Figure 10 for animal 2. Between Phase 1 and Phase 2 were interposed 100 extinction trials, that is, trials in which no reinforcement was given. The effect of the cessation of the reward contingency was that PL, which had been trained to decrease in latency, rose rapidly toward Baseline levels. Apparently, the latency of PL only stays decreased as long as the state producing that decrease is in effect.

Mechanisms of Latency Changes in PL

It might be argued that latency is merely an effect of the task situation, that latency decreases as the animal becomes aroused. Such an arguement would be in keeping with findings of Magoun (1965) and Steinberg (1965). If this is the case, latency would "automatically" decrease as the animal becomes more aroused by attending to the task. Other data in this study provides more evidence against this statement than it does for it. First, the time course of the Phase 1 latency changes is not compatible with the arousal hypothesis. If arousal is to

be used as an explanation of conditioning then the latency of PL would decrease drastically during the first day of conditioning when presumably the animal would be more aroused because the task had not yet been learned. This decrease in early training should then be the shortest latency recorded in training, which was not the case. Second, during extinction the latency jumped back up toward Baseline levels. Again, if latency were simply a reflection of arousal this rapid increase should not have occurred. Most contradictory to the assertion that latency is a simple reflection of arousal is that animal 2 was able to substantially increase the latency of PL, and that animal 1 showed increased latency in the first three days of Phase 2 conditioning. May it then be assumed that the animal became less aroused? This is not likely to be the case because the animals were still in a situation where a task had to be learned. Phase 2 was just as much an arousal situation as was Phase 1.

It might further be argued that the latency of a SW component is a function of stimulus intensity as it seems to be in an action potential (Aidley, 1971), the stronger the stimulus the shorter the latency. If this were true latency would be merely epiphenomenal, that is, a simple by-product of stimulus intensity through the classical visual system. The evidence in this study tends to refute the arguement that latency is simply a reflection of conduction delays in the classical visual system. If a stimulus is delivered at the same intensity during all trials in conditioning, an animal can alter visual input in two ways, either by eye closure or orienting in a specific way during conditioning. However, if the animal simply diminished input by eye closure, this would increase latency so this tactic would only work in Phase 2 conditioning; yet task acquisition was even more in evidence in Phase 1. Also, from the obser-

vations during conditioning, the animals remained in one posture with their eyes open. Therefore, to decrease latency the animals would have to increase visual input, possibly by assuming a posture toward the light. This is an unlikely postulation. If the task to decrease latency were as simple as orienting toward a light source, a minimum latency should have been obtained in Baseline, or at the latest, in the initial days of Phase 1 conditioning. As the results showed however, the latency of PL showed considerable decrease in both animals in the initial days of Phase 1 conditioning as compared to Baseline. This decline continued during Phase 1 with no evidence of asymptotic performance in either animal.

Further evidence that neither eye closure or selective orientation is responsible for the changes seen in conditioning has been presented by Rudell (1970) and Rosenfeld and Owen (1972). Rosenfeld and Owen have reported, that during conditioning of the amplitude of a late component in cats, that eye closure, other than an occassional eye blink, was not in evidence. Thus, eye closure, which would limit the stimulus intensity, could not be evoked as a mediator of the conditioned effect. Rudell ruled out selective orientation to the stimulus as a mediator of the conditioned effect by experimentally manipulating body orientation. He found that animals were still able to significantly alter a specified brain potential parameter regardless of orientation to the stimulus.

Since the present findings and those cited from previous research tend to rule out selective orientation as the explanation of evoked potential conditioning this means that the only other way the visual system might influence conditioning is via efferent control of sensory function. Reporting on the research of others, Morell (1961) has reported that the sensitivity of the retina and central visual pathways varies with

changes in cortical, thalamic, or reticular function. The possibility that any other central structure might be responsible for mediating evoked potential conditioning by influencing the visual pathways was also ruled out by Rudell. Rudell recorded from both a trained site in the visual cortex and a colateral site in the lateral geniculate body, and compared averaged geniculate potentials associated with criterion responses at the trained site to averaged geniculate potentials associated with non-criterion responses at the trained site. He found these geniculate averages to be superimposable, whereas the averages from the visual cortex displayed a difference in the trained segment. Since these differences did not appear in the sorted geniculate averages, Rudell concluded that conditioned changes in visual evoked responses were not the result of changes in the visual pathways. Rosenfeld (1974) further stated that these findings ruled out the possibility of any central structure mediating the effect by influencing the visual pathways.

While it is unlikely that the classical visual system is responsible for the conditioned changes in the evoked response, it is possible that other behavioral factors may mediate the conditioned effect. That conditioning may be mediated by the animals modifying the input producing potentials was suggested by earlier work by Fetz and Finocchio (1971) and Rosenfeld and Fox (1971). Fetz and Finocchio had found that reinforced changes in upper motor neuron cells were accompanied by muscle activity in the muscles previously associated with those cells. Rosenfeld and Fox trained animals to execute limb movements which produced potentials in the contralateral somatosensory cortex. They found that reinforcing animals for modifying these potentials produced changes in the movement which evoked the potentials.

These results led to investigation of other possible mediators of a conditioned brain potential response besides the visual system. Previous observations (Fox and Rudell, 1968, 1970) had not shown any gross motor behavior which could be related to brain potential changes. Rosenfeld and Owen (1972) investigated the possibility that feedback from motor acts might influence the synaptic organization producing the criterion response. However, by presenting a random interstimulus interval and reinforcing a 20 msec criterion segment (which was less than the animal's reaction time) they ruled out discrete motor acts as responsible for the conditioned amplitude changes.

Rosenfeld, Hetzler, Birkel, and Kowatch (1976) further ruled out what they termed trivial mediation in the conditioning of amplitude. By using shocks to the optic chiasm they were able to rule out receptor orientation as a mediator of the conditioned response. By random stimulus presentation they were able to rule out timelocking by the animal to the stimulus, and any slow potential shifts related to movements.

Thus, all previous findings tend to disagree with the contention that conditioning effects are all the result of changes mediated through other systems (visual or muscular). Rosenfeld and Owen (1972) state quite clearly that the subject generates a state which "presets the excitabilities of synapses generating the criterion component before the stimulus is presented." (p. 857). They further conclude, from noticing new widespread relationships in different areas of cortex, that the training procedure leads to new cortical processes. That is, the cortex is in a novel state, a state which does not exist prior to training.

Using the previous research cited above in conjunction with the present study, it must be concluded that changes in latency do not reflect

simple alterations in peripheral visual phenomena (posture toward or away from the light or eye closure). It is also doubtful that the conditioned potential changes reflect any changes in the visual system at all, either peripherally or centrally. This conclusion is best supported by Rudell and Fox (1972) who state that the light flash is largely irrelevant to the conditioning procedure.

Independence of PL from Other Components

It has been demonstrated in this study that it is possible for an animal to manipulate the latency of PL to gain food reinforcement and that this change is not the result of simple control of stimulus intensity by the animal, nor is it the result of arousal brought on by a task situation. Since the internal change of conditioning PL effected a change in the environment, the latency of PL has been demonstrated to be adaptively significant. That is, if a required change in a brain wave is the only event for which an animal may obtain reinforcement, and it does so, then the component must be of adaptive significance.

Attempts to demonstrate the significance of amplitude in the EP have been frequently reported both in correlative studies and in non-correlative (OCNE) studies. Haider, Spong, and Lindsley (1964) reported that reduced attentiveness was accompanied by a corresponding reduction in amplitude of visual evoked potentials in humans. Davis (1964) using an auditory discrimination task, indicated that enhanced attentiveness increased the amplitude of evoked potentials. Many researchers (Galambos, 1958; Galambos, Sheatz, and Vernier, 1956; Hernandez-Peon, 1960) have reported a reduction in amplitude to auditory (click) or visual (flash) stimuli if the stimuli were repeatedly without any consequence. This reduction in amplitude with habituation can be reversed by brain-stem lesions (Hernandez-Peon and

Scherrer, 1956; Sharpless and Jasper, 1956).

OCNE studies (Fox and Rudell, 1969; Rosenfeld, 1975; Rosenfeld and Fox, 1972; and Rudell and Fox, 1971) have indicated that amplitude is of functional significance by virtue of its having been conditioned.

However, the significance of latency of any component in the EP has generally been viewed as suspect. It has been pointed out (Mountcastle, 1969; Burns and Pritchard, 1964) that it is doubtful that latency can be of much use as a measure of intensity, or indeed, of any complex behavior because the organism has no information about the real time of stimulus onset. However, this study has demonstrated that the latency of PL can be made adaptively significant, that animals can effect a change in the environment when these changes are contingent on a modification in the latency of a brain wave component. A problem arises here in that if the latency of PL is not independent of other components, and other parameters of components, then it cannot be, by itself, adaptively significant. The larger question which arises here is "is the evoked potential capable of encoding only one bit of information or many bits?"

The issue of independence of PL from other components may be demonstrated in a variety of ways. If components are dependent on one another then this dependence should be evident in the relationships between occurrence, latency, and amplitude of different components. In any study of neural function one goal would be to be able to predict one aspect of neural function by another. In this basis then, it would be desirable to be able to predict that as one parameter of PL changed with conditioning this same parameter or another parameter of another component should also change to a reliable and predictable degree.

Occurrence. The results showed quite clearly that the correlation between the occurrence of P2 and PL declines over conditioning in both animals. This tends to refute any arguement for dependence between the two components as far as simple occurrence is concerned. If anything, the breakdown of rather large Baseline relationships during conditioning argues for an increasing independence of P2 and PL and indicates that there are contingencies operating in particular situations to break up the synchronized activity seen in Baseline. For animal 1 the correlation between P2 and PL was +.69 in Baseline which decreased to +.38 in Phase 1 to +.03 in Phase 2; for animal 2 the correlations were +.70 in Baseline which decreased to +.18 in Phase 1 and -.27 in Phase 2. This breakdown in Baseline relationships is clearly shown by the percent of variance accounted for in each phase of conditioning. During conditioning the percent of variance accounted for in animal 1 dropped from 48% in Baseline to 14.59% in Phase 1 to .09% in Phase 2. For animal 2 it dropped from 49% in Baseline to 32% in Phase 1 to 27% in Phase 2. Clearly then, the ability to predict the occurrence of P2 from the occurrence of PL declines over conditioning. It appears that the task performs a dissociative function, breaking down preconditioning relationships. This same trend of decreasing relationships over conditioning was also seen between P2 and P3. In both animals, especially animal 2, the components became more independent over conditioning.

As opposed to the independence mentioned above, the relationship between percent of occurrence between P3 and PL definitely increased with conditioning in both animals, with one exception. The exception to this trend was that animal 1 showed a decreased relationship between PL and P3 during Phase 2. This may simply be the result of the minimal task acquisition in Phase 2 for this animal. This conflicting result was

not due to changes in the percent of occurrence of PL in Phase 2 however. The data from Table 6 shows that the occurrence of PL decreased from Baseline through Phase 2, but that the occurrence of P3 increased in the same period. If changes in the percent of occurrence of PL were responsible for the decreased relationship between PL and P3, then it would be expected that the trends in Baseline and Phase 1 would have changed in Phase 2 which was not the case. That the mean percentage of occurrence of PL and P3 does not determine the magnitude of the relationship between PL and P3 is further seen in animal 2. For this animal the percent of occurrence of both PL and P3 decreased from Baseline to Phase 1, and then increased in Phase 2, but the correlations showed continual increase over all conditions as seen in Table 7. What this indicates then is that PL and P3 tend to occur more often together with the advent of conditioning when P3 does occur, but that PL still occurs more often than does P3.

It appears from the data that there is some increasing dependence between PL and P3 in occurrence as conditioning takes place. This dependence, however, is not in evidence between P2 and PL or between P2 and P3. Thus conditioning does seem to increase dependence between late components but not between early and late components. The indication here is that PL and P3 may reflect <u>similar</u> processes which make them occur together, processes which are distinct from those in a primary component. However, from the mean percentage of occurrence, it seems as though PL and P3 are not reflections of the same process because they do occur at different rates. Apparently the process which makes them occur together has nothing to do with the process which makes P3 occur in the first place.

<u>Amplitude</u>. All amplitude relationships were either low or inconsistent across animals. In no case does there seem to be a solid, or even marginal,

case for any dependence between or within components. All correlations listed in Tables 4 and 5 argue for independence between the latency and amplitude within any particular component. What this means then is that it is possible to condition the latency of a component with no dependent change in the amplitude. Independence between amplitudes of PL, P3, and P2 also seems to be the rule in Baseline, Phase1, and Phase 2. What should especially be noted here is that the dependence in occurrence seen between PL and P3 is not in evidence when amplitude is considered; PL amplitude is independent of P3 amplitude.

Latency. From all the data in this study the latencies of P2, PL, and P3 appear to be independent. As seen in the relationships of the occurrence of P2 with PL and P2 with P3, the general trend in the relationships of the latencies of components is for decreased dependence (lower correlations) with conditioning, which indicates that there are contingencies operating which break down the synchronized activity seen in Baseline.

This trend is seen quite clearly between the latency of P2 and P3 as shown in Tables 4 and 5. For both these animals substantial Baseline correlations decreased during Phase 1 and Phase 2. It should be pointed out here that animal 1 showed a change from a negative correlation in Baseline to a positive correlation in Phase 2. However, this does not argue for increased dependence of components; as far as dependence is concerned, the direction of the relationship makes no difference. This dissociation of latencies between components is seen to a lesser degree between the latency of P2 and PL. Correlations between the latency of PL and P3 did not show the consistent decrease of Baseline relationships over conditioning, but correlations were so low so as to account for only a small portion of the variance.

None of these relationships show increased ability to predict the latency of one component from changes in another, indeed, the reverse is to a large extent true. Thus, conditioning the latency of PL does not increase the dependence between the latencies of other components. This independence is also seen when the latency of any component is compared to the amplitude of any other component. Here too, it must be stated that by knowing the latency of any component it is not possible to predict the amplitude of any other component.

It has been shown in this study that the specified changes in the latency of PL are independent of other parameters of EP components; that as the latency of PL is changed other parameters of other components do not change in a reliable or predictable fashion. Both the amplitude and latency of PL are independent of the latency and amplitude of P2 and P3, and P2 and P3 are independent of one another. It has also been shown that P2 and P3 and P2 and PL are independent in terms of occurrence, but that PL and P3 do show some tendency to increase the relationship with conditioning.

The results of this study tend to agree with other studies in which independence of processes has been demonstrated. Fox and Rudell (1969) state that the late potential in the cat photic EP represents different independent events from the primary components. In a later study (Rudell and Fox, 1972) they again argue for independence of components when they found that it was possible to condition amplitude in primary components with no dependent change in late components. They conclude that the EP is "...not a unitary message, but encodes a number of separate behavioral states." (p.560). The independence in latency and amplitude among components indicates that PL, by itself, is adaptively significant. Thus, specified changes in the latency of PL are unique to only PL. More importantly,

it has been shown that the amplitude of PL is independent of changes in the latency of PL. Thus, not only has PL been shown to be adaptively significant, the latency of PL has been shown to be such also.

One of the more important aspects of the conditioning of PL latency are the changes seen in those relationships which were strong in Baseline but which were disrupted by conditioning. Most notible among these correlations are in animal 2 between the latencies of PL and P2 and between the amplitudes of PL and P2, and in animal 1 between P2 latency and PL latency, and in occurrence between P2 and PL and P2 and P3 for both animals. It is these types of dramatic changes from preconditioning relationships to conditioning relationships where evidence is most obvious for changes in conditioning which tend to establish new relationships of the component PL with other components. If relationships which are established prior to conditioning are disrupted by conditioning then this argues that whatever is responsible for this relationship may be causal but not necessary. Hence, components which occur reliably in a certain relationship need not stay in that relationship so events which mean one thing at one state of the cortex (i.e. pretraining) may mean something else in another state (after training). While latency may not be significant to the animal before training it may be conditioned to become so. Thus, this study tends to agree with previous research (Fox, 1970; Rosenfeld, 1974; Rosenfeld and Fox, 1972; and Rudell and Fox, 1970, 1972) which argues that the Operant Control of Neural Events establishes a novel state of cortex. That is, that the training procedure leads to new cortical processes which are not present in the unconditioned animal.

What these new cortical processes are can only be hinted at from the results of this study. It has been seen that specified changes in the

latency of PL are unique to that parameter of that component, but it is also apparent that there are both latency and amplitude changes in other components as a result of conditioning. Thus, there are widespread changes occurring in those generators responsible for the production of the EP, and those changes are independent of one another. No conclusive statement can be made here about how much of the cortex was involved in these changes, but there is one fact which may clarify the issue. The fact that the electrode tips were only 1-1/2mm apart definitely restricts the amount of volume conducted activity likely to be recorded. This, however, only indicates that a relatively specific population of cells were being recorded from, not how many cells were actually changed by the procedure. It does indicate though, that whatever the extent of the change, it can be recorded from a relatively small tissue volume.

The summated DCPs shown in Figures 7 and 8 also may give a clue to the nature of the processes. Since synaptic and dendritic events are largely responsible for the genesis of the EP (Landau, 1967; Purpura, 1967) the emergent bimodality of animal 1 and the trimodality of animal 2 may be a reflection of new synaptic events with the advent of conditioning. If this is the case, it is clear that the new "set" of events can operate in conjunction with the old "set". Thus, the different modes which reflect different latency classes of PL may also reflect different sets of synaptic processes which can operate conjointly.

Another finding of interest in regard to the cortical processes involves component P2. It was pointed out previously in this study that the early components are thought to be a reflection of sensory mechanisms. If this is indeed the case then the relatively high Baseline correlations between the latency of PL and the latency of P2 argue for the fact that

PL in Baseline may be sensory linked. The relationship of the latencies of these components showed some of the largest disruptions during conditioning, which indicates a dissociation between PL and the sensory processes. Thus, it again appears that the conditioning of PL may lead to new cortical processes which are not sensory linked.

All of the above mentioned points bear on the question of coding by the evoked potential. The independence seen among components in terms of amplitude, latency, and to a lesser extent, occurrence, indicates that the evoked potential is not a single message, but has the potential to encode a number of behavioral states. The further finding that within any one component latency is independent of amplitude further indicates that each parameter of a component is capable of becoming an encoder of a behavioral state. Thus, it appears as though the EP is capable of encoding as many bits of information as there are separate parameters. However, the potential of the EP to encode behavior does not mean that components or their parameters are related to any unique behavioral state (Fox, 1971; Fox and Rudell, 1970). Fox has explained that it is more likely that any behavioral state is related to combinations of parameters at a given state of the cortex at a particular cortical location. This study does not present any evidence, nor did it intend to, that any change in a brain wave parameter is related to a particular molar state.

Another aspect of this study is the use of averaged evoked potentials as an indicator of the occurrence of conditioning, and the process involved in conditioning. It can be seen from a comparison of the DCPs in Figures 5 and 6 with the AEPs in Figures 3 and 4 that averaged waves obscure many of the processes occurring in a particular component. The AEPs consistently showed that PL occurred as a single potential from Baseline through Phase

2 for both animals. However, the DCPs showed that PL was not a potential which occurred at one latency but at two, and sometimes three, different latency zones within what was defined as the PL range. The bimodality which was obvious in the DCPs was apparent in the AEPs only if information from the DCPs indicated what to look for. The variability seen over days in the DCPs showed that animals may approach the same problem different ways yet arrive at the same solution, or at least, the same percentage of reinforcement. Averaged evoked potentials, like any mathematical averaging procedure, present the reliably occurring information while obscuring the variability which may be the aspect of most interest in a study of brain function. To believe that the infinite variability of response of brain cells can be described and understood by using the product of a procedure which cancels out this variability is certainly illogical and suspect as a neurophysiological tool.

This study has shown that it is possible to operantly condition the latency of PL, that this conditioning creates a situation in which pre-training processes are disrupted - that potential parameters previously related become unrelated. These findings indicate not only that conditioning creates a new state in some volume of cortical tissue, but also that components are not locked into one meaning or use. The significance of any component is variable and dependent on a particular state imposed at a certain time. Fox (1970) has put this rather cogently when he said of the "traditional" view of components and their sequence that this indicates "...the severly limited conditions under which evoked potentials have been studied." (p. 254)

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Figure Captions

Figure 1. Traces 1 - 6: Selected single sweeps from Baseline for animal 1. Line at 100 msec denotes delivery of the stimulus. Trace 7: AEP of 300 single sweeps. Time calibration: 512 msec; negative up.

Figure 2. Traces 1 - 6: Selected single sweeps from Baseline for animal 2. Line at 100 msec denotes delivery of the stimulus. Trace 7: AEP of 300 single sweeps. Time calibration: 512 msec; negative up.

Figure 3. Sequentially presented daily AEPs for animal 1. Traces 1 - 6 are from Baseline, traces 7 - 18 are from Phase 1, and traces 19 - 30 are from Phase 2. Each AEP in Phases 1 and 2 represents 300 single sweeps; line at 100 msec indicates demarcation between prestimulus and poststimulus times. Time calibration: 512 msec; negative up.

Figure 4. Sequentially presented daily AEPs for animal 2. Traces 1 - 5 are from Baseline, traces 6 - 20 are from Phase 1, and traces 21 - 31 are from Phase 2. Each AEP in Phases 1 and 2 represents 300 single sweeps; line at 100 msec indicates demarcation between prestimulus and poststimulus times. Time calibration: 512 msec; negative up.

Figure 5. Sequentially presented daily DCPs for animal 1. Each trace corresponds to the same numbered trace in Figure 3. Time calibration: 512 msec.

Figure 6. Sequentially presented daily DCPs for animal 2. Each trace corresponds to the same numbered trace in Figure 4. Time calibration: 512 msec.

VITA

Surname: Westerberg Given Names: Verner Stuart

Place of Birth: Washington, D.C., U.S.A. Date of Birth: October 7, 1948

Educational Institutions Attented, with Dates of Entering and Leaving

Miami University, Oxford, Ohio U.S.A.	1966	to	1968
University of Colorado, Boulder, Colorado, U.S.A.	1968	to	1970
California State University, Los Angeles, U.S.A.	1973	to	1974
University of Victoria, Victoria, B.C.	1974	to	1977

Degrees, Diplomas, Etc.,Awarded, with Dates and Names of Institutions:

B. A.	1970	University of Colorado, Boulder

Honors and Awards:

Publications:

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Title of Thesis

OPERANT CONDITIONING OF THE LATENCY OF A VISUAL EVOKED POTENTIAL: INDEPENDENCE OF EARLY AND LATE COMPONENTS

Author

Verner Stuart Westerberg
Name

August 8th, 1977
Date