CONDITIONABILITY OF A LATE POSITIVE COMPONENT OF THE VISUAL EVOKED RESPONSE IN CHRONICALLY IMPLANTED HOODED RATS

by

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ABSTRACT

A contingent reinforcement paradigm was employed to alter the latency of the late positive potential (PL) in the visual evoked potential of the rat. Three groups of adult male rats were prepared with indwelling electrode implants. One group was trained to decrease latency, a second group was trained to increase latency, and a third group was employed as a control. It was found that animals in the decreased latency group showed some mean latency decrease but only two changes were statistically significant. In the increased latency group, two animals produced the appropriate shift but only one animal produced a statistically significant latency shift. In the control group, no reliable latency changes occurred. Examination of selected cases support the proposition that latency of PL as a neural event is an adaptively significant part of the neural language.

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CHAPTER 1

INTRODUCTION

There is a growing body of knowledge concerning CNS information processing. In recent years, researchers studying the neurophysiological substrate of behaviour have shown that many aspects of brain potentials are conditionable. These aspects include rate of unit discharge in hypothalamus (Olds, 1965), and in cortex (Fetz and Finocchio, 1971); hippocampal theta activity (Black, Young and Batenchuk, 1970); human scalp recorded potentials (Rosenfeld, Rudell and Fox, 1969); the sensory motor rhythm (Chase and Harper, 1971); components of epicortical EPs (Fox and Rudell, 1968, 1970; Rosenfeld and Fox, 1971; Walker and Shaver, 1972, a, b; Walker, 1974; Walker and Long, 1976; Rosenfeld, Rudell and Fox, 1970) and alpha rhythm (Kamiya, 1969; Martindale and Hines, 1975; Plotkin and Cohen, 1976; Prewitt and Adams, 1976; Woodruff, 1975); lateral geniculate spikes (Linnstaedler and Perachio, 1974); visual cortical unit activity (Shinkman, Bruce, and Pfingst, 1974); movement-evoked cortical potentials (Rosenfeld and Fox, 1971; fast (Walker, 1974); and slow (Fox and Rudell, 1968, 1970; Rosenfeld, Hetzler, Birkel, and Kowatch, 1975). These studies are concerned with the conditionability of neural events, that is the occurrence or non-occurrence of EP components as well as

their frequency and amplitude. In this study the direction and conditionability of a late (200 msec latency) potential (PL) in the rat will be discussed.

The commonality displayed by the previously mentioned studies rests in the fact that in each case the bioelectric response could be controlled by contingent reinforcement. In the past, physiological psychologists have attempted to determine the neural correlates of some adaptive behaviours (Morrel, 1961; John, 1961; Thompson, Patterson and Tyler, 1972).

The major difficulty encountered in correlative studies lies in determining the aspects of bioelectric activity that co-vary with behaviour. In an attempt to emphasize the problems of the correlative paradigm and to resolve the problem of what aspects of the neural signal should be measured, Fox delineated some of the sources of instability involved in the correlative approach and proposed a new strategy for determining the important parameters of neural function. Fox and Rudell (1968) suggest that the significance of any bioelectric event can be assessed by determining whether or not that event can be manipulated under reinforcement control. If the bioelectrical configuration can be manipulated for adaptive purposes, it must be an adaptively significant neural process and, as such, constitute a part of the neural language.

Fox asserts that the foregoing is a necessary approach in light of the following: first, measurement of

behaviours are not precisely specified. Consequently, they cannot be adequately correlated to neural events since deviations in behaviour must relate to changes in neural events. The definition of behaviour employed may only account for one or a few end point responses and not the infinitely complex set of responses that occur and possibly were conditioned simultaneously. Second, reliable brain correlates of behaviour are almost impossible to obtain due to subject variability in response acquisition sequence when acquiring gross behaviour. Third, the exact stimulus conditions are not adequately known, such that a stimulus defined as neutral to a given behaviour paradigm may not be neutral if the subject has had previous experience with that stimulus under different environmental conditions. Fourth, our knowledge regarding neural representation of complex behaviours does not allow us to make assumptions regarding unique representation of behaviour at any particular brain locus. Fifth, behaviour may be both spatially and sequentially represented in the brain, and learning may be accompanied by shifting of bioelectrical process during the acquisition phase. Sixth, correlation of a discrete bioelectrical event with a motor behaviour may result in gross error as behaviours of a gross nature are in all likelihood multiply determined. Seventh, it is not feasible to make meaningful comparisons of behavioural and bioelectrical measurements on a common time base with a common zero point, since the time base for the bioelectrical event and behav-

iour may vary greatly. Eighth, the parameter specified for correlation with motor behaviour is necessarily arbitrary and very possibly unrelated to the major neural response system under conditioning control by the animal.

These factors have led to an alternate approach to the study of behaviourally significant bioelectrical events. Instead of the bioelectrical signal being made a dependent variable, it is used as a criterion for reinforcement, the independent variable. This technique appears to provide a logical method since there is some certainty in our knowledge of the bioelectrical signal. For example, in the case of macropotentials, we can measure parameters such as rise time, amplitude, phase and latency. These events can be related to activity in the underlying neuronal population, permitting systematic study of bioelectrical response parameters that potentially relate to or encode behaviour.

The evoked potential (EP) is a macropotential, representing a cell population response. This response is produced by a sudden peripheral stimulus creating a series of potential changes that are time-locked to the stimulus. A photic EP, recorded from the surface of the visual cortex, is characterized by two major components: primary and secondary.

In the cortex of the rat, the primary component consists of a fast positive polarity shift (P1) occurring at approximately 20 msec post stimulus time (PST), followed by a large negative potential (N1) that peaks at 30-35 msec

PST, and a large positive potential (P2) that peaks at 50-60 msec PST. There is very little latency variability observed in P1, N1, and P2. The probability of occurrence of these primary components is extremely high since they are the direct result of the afferent volley from peripheral sensory pathways.

The secondary component consists of a slow negative potential (NS) terminating in a late positive potential (PL). The latency of PL is considerably more variable than P1, N1, P2, ranging from 197 to 248 msec (Walker, 1974). The neural activity underlying these later waves results from activity outside of the primary afferent pathway. The after discharge (AD) or late positive component (PL) is correlated with a low level of activity in the brain stem activating system (Kimura, 1962). Kimura pointed out that AD only occurred when the animal was accustomed to the experimental situation and after short term habituation to the light stimulus. This is supported by Buchwald (1961) and Heuser (1961) who describe an inhibitory system called the "Caudate-loop" involving the caudate-nucleus ventral anterior nucleus of the thalamus which effectively counteracts the effects of the reticular activating system. Landau (1967) substantiates this position. He found that high-frequency stimulation of the medial thalamus potentiates the primary response and eliminates the later AD.

Most of the recordable parameters of brain activity can be related to activity in the underlying neuron popula-

tion. Gross cortical evoked potentials are believed to be the result of 1) graded dentritic activity with a time constant, 2) all or non 1 msec soma axon hillock spikes, 3) 0.5 msec axom spikes which are probably of least significance in the integrated field potential (Landau, 1967).

A high correlation between the probability of single-cell discharge and the EP amplitude has been demonstrated (Fox and O'Brien, 1965; Johns and Morgades, 1969). High correlations have been shown between single-unit activity and rise time (Verzeano, 1970) and between EEG and single unit discharge (Fox and Norman, 1968; Krekule and Walker, 1971; Frost, 1966). In light of the above, it is reasonable to consider the EP as reflecting compound activity in neurons.

An early interpretation of slow waves was that they were due to action potentials (Li and Jasper, 1953; Bishop, 1952). Subsequently, it became obvious that highly synchronous activity, both localized and unequal at different regions, and slower than that of an axon spike, must be involved for cortical waves; the inference being that extracellular recorded potentials are a result of extracellular current flow from an internally negative region toward an internally less negative region. Post synaptic potentials (PSPs) make the major contributions to the inequalities in membrane potentials. For example, an excitatory dendritic synapse near the upper layer of cortex results in the inward movement of sodium ions from the extracellular fluid. This

move of positive ions gives rise to a surface negative wave (recorded above the area) since the area is negative relative to the inactive reference electrode. Conversely, synaptic activity in deeper layers of the cortex will cause the extracellular current (ions) to flow downward resulting in a surface positive recorded wave. This means that surface-recorded activity is a result of the spatial and temporal summation of the synaptic potentials. It is the first derivative of the original function, i.e. a plot of the rate, of the rate of change of surface negativity. In conventional recording, if the area from which the recording is taken has a negativity shift in respect to the reference electrode, an upward oscilloscope deflection is displayed. Conversely, a downward deflection is recorded when a negative change begins to decrease.

Fox has demonstrated that SW is representative of the momentary excitability of the area from which recordings are obtained by showing the correlation between single-cell firing to slow wave (SW) activity (EP, and EEG). This excitability is reflected in the probability of occurrence of action potentials. A high correlation between spike probability and SW indicates that a particular neuron may not fire reliably to a given stimulus. Thus the single unit cannot provide much information (Gerstein, 1969).

John (1972) suggests that the processing of sensory information and other complex integrative functions must be mediated by simultaneous activity in large groups of neurons,

organized into functional units with boundaries and relationships which vary from task to task and from time to time. He proposes that the critical event in learning is the development of a common mode of activity, a temporal pattern that is coherent across various effected regions and specific for that stimulus complex. In this model, information is read out stochastically, the firing of particular cells is not relevant in reading out information.

In light of the above, the significance of any recordable bioelectric configuration could be assessed by determining whether the probability of that configuration can be manipulated for adaptive purposes. It is this reasoning that Fox and Rudell (1968) have proposed to determine the important parameters of neural functioning. They imply that if any bioelectric event can be manipulated, it must be an adaptively significant neural process and as such, constitute a part of the neural language.

In reference to the previously discussed problem of what should be measured, the above suggests that brain activity should be studied in terms of the demonstrated neural language. Specifically, this means we should measure brain activity that can be manipulated for adaptive purposes, with the intent of understanding general neural processes such as coding, storage, and retrieval rather than specific brain behavioural correlates. The fact that brain phenomena, mentioned in the opening paragraph, could be controlled by contingent reinforcement indicates that they are part of

neural language in the manner previously discussed, and as such, some have been employed as indices of brain function (Walker, 1975, 1976). The rationale is that of all possible parameters of bioelectric responses, those that are conditionable are the only ones known to be adaptively important (Walker, 1974). In conjunction with these terms of reference, the components of the EP demonstrated to be important as neural codes are amplitude (Fox, 1970), temporal position and occurrence of particular components (Walker, 1974). However, latency of any given component is not believed to represent a code in itself (Mountcastle, 1969; Burns and Pritchard, 1964; Fox, 1970; Walker, 1974). This statement may be true since it is obvious that in order for latency to exist, something else must exist to which latency is being referred. On the other hand, it appears reasonable to believe that the latency of bioelectric events in reference to each other may constitute a part of neural language. Walker, 1974, discusses neural events as Type I and Type II. Type I events are those occurring with each stimulus and in an ordered response sequence. The Type I events occur with greater variability in both amplitude and latency (Miller, 1973).

Walker (1974) suggests that the reliable Type I events are not suitable to encode the necessarily variable perceptual phenomenon. This belief is supported by the fact that these events can be observed after 98 hours of continuous presentation of a meaningless stimulus (Hertz, Beer,

Sheatz, and Galambos, 1960). However, the Type I components may serve as a carrier function on which elements of neural language are superimposed. In this regard they may contribute significantly to the neural code, or represent a code in itself. Regardless of the influence latency has on any given component, if it can be shown that a neural event (PL) can be brought under stimulus control, such that it occurs at or before a previously prescribed latency, we will have demonstrated that it, too, is a significant part of neural language, thus demonstrating that Type I events can be considered part of the neural language, contributing to our knowledge of aspects of brain activity.

It is important to note that it is not directly an EP component that is being conditioned but the time of occurrence of an underlying neural event. More specifically, the latency of the PL component is a signal of the conditioned activity but not the activity that is undergoing change. Therefore, we cannot speak of these observable events as behaviours but should, more appropriately, speak of them as functions, or resultant neural functions, codes.

In a physiological sense, if PL latency can be manipulated through the use of conditioning techniques, it simply indicates that somehow the processes involved in initiating that component have been changed. This is to say that following the stimulus presentation, the temporal firing pattern of a great number of cortical cells have

been altered to take place either earlier or later than usual. How this is accomplished by the animal is unknown and cannot be determined.

In this study, a conditioning technique will be employed in an attempt to alter the latency of PL. If the animals utilized are able to accomplish a latency shift in PL, we can accept the fact that this neural event is part of neural language which provides information to the animal.

CHAPTER 2

METHODS

Surgical Procedures

Twelve male hooded rats, six to eight months old and weighing 400-500 gms, were surgically prepared with indwelling electrode implants. Each rat was intraperitoneally administered 50 mg/kg sodium pentobarbitol. The animal was secured in a sterotaxic instrument when thumb pressure to the hind foot pad no longer elicited leg flexion. Sterotaxic plane and co-ordinates as described by Skinner, 1971, were employed for location of electrode placements. The head was shaved and the scalp cleansed with merthiolate. A saggital incision extending from the sinal area posterior, approximately 2 cm past bregma, exposed the calvarium. Wound margins and the skull were cleansed with a solution of 500 units/cc Bacitracin. The wound margin was clamped in four areas, a clamp on either side of the wound approximately one cm from either end of the incision, to retract the skin and to assure the skull exposure. Air forced through a 20 cc syringe was utilized to completely dry the skull. Electrode placement holes were trephined with a No. 3 rose burr, in a dental drill. Bregma and Midline sutures and the top of the cortex were used as

references for locating visual, motor, and frontal bone areas. Burr holes for right and left visual cortex were located at Bregma; L+3.0 mm; for frontal bone reference, B-16 mm. Stainless steel screws (080) 4 mm long and attached to mini jacks were screwed two turns into the calvarium at all locations. The mini jacks were then inserted into a plastic electrode cap. Both the electrode assembly and electrode receptacle were then attached to the skull with cranial plastic. The wound was closed with 4.0 mm wound clips. All animals were allowed ten days recovery prior to experimental use.

Experimental Environment

All recording was done in a grounded refrigerator shell which served as a shielded cage. The refrigerator had been adapted for this purpose in the following manner: a six inch diameter hole had been cut in the top over which a Grass PS - 2 photo stimulator head had been placed; a four inch hole had been cut in one side to accomodate a ventilation fan; and two holes of one-half inch diameter were drilled, one through which a feeder tube was inserted and the other through which recording leads and photo cell unit wires were taken.

The animals were placed in the refrigerator during experimental sessions, unrestrained within a 16 inch square plexiglass box with grid floor. The box was constructed such that a narrow passage 3 inches wide and 6 inches deep was located on the feeder side. In this passageway, just

before the feeder tray and directly beneath the head of the photo stimulator, two photo cell units were placed. The roof of the box supported a five channel swivel commutator to which a five conductor shielded cable was attached.

Recording

Recording electrodes were connected through the cable and commutator to a Tektronix amplifier. The amplifier was set at a gain of 5,000 and filter set at 10 CPS low pass and 100 CPS high pass. The output of the amplifier was monitored on a Tektronix Type 502 A oscilloscope and connected in parallel to the A/D input of a PDP-8e computer with input and output devices, including a teletype, relay drivers, and analog to digital (A/D) converters. Presentation of stimuli, response detection, reinforcement and recording time were under the control of a machine language program (OCNE 8E, D. Shaver, 1972). The program and apparatus operated in the following manner. If the animal (who had freedom of movement) walked into the trial area and interrupted a photo cell beam, the photo stimulus (high intensity light flash) was triggered. Analog inputs following the light flash were sampled for 512 msec, digitized (into 256 points) and stored in the core memory of the PDP-8e computer. Following this, a portion of the digital wave form was examined by a programmed computer stencil designed to detect PL. Within a window between Y and Y + Z msec after the stimulus, the computer examined to determine if

any two points separated by 8 msec differed by a particular amplitude criterion (C). If a criterion amplitude difference was found exceeding C units, the EP was labelled as a criterion EP. In the event of the detection of a CEP, the computer was programmed to close a relay in the reinforcing circuit and activate the feeder to administer one food pellet, simultaneously activating a 1000 Hz tone in the shielded cage. The teletype would print a "+" and the buffered EP was averaged with other CEPs located in computer core from previous trials. If the parameters of the stencil were not satisfied, the EP was labelled as a non-criterion EP (NCEP), which caused the "-" key on the teletype to be activated and the EP averaged with other NCEPs in core reserved for NCEPs. An average of all EPs, CEPs, NCEPs, and standard deviation waves was computed. In addition, the times of occurrence of fits to the reinforcement stencil were put into a time histogram called a Distribution of Criterion Potentials (DCP).

The conditioning procedure was continued for a fixed number of trials. Program variables such as interstimulus interval, number of trials, and delay of reinforcement could all be entered through the teletype keyboard.

Experimental Design

All animals were placed on 24 hour food deprivation five days prior to the experimental manipulation. This deprivation schedule was maintained throughout the study.

Each animal received 3 food pellets daily following the training session in the apparatus.

On the sixth day each animal was placed in the apparatus as described. On this day and the following day each animal received 100% reinforcement. The purpose of the procedure was to obtain baseline data regarding the amplitude and latency range of the late positive EP component. This date was then employed in determining the stencil that would be used for conditioning. On all following days each animal was manipulated according to the stencil designed from baseline data.

Five animals were randomly chosen to increase the latency of PL; four animals were chosen to decrease the latency of this component; and three animals (control) were reinforced for producing PL of a previously determined amplitude. Computer stencils employed in this study were constructed such that an animal required to increase PL latency would be reinforced for a latency range commencing at the 75th percentile of the final day of the baseline distribution. Similarly, animals conditioned to decrease PL latency would have stencils constructed that would reinforce a PL component occurring prior to or during the time period represented by the 25th percentile of the DCP of the final day of baseline. One animal was subjected to non-reinforcement (extinction) following training.

Originally, it was intended to have four animals in each group. However, one animal in the control group died

prior to beginning data collection and an extra animal was added to the increase latency group due to difficulties encountered in conditioning these animals.

Data Analysis

On-line data analysis carried out by the computer consisted of sorting EPs into CEPs and NCEPs; averaging all EPs, averaging CEPs and NCEPs; determining the standard deviation of each of the above wave categories; and compiling histograms of all wave components that met the amplitude criterion in each of the above categories. All nine pieces of data obtained on-line in the PDP-8e computer were stored in core memory to be recalled later. This data was transferred to paper tape. Following the completion of the prescribed number of data trials on each day, the percentage of CEPs was calculated and printed by teletype from computer core.

Off-line analysis programs consisted of processing data from the digital tape. A plotting program was employed for graphing data DCPs and AEPs. This plot produced a series of graphs representing each day's data in chronological order. Finally, correlated t tests were obtained on each animal's data comparing the median latency of PL on the first three days to the last three days of conditioning.

PL latency was obtained in the following manner. First, the range in which the PL occurred on the DCP computer print out was determined. Second, all the PL compo-

nents that occurred during that time interval were totalled. Third, the latency at which one-half of the total number counted occurred was considered to be the median latency of the PL component. This latency was employed in the correlated *t* tests.

There was some difficulty in determining the range employed in calculating the median latency since some animals did not have a clearly defined range of CPs. However, in all cases, counts of all CPs within the apparent time frame were made. In cases of unclear latency range for the DCPs, the count was made within the time frame to a point in time where a zero count occurred.

CHAPTER 3

RESULTS

Data was obtained from three groups of animals experimentally manipulated as described in the procedure section. The data represents monopolar photic evoked potentials (EPs) recorded from area 17 of the Hooded Rat in response to a brief flash of light. Animals 1, 2, 3, and 4 comprised the first group. This group was conditioned to decrease latency of the PL component in the photic EP. The second group, comprised of animals 5, 6, 7, 8 and 9, were reinforced to increase the latency of PL. Finally, the third group, comprised of animals 10, 11, and 12, received reinforcement not contingent upon the latency of PL. This group was employed as an experimental control procedure.

The correlated t test was employed as a statistical verification of latency shift in the PL component of the EP. Table I provides the t value, df, and p value for differences in latency of PL between the first and last three days of training for each animal in the study.

Figure 1 depicts sequentially the DCPs of fifteen consecutive recording sessions obtained from Animal 1. The first two DCPs represent baseline recording data during which time the animal received 100% reinforcement. Graphs 3 to 10 inclusive represent ten consecutive conditioning

sessions. Graphs 13 to 15 illustrate the result obtained on the 60th to 62nd day of extinction.

Figure 1, if read from top to bottom, illustrates that the latency of PL was shifted by the animal to increase the probability of reinforcement. This observed shift is significant at $p<.01$ ($t = 7.14$, 3df). Figure 1 also reveals that the range of PL variability also decreased during the conditioning sessions. Figure 2 depicts the average EP obtained from each of the recording sessions as described above. The wave form of the PL component is not substantially altered. This indicates that the generator of PL itself remained relatively constant throughout the study. The latency shift of PL is observable in Figure 2 when the top or bottom points of the PL trace are compared across successive days of training.

Data obtained from Animal 2 on eight conditioning sessions is represented in Figure 3 (DCP) and Figure 4 (AEP). Animal 2 produced a much more variable distribution of PL components. It is possible to observe the distribution shift in Figure 3 between the hatch marks on either side of the PL distribution range. The DCPs from latter sessions show a smaller range that is shifted to an earlier latency. This change in the DCP denotes a latency shift in the desired direction, although not statistically significant ($t = 3.005$, 3df, $p<.06$).

Figure 4 illustrates the AEPs for the eight consecutive conditioning sessions. The line drawn through the PL

trace aids in denotation of the latency shift of the bottom point in the trace. Note, the top point of the PL component is basically unaltered in latency. As represented in Figure 3, it appears that the bottom edge of the wave was altered to decrease latency; the slope is steeper.

Figures 5 and 6 represent the DCPs and AEPs obtained through seven experimental sessions from Animal 3. The DCPs for this animal represented in Figure 5 are very difficult to read. It provides minimal information, other than the latency range diminishes in the latter sessions. Figure 6 (AEPs) demonstrates that the downward slope of PL becomes steeper in latter sessions. The top point of the PL wave trace remained relatively stable while the bottom point shifted to a point of decreased latency. This latency shift is not significant ($t = 2.756$, 3df, $p<.08$).

Animal 4, the last animal to receive reinforcement contingent upon decreasing PL latency, is represented in Figure 7 (DCP) and Figure 8 (AECP). As in the previous animal, the DCP, Figure 7, provides little information. The print out representing the DCP indicates an increased range in the PL latency distribution. Figure 8 clearly illustrates the latency shift of the bottom point in the wave trace. The slope, in this case, became more gentle in later sessions, indicating that the top point of the wave decreased latency to a greater degree. The shift illustrated in these Figures represent a statistically shorter latency ($t = 4.89$, df3, $p<.05$) (Table I).

Animal 5 was conditioned to increase PL latency. Data obtained on eight recording sessions are represented in Figure 9 (DCPs) and Figure 10 (AEPs). The desired distribution shift is discernible on Figure 9. The portion of the graph between the hatch marks illustrates a distribution shift to the right indicating an increased latency of the PL component. A decrease in the distribution range is exhibited in the latter DCP traces. Figure 10 illustrates the increased latency shift of the average wave if we observe the bottom point of the PL wave trace. The downward slope represented in each wave remained relatively constant, indicating that the top point of the wave resulted in a comparable latency shift. In total, this observed shift is not statistically significant (Table I) ($t = 2.343$, df3, $p<.1$).

Figure 11 and Figure 12 represent data obtained from Animal 6. Figure 11 indicates a decrease in latency. This shift was inappropriate since the animal was reinforced for an increased latency of the PL component.

Figure 11, representing the DCPs of eight recording sessions, portrays an extremely variable latency range if we observe the bottom point of the wave trace. Note the observed shift affects both the top and bottom points of the downward slope, but not the slope itself. Application of the correlated t test indicates a statistically significant but inappropriate shift ($t = 3.16$, 3df, $p<.05$).

Animal 7 was conditioned to increase latency of the PL component. Data obtained from seven conditioning ses-

sions are represented on Figure 13 (DCP) and Figure 14 (AEPs). The shift portrayed in these figures is not statistically significant ($t = 1.74$, 3df) (Table I). However, Figure 13 illustrates that the appropriate distribution shift was occurring during the first three sessions. On the fourth session a substantial inappropriate shift took place, followed by three more sessions in which the desired latency distribution is observable. Figure 14 provides little observable information regarding the latency shift desired. However, a very noticeable alteration in the wave form is observable in the later wave traces which may partially explain the sudden and inappropriate shift mentioned above.

Animal 8 was conditioned for twelve consecutive days to increase the PL latency. Figure 15 (DCPs) and Figure 16 (AEPs) represent the data obtained during these sessions. Figure 15, the DCPs, obtained during twelve recording sessions, illustrate an extremely variable PL latency. The PL distribution appears to increase in variability and to shift to a decreased latency in the later sessions. Figure 16 illustrates a slight alteration in the PL distribution toward the desired direction until session eight. Following session eight, the response produced by the animal changed substantially. This change is observable in the double PL component visible between the hatch marks ($t = 5.657$, 3df, $p<.02$).

Animal 9, the final animal to receive reinforcement contingent upon increased latency of the PL component, pro-

duced the desired response. The data represented in Figure 17 (DCP) and Figure 18 (AEP) is obtained from eleven recording sessions. The distribution shift is significant at $p<.05$ (Table I). The wave traces in these two figures are difficult to read. However, in both graphs, a double wave (top) and triple wave (bottom) is prominent. Data obtained were significant ($t = 3.563$, 3df, $p<.05$).

Animal 10, employed in a control group, has data illustrated in Figure 19 (DCP) and 20 (AEP). Figure 19 depicts an extremely stable distribution of PL component. The range and latency of PL are relatively constant throughout all sessions. Figure 20 (AEP) portrays a few wave trace shifts. However, if we read from top to bottom, no directional shifts in either bottom point or top point of the wave are maintained ($t = .948$, 3df, p n.s.).

Data from Animal 11 is represented in Figure 21 (DCP) and Figure 22 (AEP). Depicted in Figure 21 is a PL distribution shift to the left (decreased latency). The distribution range remained constant throughout the study. Figure 22 illustrates the wave shift, both bottom point and top point of the PL wave trace shift to the left. This shift is not statistically significant ($t = 2.933$, 3df, $p<.1$) (Table I).

Data obtained from a third control, Animal 12, is shown in Figure 23 (DCP) and Figure 24 (AEP). Two statistically significant shifts are portrayed in this data. The PL distribution illustrated in the DCPs first shift to an

increased latency and then return to a baseline position. The PL distribution range illustrated in Figure 23 remains relatively constant. However, Figure 24 portrays a great deal of variability in the PL component wave form. Reading from top to bottom, both an increased amplitude and steeper slope are observable. A shift in both directions is discernable in Figure 24. Correlated t tests taken between sessions one to three and six to eight resulted in $t = 3.928$, $p<.05$; between sessions six to eight and then ten to twelve resulted in $t = 4.99$, $p<.05$. However, t obtained in the manner employed for all other animals indicates a significant shift toward a decreased latency ($t = 3.485$, 3df, $p<.05$) (Table I). The former tests were included to express the fact that latency shifts for this animal were significant in both directions.

In summary, six animals in the experimental group were able to produce the desired PL component latency shifts. One animal produced no appreciable change, and two animals produced inappropriate latency shifts. The three control animals did not illustrate any reliable latency shifts. See Figure 25 of data means illustrated in graph form as a summary.

CHAPTER 4

DISCUSSION

According to Fox and Rudell (1968), if a neural function can be manipulated for adaptive purposes, it must carry information for the animal. Further, if this condition is met, that neural function can be considered a code, or part of the neural language. The present findings provide evidence that pertains to the animal's ability to shift the PL latency for adaptive purposes. Consequently, we must conclude that PL latency is a signal that carries information for the animal. The information carried by the signal may have been created by the arbitrary experiment, but, regardless of how the signal came to carry the information, the PL component must have become part of the information carrying code that represented reinforcement. Otherwise the animal would not have altered the PL latency for reward.

The experimental results obtained from animals in the decreased latency condition all substantiate the role of PL as code in the sense proposed by Fox and Rudell (1968). Although not all animals produce statistically significant PL latency shifts, Figure 25 clearly illustrates that the latency trends were in the desired direction. An inspection of Figure 25 indicates that the performance of the animals who failed to show statistically reliable trends (Animals 2

and 3) was by no means asymptotic.

The lives of Animal 2 and Animal 3 were terminated prematurely when their electrode caps became loose. The desired latency shift displayed by these animals might have been greater if it had been possible to continue conditioning.

Analysis of the results obtained from Animals 1, 2, 3, and 4 illustrated that three different processes contributed to the latency shifts observed in Figures 1 to 13. Animal 1 shifted the latency of the PL component intact, such that the downward slope of the component remained relatively constant. Both top and bottom points of the wave trace display a parallel shift. Animals 2 and 3 shifted the bottom point of the wave while the top point remained relatively unchanged. The latency shift observed in Figures 4 and 6 illustrate a steeper slope in the waves from later sessions. The decreased latency of the bottom point of the wave is the major contributor to this observable shift. Conversely, Animal 4 shifted the latency of the top point of the PL wave while the bottom point remained relatively constant (Figure 8). The general result was a wave trace with a more gentle slope. The fact that three different responses are displayed in decreasing the PL latency supports Fox's point regarding variability in acquisition of motor behaviours.

Fox stated that reliable brain correlates of behaviour are almost impossible to obtain due to subject varia-

bility in response acquisition sequence when acquiring gross behaviours. In light of the above, the problem would be further complicated by the inter-animal variability in neuronal response acquisition. Further, Fox states that behaviour may be both multiply and sequentially represented in the brain and learning may be accompanied by shifting of bioelectrical process during the acquisition phase. The above would indicate that if shifting of the bioelectrical process does take place, that process likely involves inter-animal differences.

The results obtained from the conditioning study with these four animals support John (1972). He proposed that the critical event in learning is the development of a common mode of activity, a temporal pattern that is coherent across various effected regions and specific for that stimulus complex. In each of these animals a change in the PL component was established. The alteration in the response was unique for each animal but within each animal portrays a common mode of activity.

A criticism of employing an evoking stimulus paradigm is that the animal could possibly generate criterion responses by selective orientation to the stimulus. However, it was shown (Fox, 1970; Rudell, 1970) that selective orientation was not a necessary method of obtaining reinforcement, nor was it preferred (Rosenfeld, Hetzler, and Konsik, 1974). Consequently, in relation to the above discussion, we can conclude that changes observed are a result

of changes in the underlying neuronal generators. This proposition is further supported by Rosenfeld, Hetzler, Birkel, and Kowatch (1975) using a random inter-trial interval which ruled out the behavioural mediation hypothesis. It was reasoned that knowledge of when to make a discrete behavioural response could be gained from a regular inter-trial interval. Therefore, if successful conditioning was obtained using a random inter-trial interval, the proposition that changes in the criterion component of the visual EP was mediated by central, not peripheral, events would have been demonstrated.

In accordance with these findings, it may be necessary to reject Walker's suggestion (1974) that Type I events are not suitable to encode variable perceptual phenomena due to the high degree of reliability of occurrence of these components. He states that the reliability raises questions concerning the perceptual significance of the Type I event. The results obtained in this study provide evidence to the contrary. The PL component is reliable but it has sufficient latency variability to be altered for adaptive purposes. Hence, the position of this component can be considered part of the information code for the animals.

The increased PL latency condition did not provide any conclusive results. Only Animal 5 and Animal 9 were conditioned to produce the desired trend in the PL latency. Animal 5 shifted PL component intact as described above

while Animal 9 produced a triple PL component in the latter sessions which aided in shifting the mean latency of criterion responses for reinforcement. This data provides further evidence in support of Fox (1970) and John (1972) as discussed above.

The mean latency shift in Animal 5 and Animal 9 was 14 msec and 10 msec respectively. Although neither shift has statistical significance, a reliable shift of that size would give way to approximately 3-4 fast positive potentials (fpp). If the assumption is made that fpps carry information, a change of that magnitude must have meaning to the animal and as such must be significant.

Animal 6 shifted the PL component in the undesired direction. This was an unexpected result that could have been due to the experimental design employed. The position of downward slope of the PL component was used as criterion for reinforcement. If, in the increased PL latency condition the upward slope had been used, the animal may have been more easily conditioned. It is likely that the slope employed as the independent variable resulted in confusion for the animal. Figure 11 illustrates that Animal 6 produced a triple PL component quite reliably which could have rendered him incapable of associating a given PL component range with reinforcement. This assumption appears tenable since the animal was reinforced for several different PL components. The experimental program allowed reinforcement of any component of the desired amplitude within a given

range.

Animal 7 produced no overall shift. He began to shift the PL component in an appropriate direction during the first three sessions but had an extreme reversal on day four (Figure 25). Figure 13 visually displays the desired shift on days five to seven. This shift was not sufficient to produce a significant latency change. Figure 13 may provide some insight into the unexpected latency shift that took place on the fourth conditioning session. According to Pickenhain and Klingberg (1965) the animal must be accustomed to his surroundings or environment if EPs of high amplitude and an AD are to occur reliably. If the animal is obstructed or frustrated, the AD (PL) component occurs either with a lower amplitude or not at all. Observation of the results displayed on Figure 14 illustrates that the animal produced lower amplitude PL components at the time this reversal took place, and increased amplitude again when the desired trend reappeared. The trend established in the later days of conditioning adds further support to the adaptive significance discussed in reference to latency.

Animal 8 produced an overall latency shift in the undesired direction. This animal displayed an appropriate trend during the first six conditioning sessions. However on the seventh session, a dramatic reversal occurred, followed by inappropriate temporal shift. A satisfactory explanation for the shift is not feasible. The data would indicate that the wave form was altered dramatically during

the later sessions (Figure 16). This could have been the result of frustration as discussed above (Pickenhain and Klingberg, 1965).

Haider, Spong, and Lindsley (1964) have shown that reduced attentiveness was accompanied by a corresponding reduction in amplitude of the EP while Davis (1964) indicated that enhanced attentiveness increased the amplitude of evoked potentials.

Morell (1961) has shown that habituation of EPs does occur, such that if a stimulus is presented repeatedly with no behavioral consequences for the animal, the EP will diminish in amplitude. Observation of Figure 16 which portrays a greatly diminished wave form on later sessions could be reasonably a result of habituation or reduced attentiveness since this animal did not learn the appropriate response for reinforcement.

Data obtained from three control animals support the fact that the animals conditioned to produce a latency shift were actually conditioned. The control animals did not make any reliable latency shifts.

Animal 10 made no shift while Animal 11 and Animal 12 shifted in opposite directions and then returned toward the initial latency position (Figure 25). This data indicates that the PL component does not necessarily have a fixed latency position. However, it does indicate that if any latency shift is going to be maintained, it is done with respect to adaptivity. In light of the results obtained on

all twelve animals, it would not be misleading to conclude that PL component latency can be manipulated. The manipulation carried out with food reinforcement indicates that the animal can control the PL component as an adaptive behaviour.

The decrease latency group showed much greater ease in conditioning. As well, there appeared to be some tendancy for animals to decrease latency over time. John (1967) has shown that the latency of PL is determined by the point in time at which ambiguity is reduced. It appears that when the animal becomes familiar with the meaning associated with the EP creating stimulus, the latency of PL may decrease.

Walker (1974) has discussed the high degree of reliability of occurrence of these components. He states that the reliability raises questions concerning the functional significance of the Type I event. Also, numerous other investigators have suggested that Type I events, like PL, are not information carrying codes, the rationale being that these events were not believed to have sufficient variability.

Marsh, McCarthy, Sheatz, and Golambos (1961) found that Type I events still occurred after prolonged periods of stimulus presentation. Consequently, it would seem that the occurrence of Type I events are not related to habituation nor do they provide substantial evidence that the animal is attending to or is aware of the stimulus since behavioural habituation is complete within a much shorter time period.

In conjunction with these findings, Pickenhain and Klingberg (1965) have demonstrated that the animal must be accustomed to his surroundings or environment if EPs of high amplitude and a PL component are to occur reliably. Furthermore, Fox (1970) proposed that it is not feasible to assess the significance of variations in component latency to the animal since the animal does not receive information about the real time of the stimulus onset. On the other hand, Shucard and Horn (1971) state that latency variation may give some information about the state of the nervous system when the stimulus is delivered.

In light of the previous findings, we can deduce that the conditioning of the PL component was a result of alteration and stabilization of the generator mechanisms associated with the PL component. The present work has demonstrated that PL latency was a signal in the sense that the state associated with its production could be controlled for adaptive purposes.

We can accept PL as an information carrying code. Although the data is not sufficiently clear to draw strong inferences, an analysis of selected animals (Animals 1, 4, 9) support the hypothesis. This is substantiated by examining the reversal, of Animal 1, obtained by use of reinforcement contingent to an increased latency.

The other animals did not provide any statistically significant data. It was felt that these animals may have achieved the required results if more time could have been

spent in conditioning, since they were not at asymptote. As well, it is difficult to condition against the direction in which arousal takes the AD.

In future studies of this nature, fewer animals, with more days of conditioning per animal, would be advised. Furthermore, animals should be selected with a good clear signal and a notch stencil should be employed.

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APPENDIX

TABLE I

Decreased Latency	t value	df	p value
Animal ONE	t = 7.140	3	p .01
Animal TWO	t = 3.005	3	p .06
Animal THREE	t = 2.756	3	p .08
Animal FOUR	t = 4.89	3	p .05
Increased Latency			
Animal FOUR	t = 2.343	3	p .1
Animal SIX	t = 3.16	3	p .05
Animal SEVEN	t = 1.74	3	N.S.
Animal EIGHT	t = 5.657	3	p .02
Animal NINE	t = 3.563	3	p .05
Control			
Animal TEN	t = .948	3	N.S.
Animal ELEVEN	t = 2.933	3	p .1
Animal TWELVE	t = 3.486	3	p .05

FIGURE 1

Distribution of Criterion Potentials obtained from Animal 1. Graphs 1 and 2 depict sequentially two days baseline. Graphs 3 to 12 inclusive represent 10 consecutive conditioning sessions. Graphs 13 to 15 illustrate the result obtained on the 60th to 62nd day of extinction.

The horizontal axis is calibrated in time. Each calibration is equivalent to 100 milliseconds.

FIGURE 1 D.C.P.

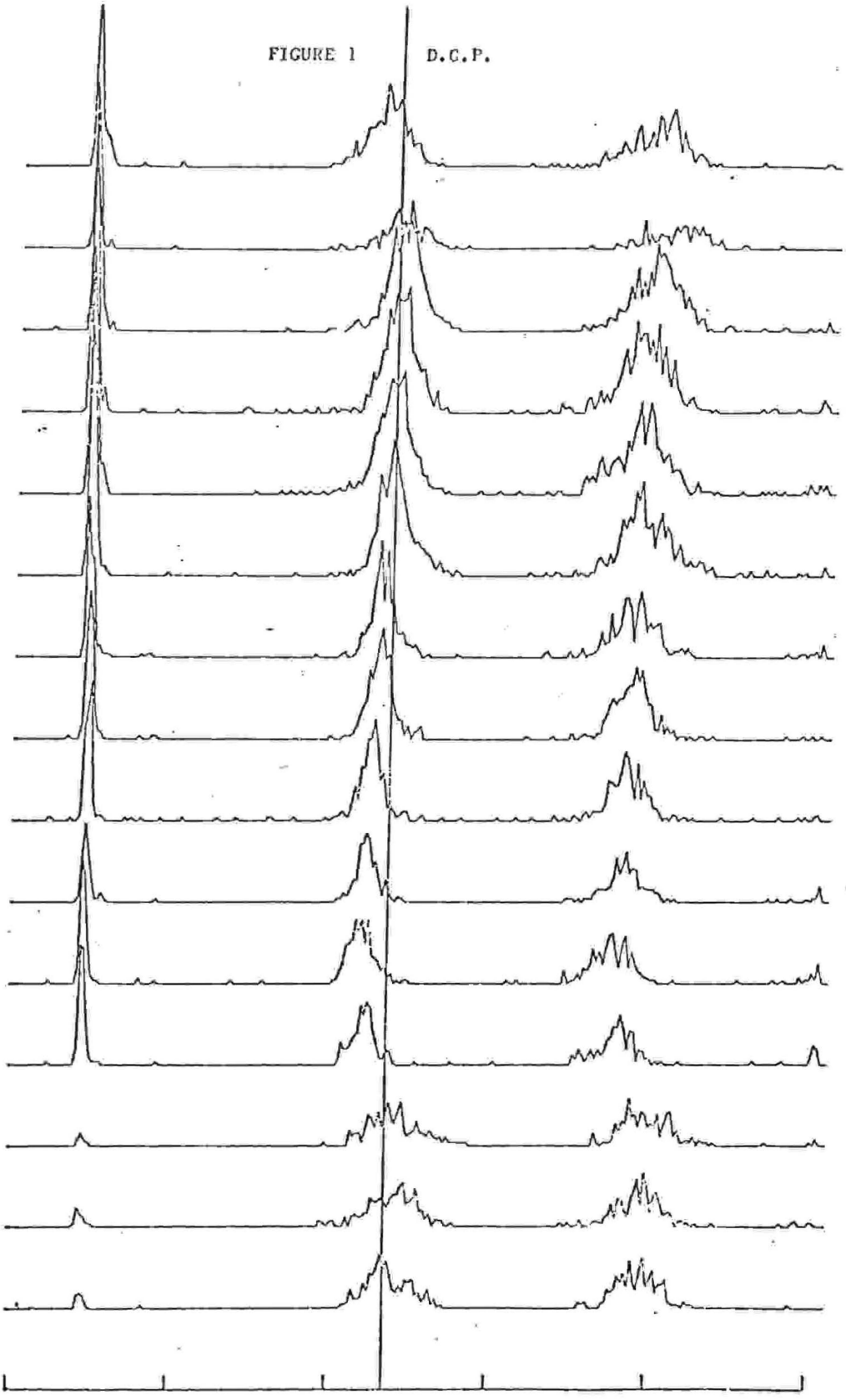

FIGURE 2

Average Evoked Potentials obtained from Animal 1. Graphs 1 and 2 represent baseline recording. Graphs 3 - 12 represent consecutive conditioning sessions. Graphs 13 - 14 illustrate the average wave obtained on the 60th and 61st day of extinction.

The horizontal axis is calibrated in time. Each calibration is equal to 100 msec.

The vertical axis calibration is equivalent to 16 microvolts, negative-up.

FIGURE 2 A.E.P.

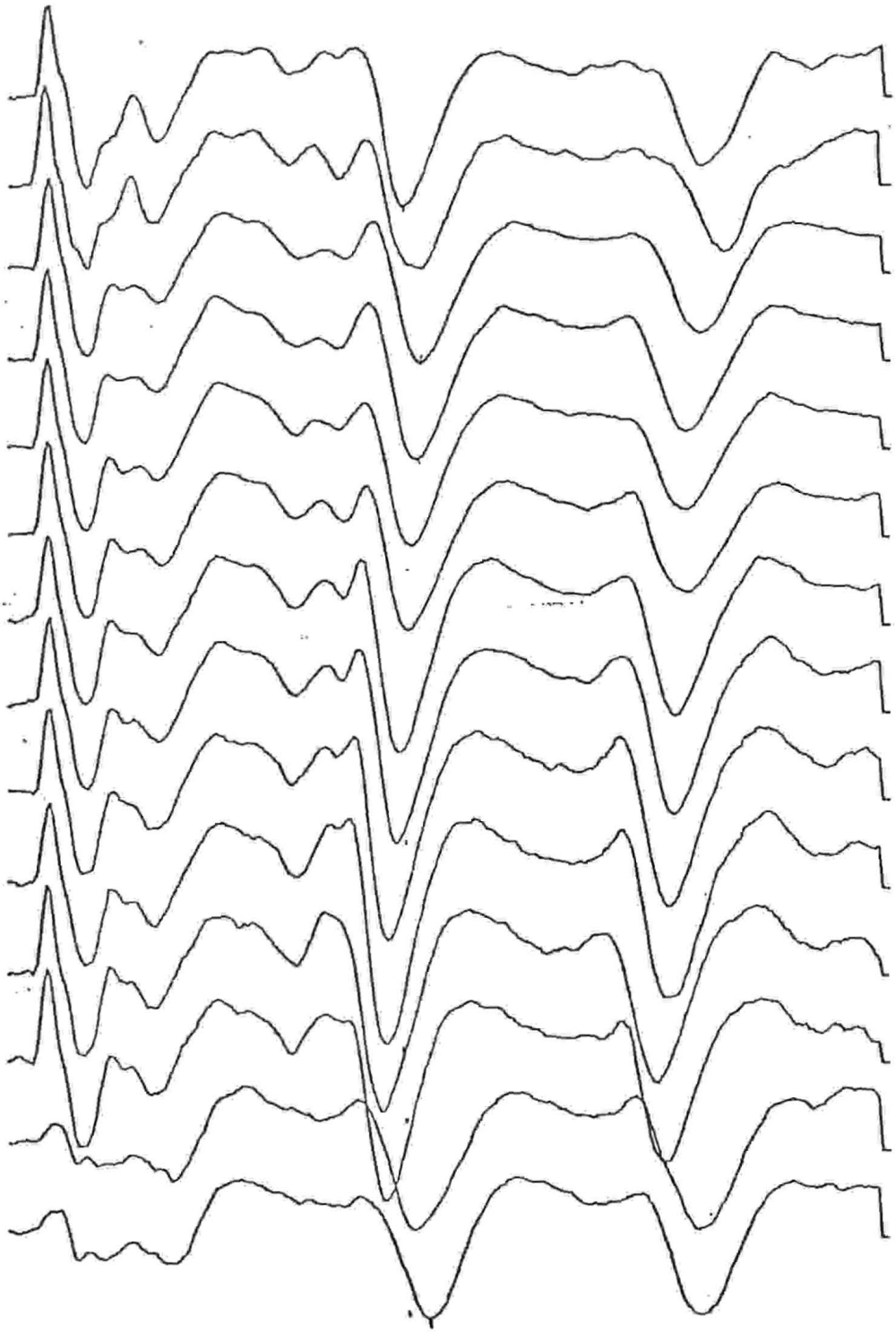

FIGURE 3

Distribution of Criterion Potentials obtained from Animal 2, represents eight consecutive conditioning sessions.

The horizontal axis is calibrated in units of time, each calibration equials 100 msec.

The vertical axis is calibrated in units, representing the number of CP's. Each calibration is equial to one CP.

FIGURE 3 D.C.P.

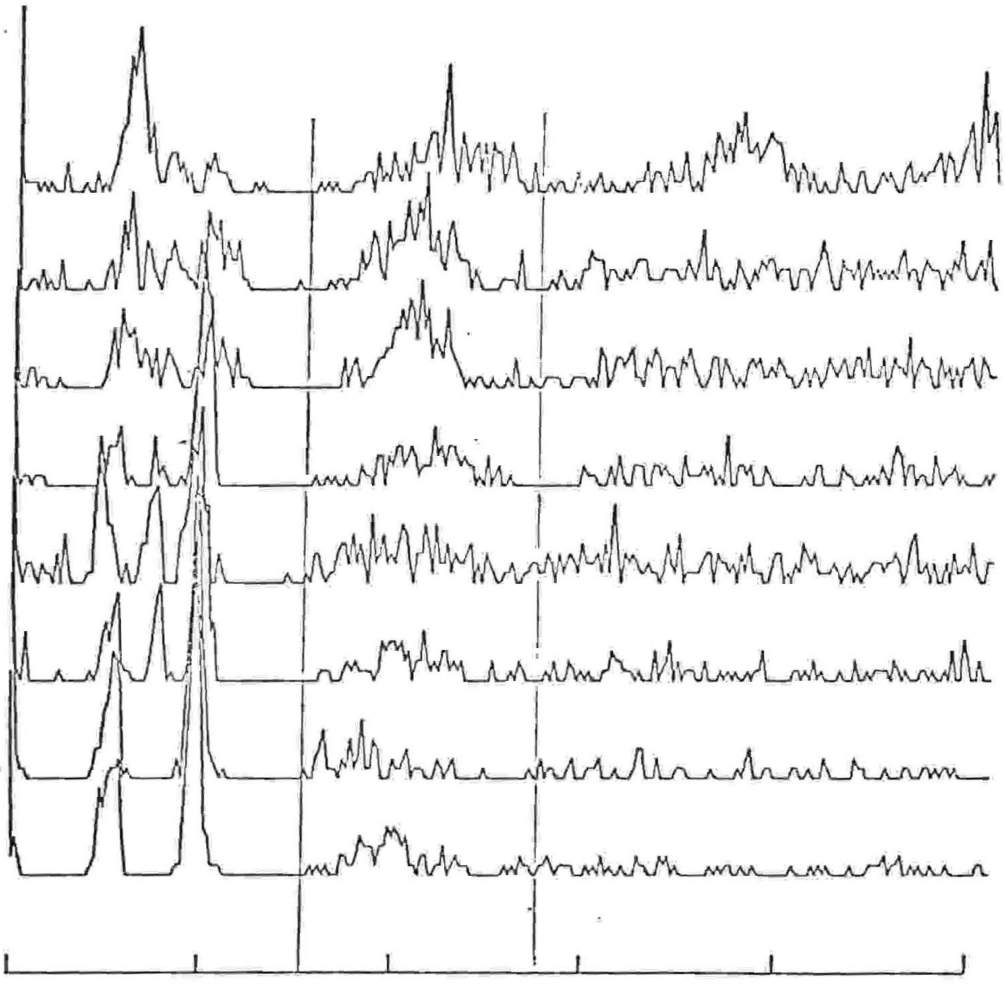

FIGURE 4

Average Evoked Potentials obtained from Animal 2 on eight consecutive conditioning sessions.

The horizontal axis calibrated in units of time, each calibration equals 100 msec.

The vertical axis calibration is equivalent to 16 microvolts, negative-up.

FIGURE 4 A.E.P.

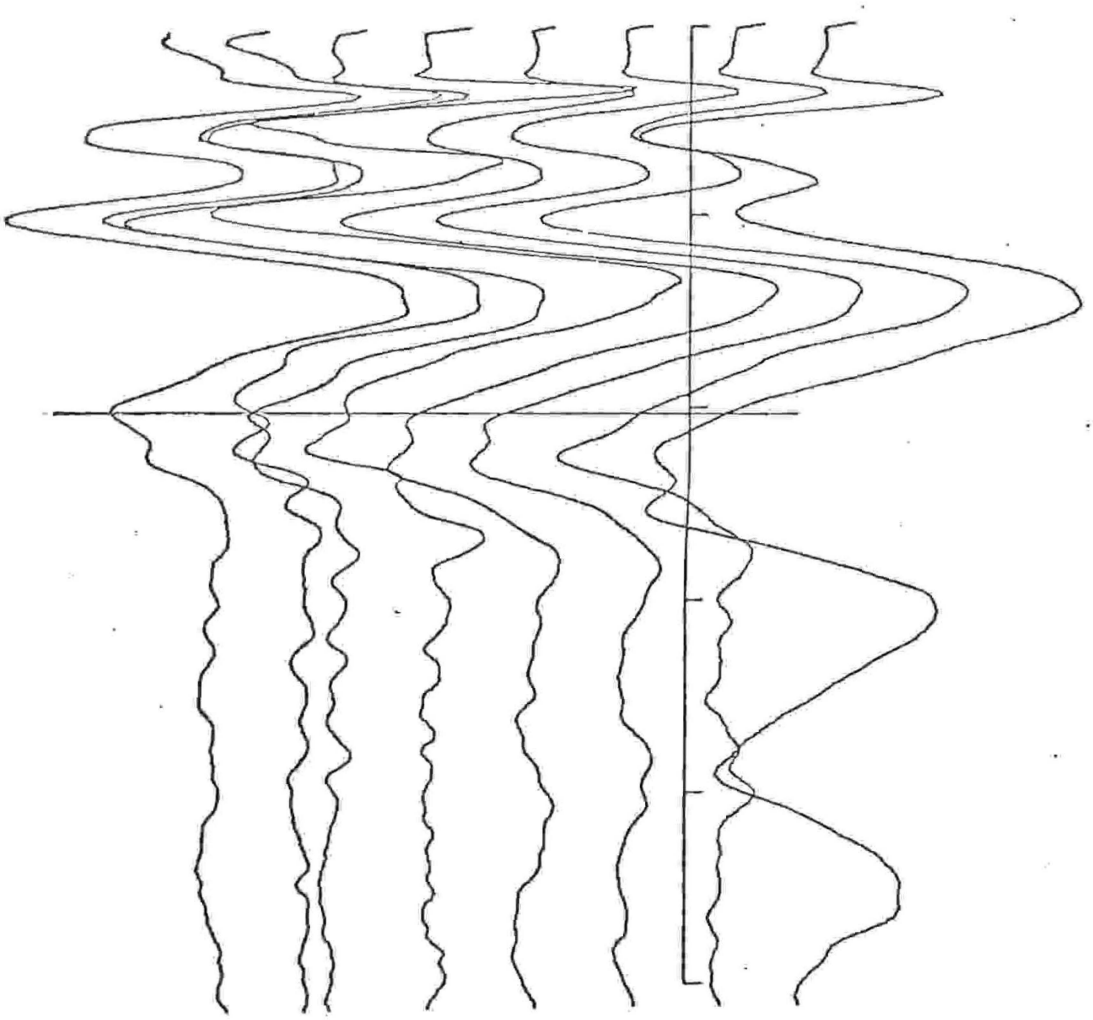

FIGURE 5

Distribution of Criteria Potentials obtained from Animal 3 on seven consecutive conditioning sessions.
The horizontal axis calibration is equivalent to 100 msec.
The vertical axis calibration represents the number of CPs, each calibration is equal to one CP.

FIGURE 5 D.C.P.

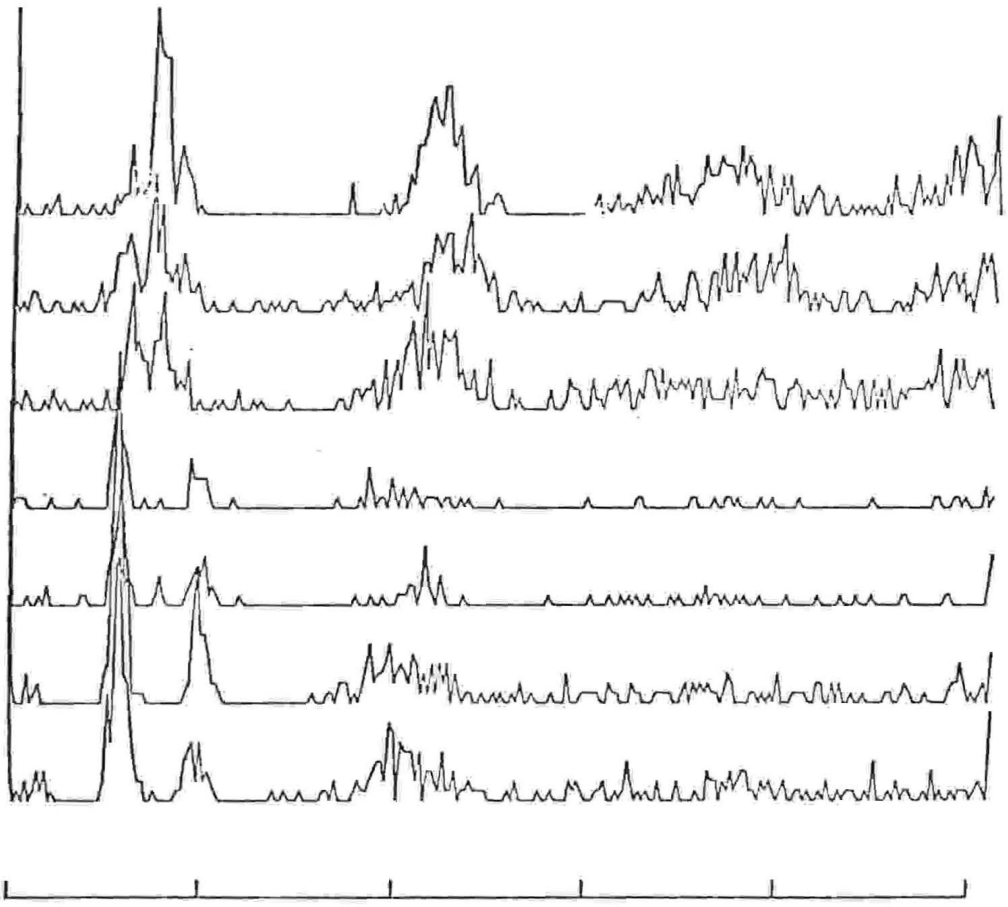

FIGURE 6

Average Evoked Potentials obtained on seven consecutive conditioning sessions from Animal 3.

The horizontal axis represents time, each calibration equals 100 msec.

The vertical axis represents microvolts, negative up, each calibration equals 16 microvolts.

FIGURE 6 A.E.P.

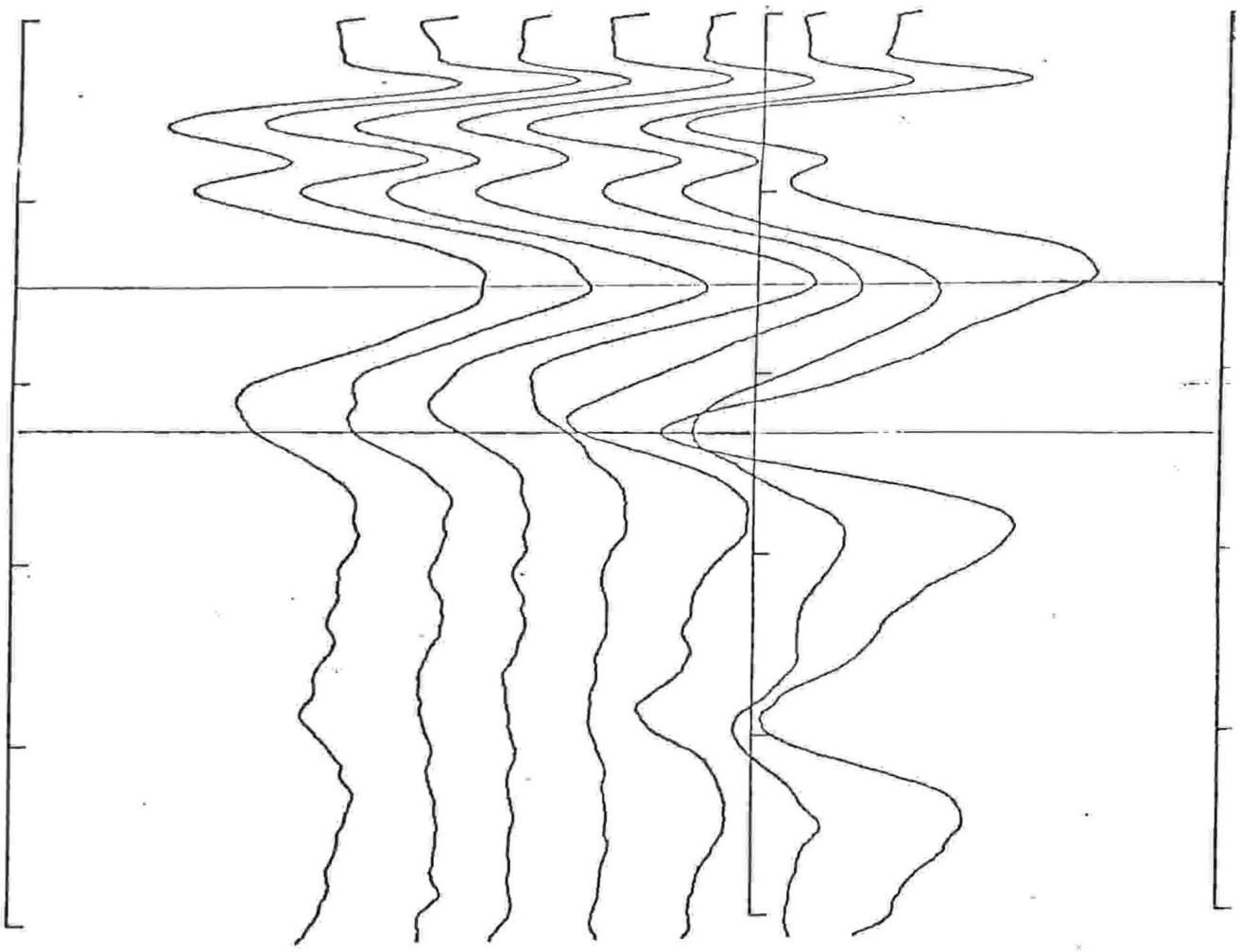

FIGURE 7

Distribution of Criterion Potentials obtained from Animal 4, on nine consecutive conditioning sessions.

The horizontal axis represents time, each calibration equals 100 msec.

The vertical axis represents the number of CPs, each calibration equals one CP.

FIGURE 7 D.C.P.

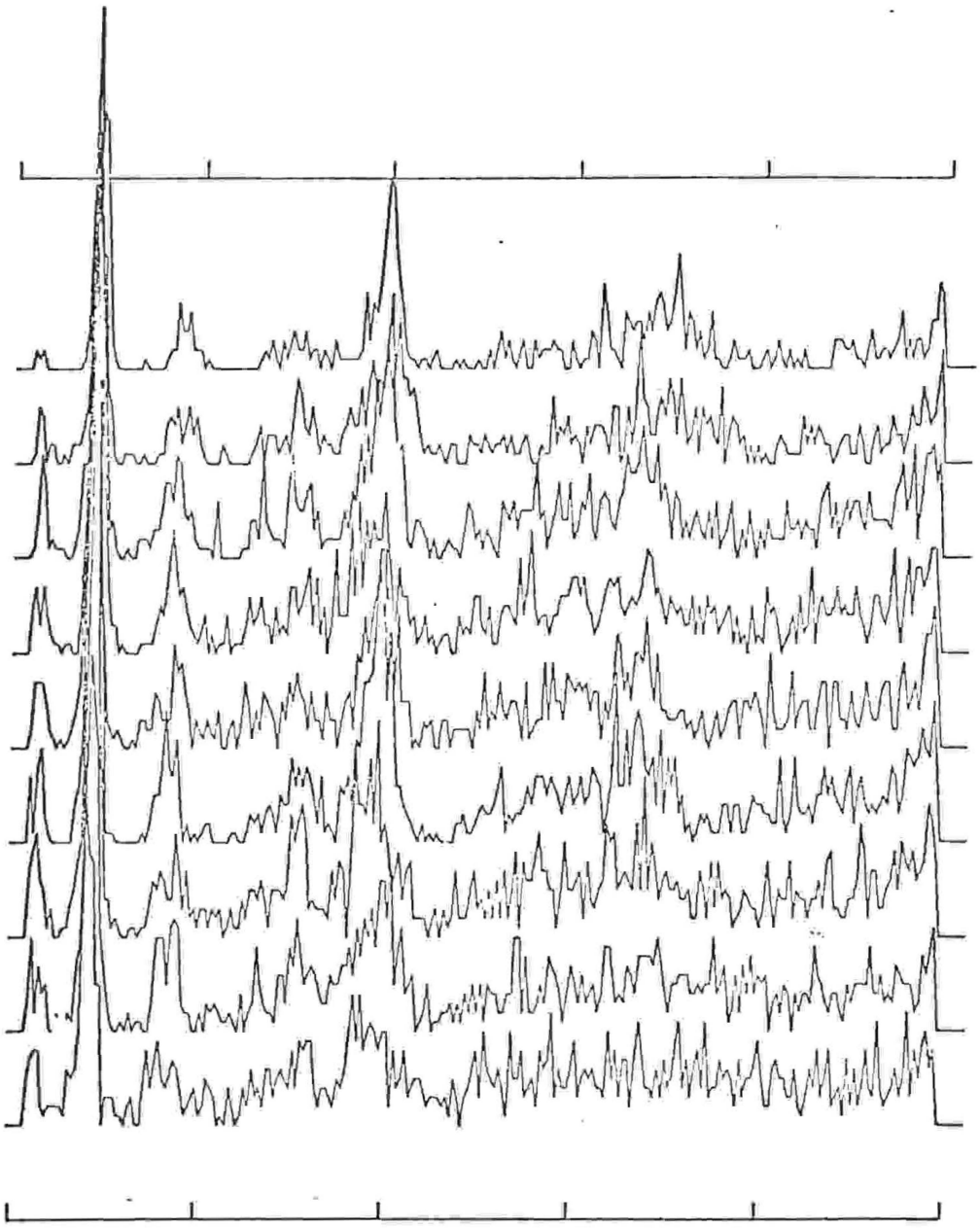

FIGURE 8

Average Evoked Potential obtained from Animal 4, on nine consecutive conditioning sessions.

The horizontal axis represents time, each calibration equals 100 msec.

The vertical axis represents microvolts, negative up, the calibration is 16 microvolts.

FIGURE 8 A.E.P.

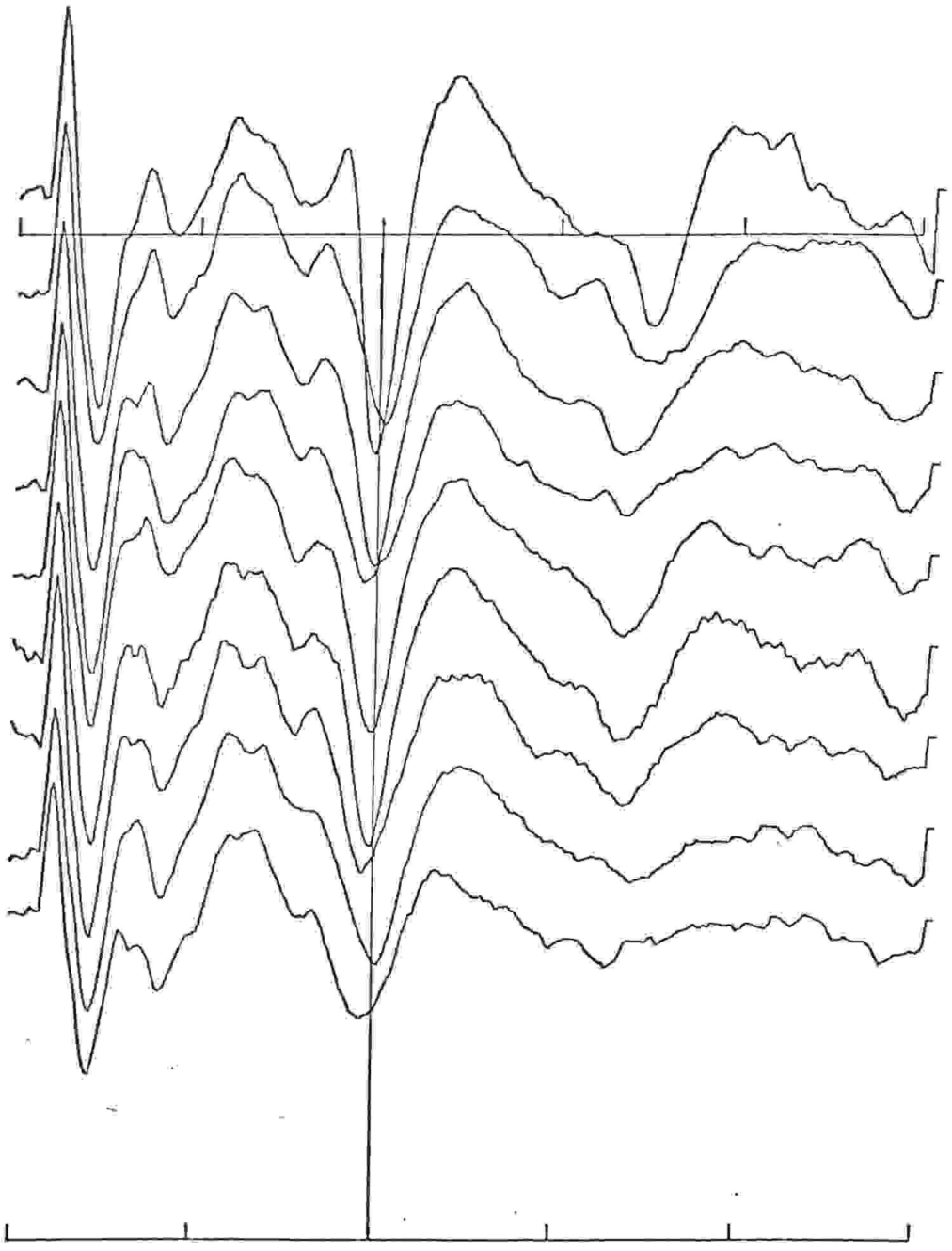

FIGURE 9

Distribution of Criterion Potentials obtained from Animal 5, on eight consecutive conditioning sessions.

The horizontal axis represents time, each calibration equals 100 msec.

The vertical axis represents the number of CPs, each calibration equals one CP.

FIGURE 9 D.C.P.

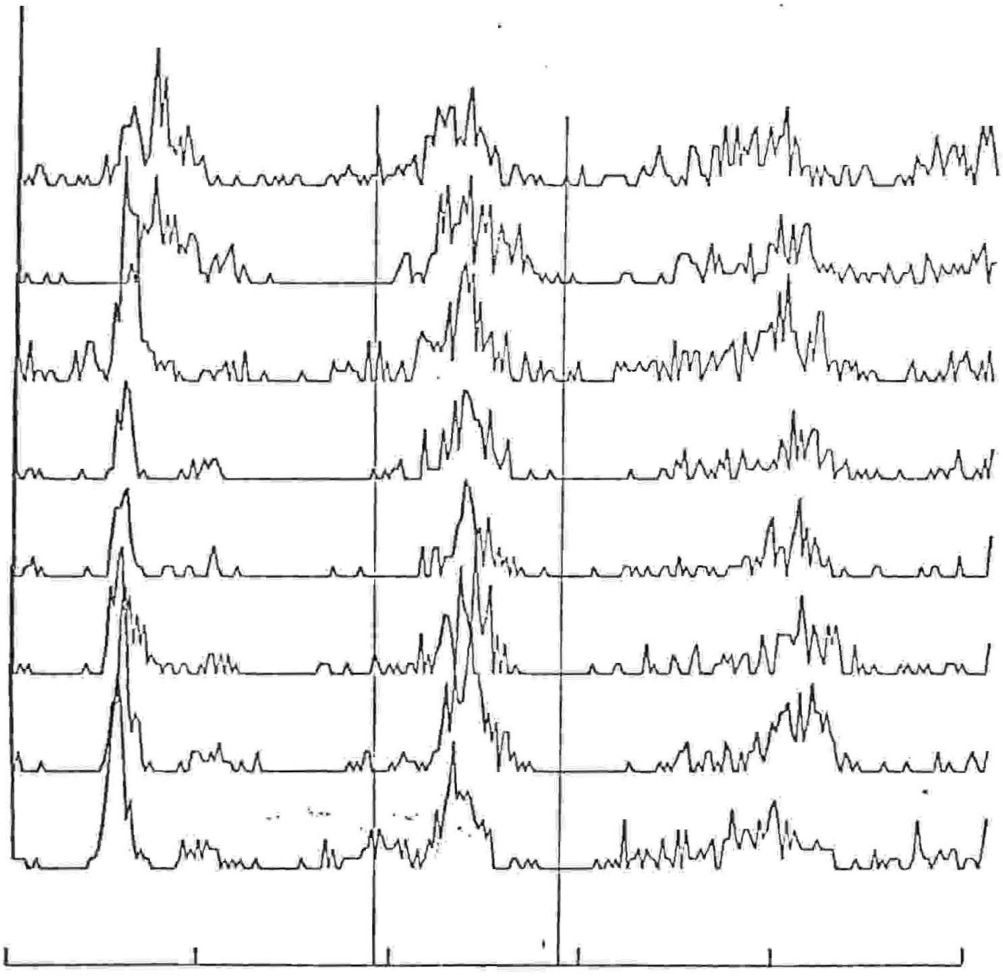

FIGURE 10

Average Evoked Potentials obtained from Animal 5, on eight consecutive conditioning sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents microvolts, negative up, each calibration equals 16 microvolts.

FIGURE 10 A.E.P.

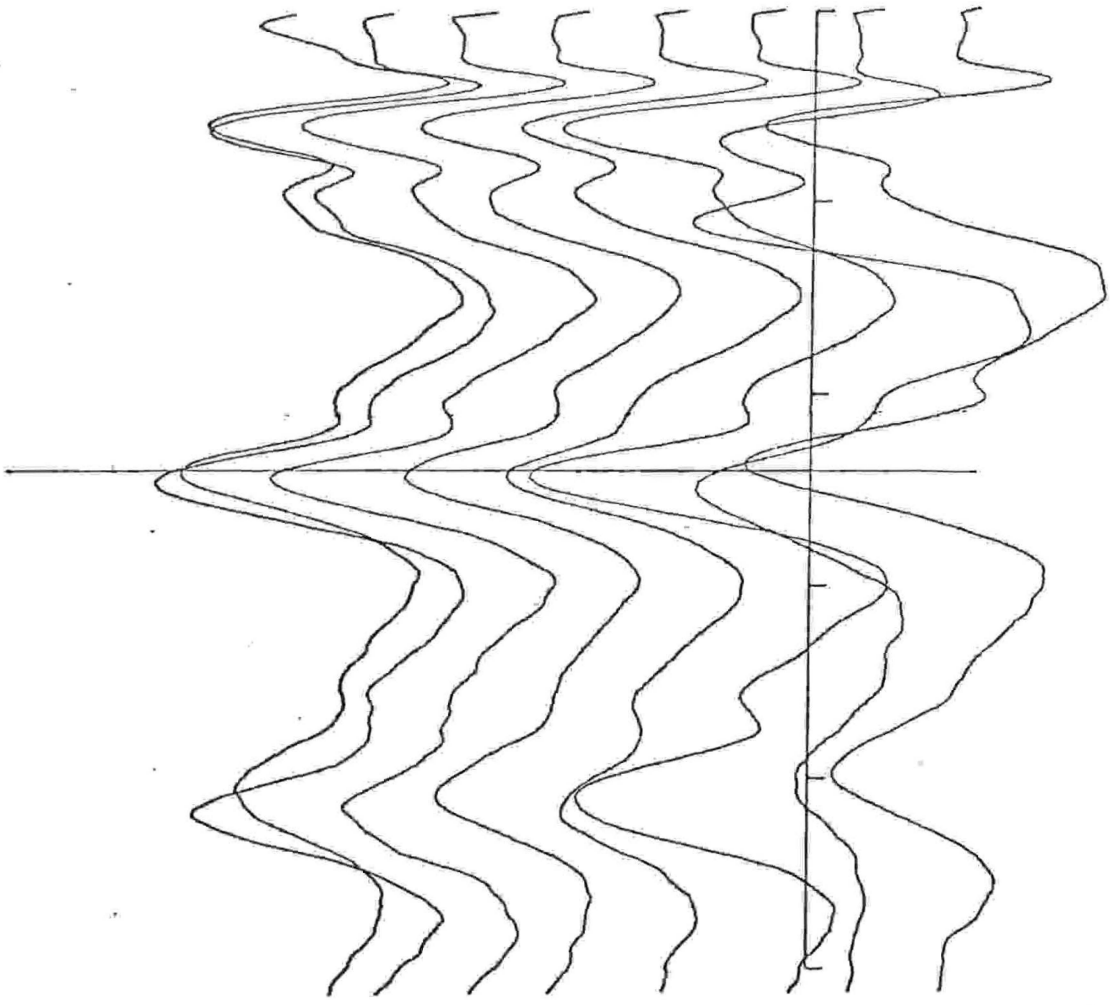

FIGURE 11

Distribution of Criterion Potentials obtained from Animal 6, on eight consecutive conditioning sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents the number of CPs, each calibration is equal to one CP.

FIGURE 11 D.C.P.

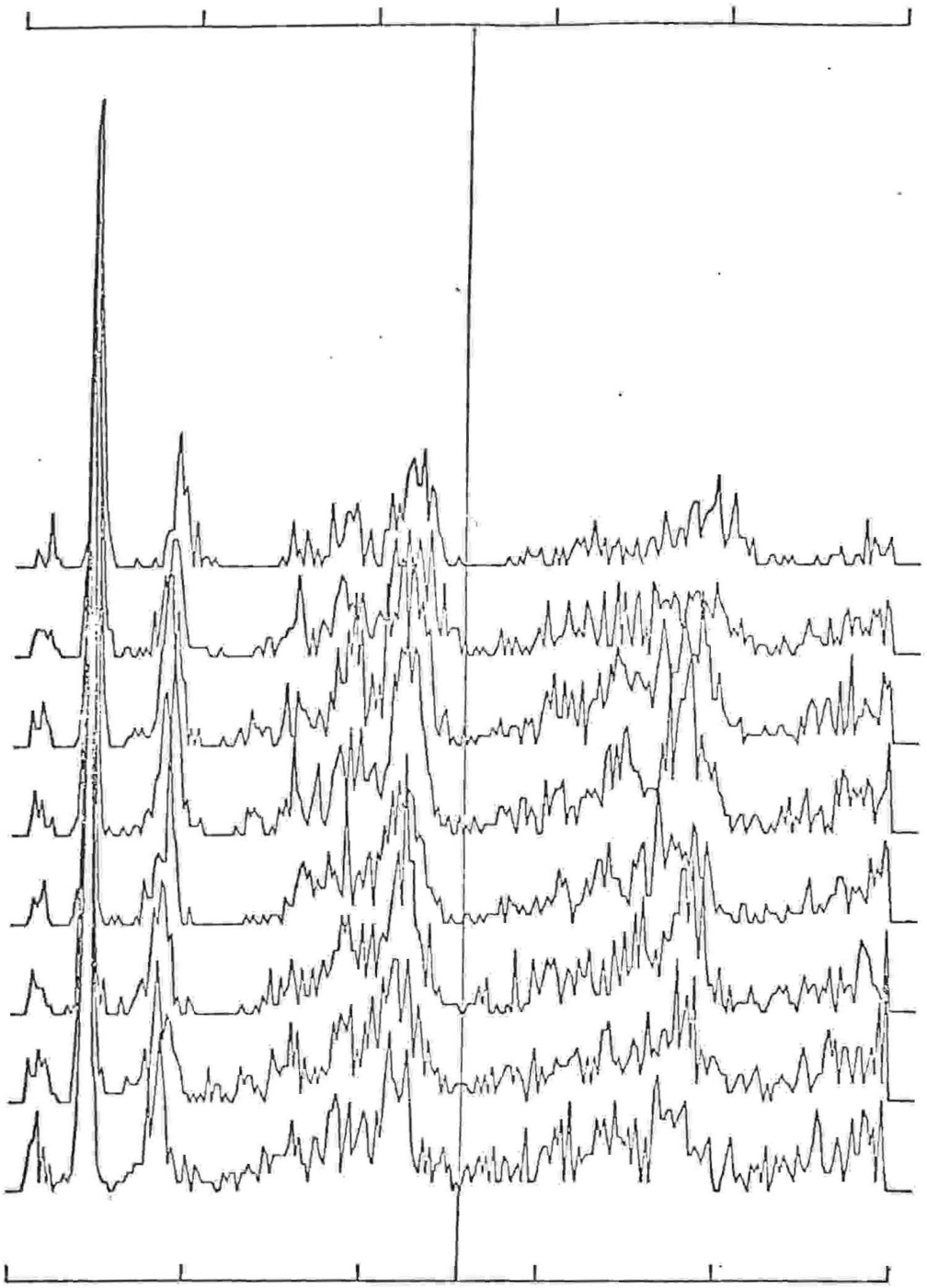

FIGURE 12

Average Evoked Potentials, obtained from Animal 6, on eight consecutive conditioning sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents microvolts, negative up, each calibration is equal to 16 microvolts.

FIGURE 12 A.E.P.

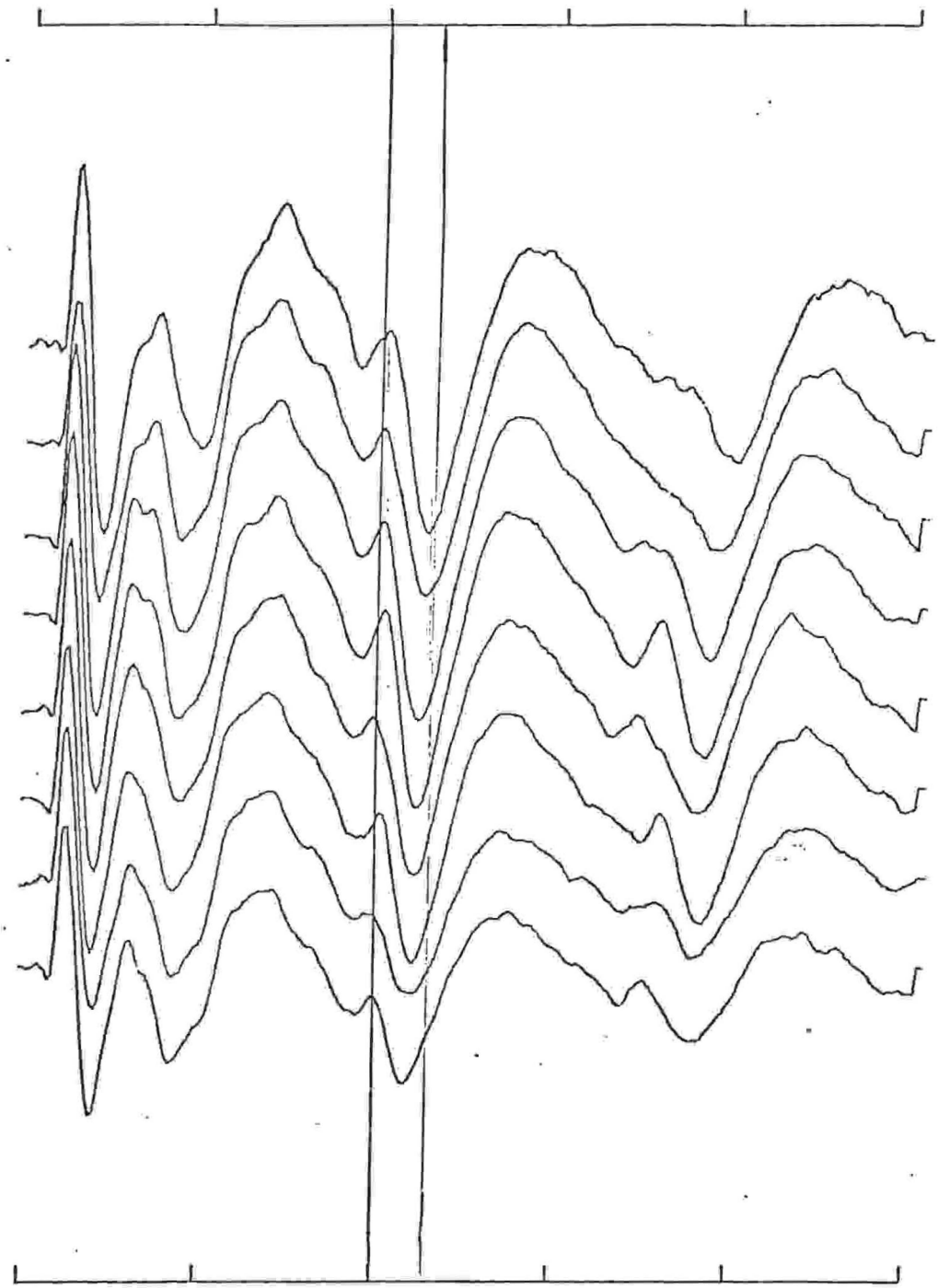

FIGURE 13

Distribution of Criterion Potentials obtained from Animal 7, on seven consecutive conditioning sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents the number of CPs, each calibration is equal to one CP.

FIGURE 13 D.C.P.

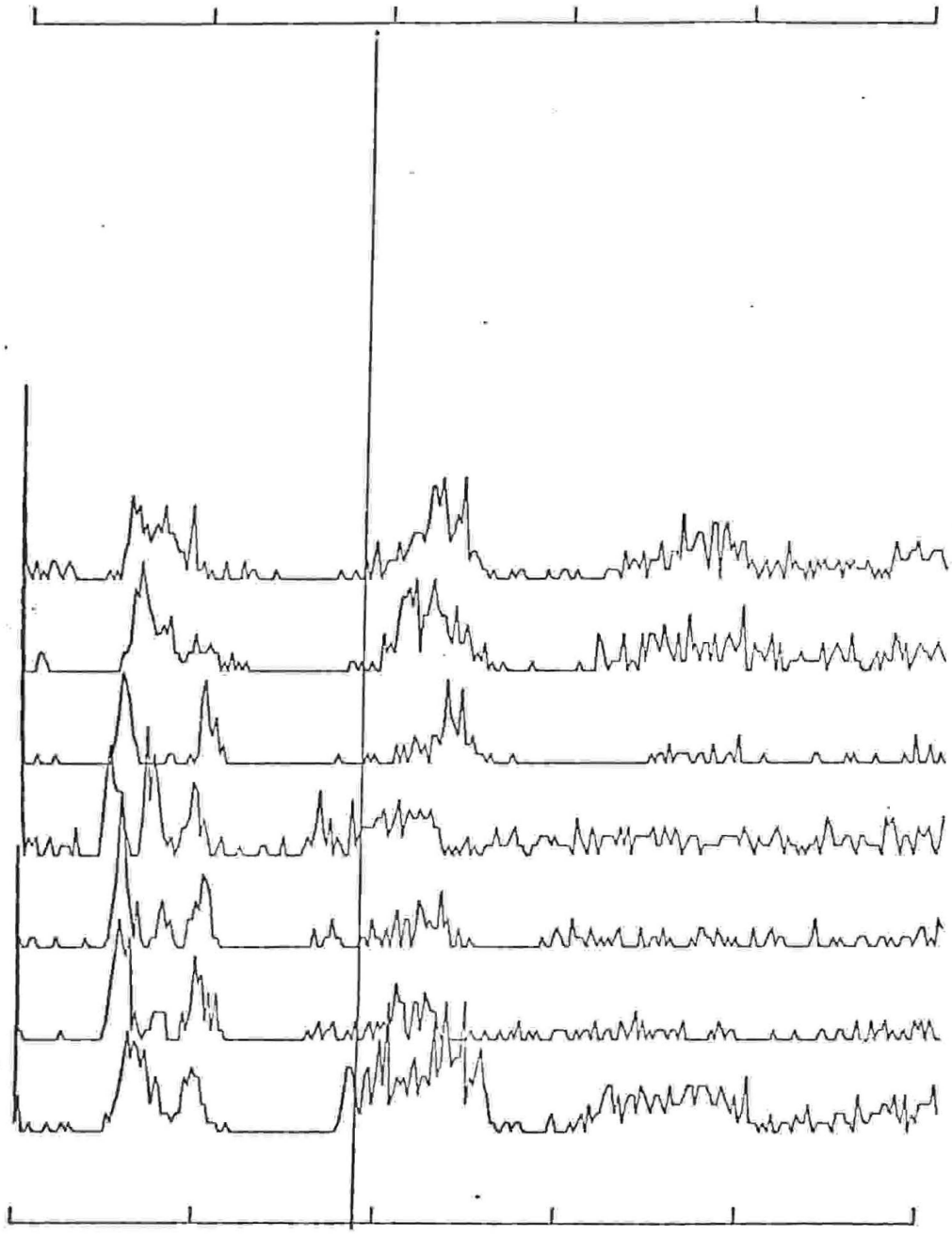

FIGURE 14

Average Evoked Potentials, obtained from Animal 7, on eight consecutive conditioning sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents microvolts, negative up, each calibration is equal to 16 microvolts.

FIGURE 14 A.E.P.

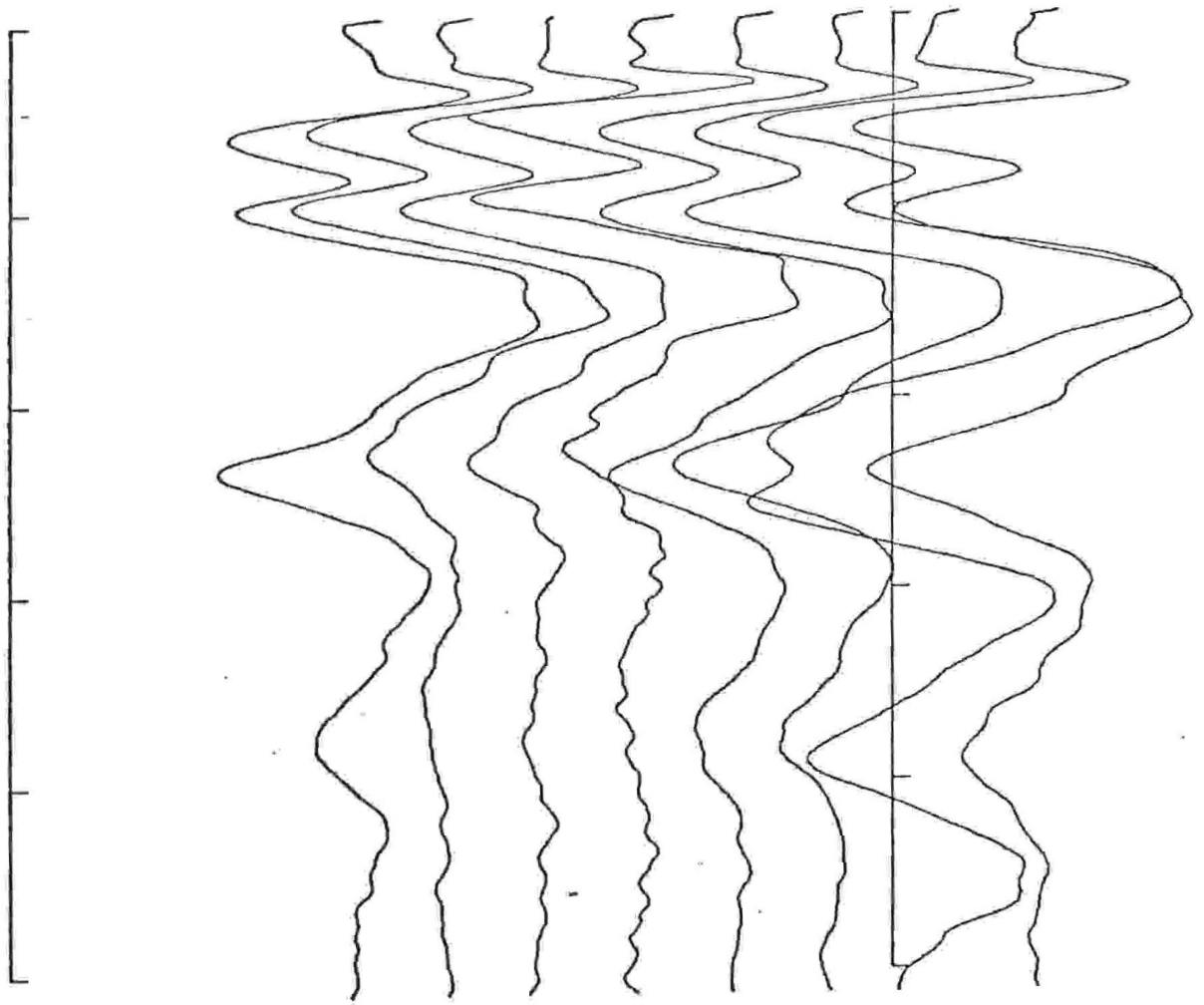

FIGURE 15

Distribution of Criterion Potentials obtained from Animal 8, on twelve consecutive, conditioning sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents the number of CPs, each calibration is equal to one CP.

FIGURE 15 D.C.P.

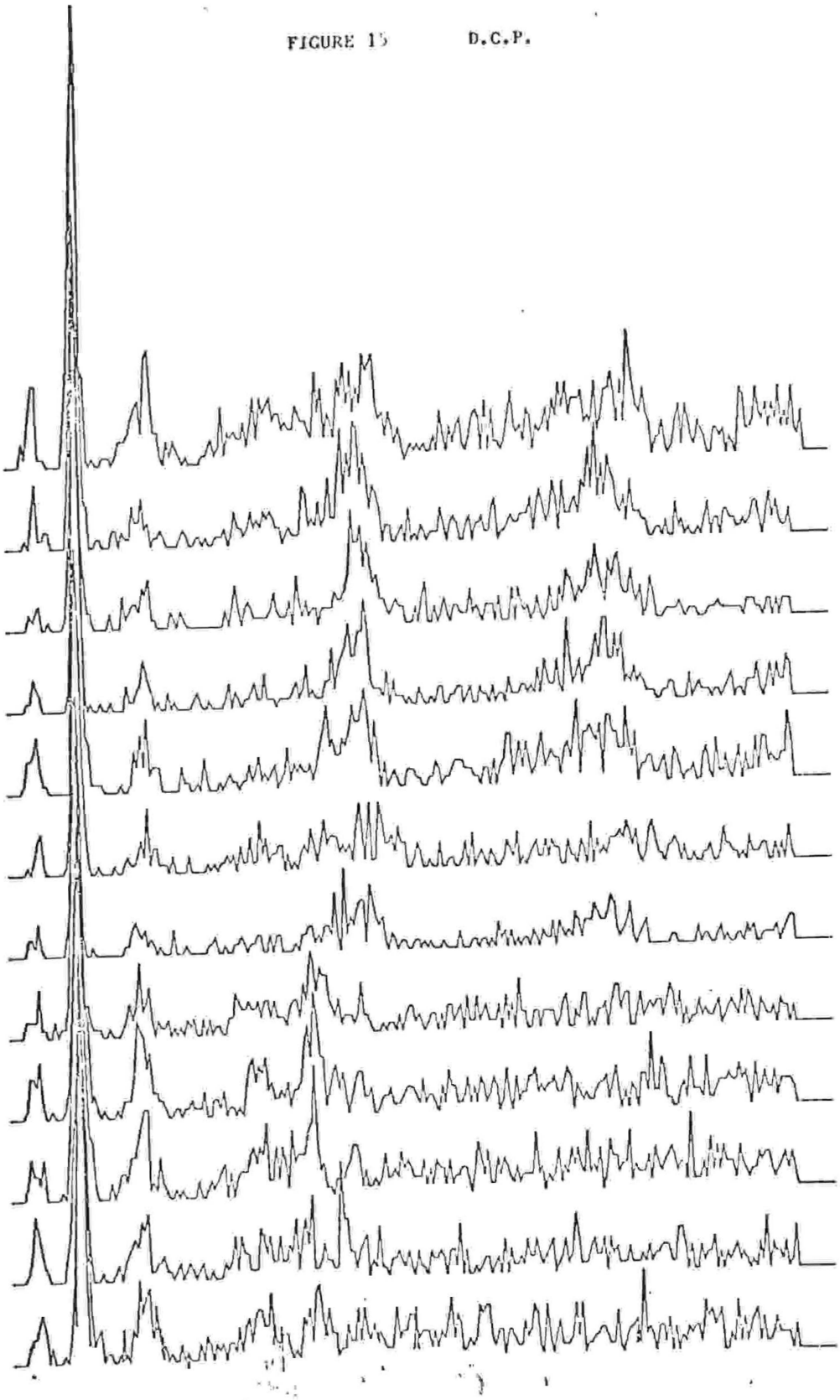

FIGURE 16

Average Evoked Potentials obtained from Animal 8, on twelve consecutive conditioning sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents microvolts, negative up, each calibration is equal to 16 microvolts.

FIGURE 16 A.E.P.

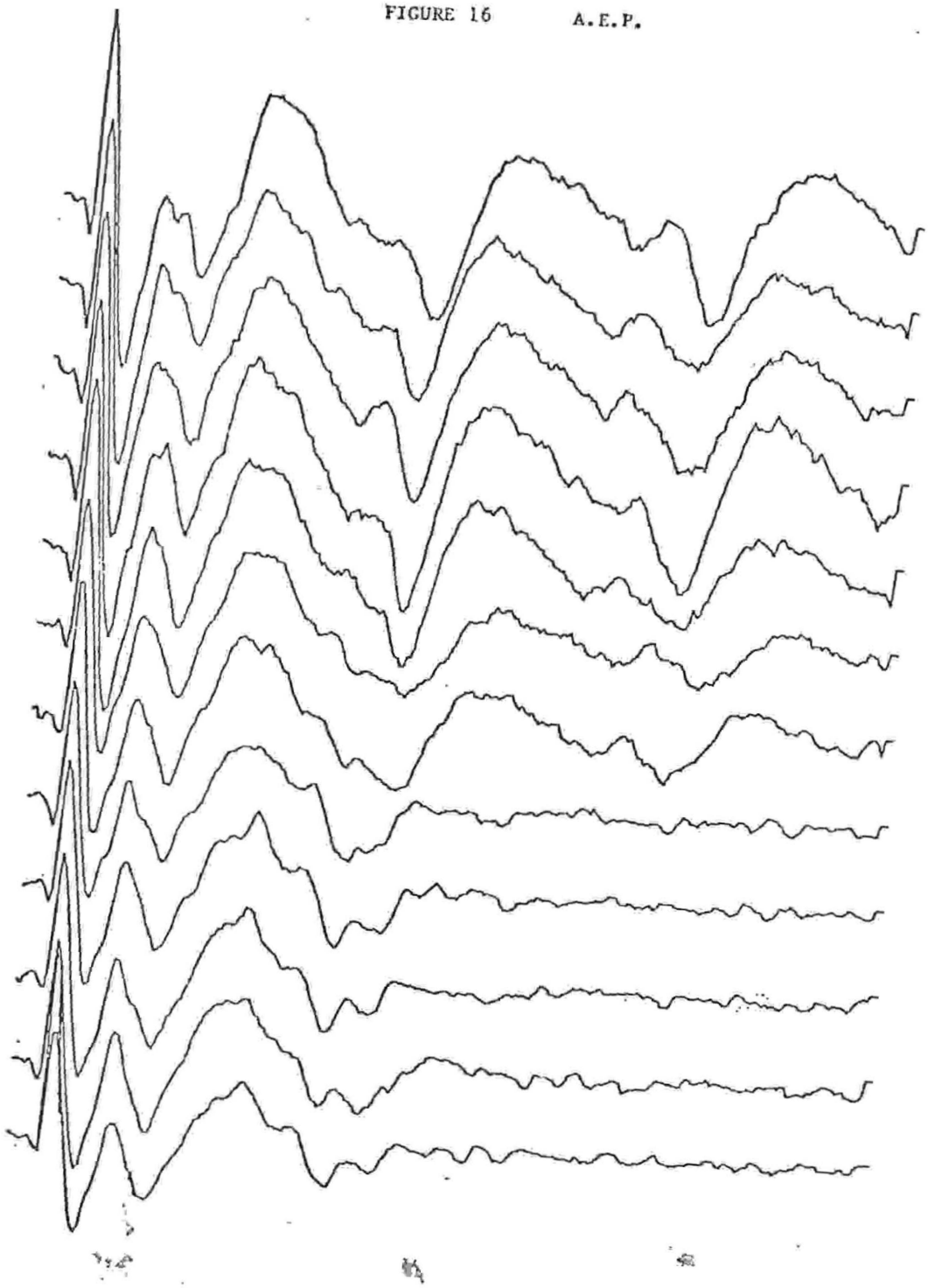

FIGURE 17

Distribution of Criterion Potentials obtained from Animal 9, on eleven consecutive conditioning sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents the number of CPs, each calibration is equal to one CP.

FIGURE 17 D.C.P.

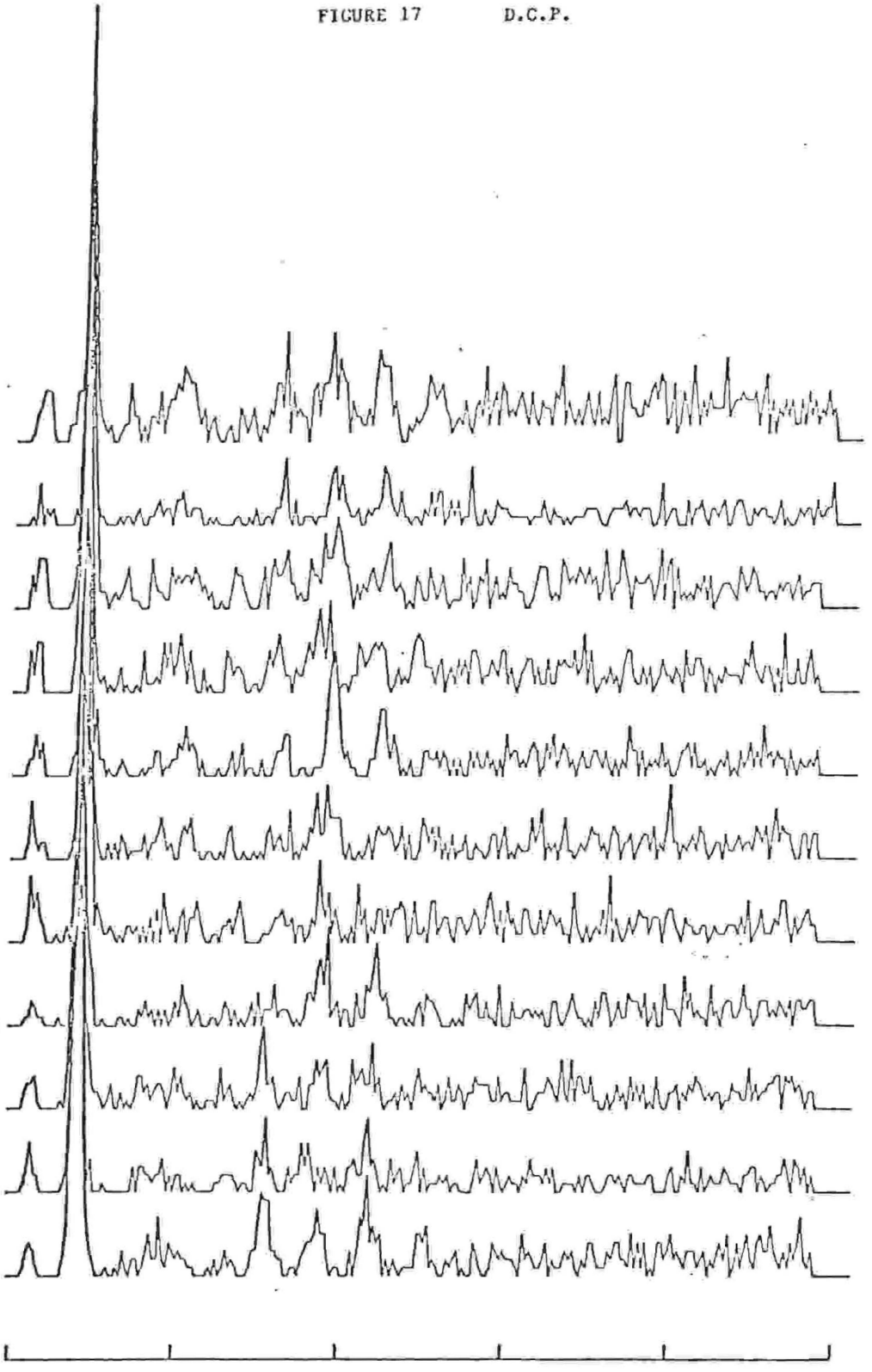

FIGURE 18

Average Evoked Potentials obtained from Animal 9, on eleven consecutive conditioning sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents microvolts, negative up, each calibration is equal to 16 microvolts.

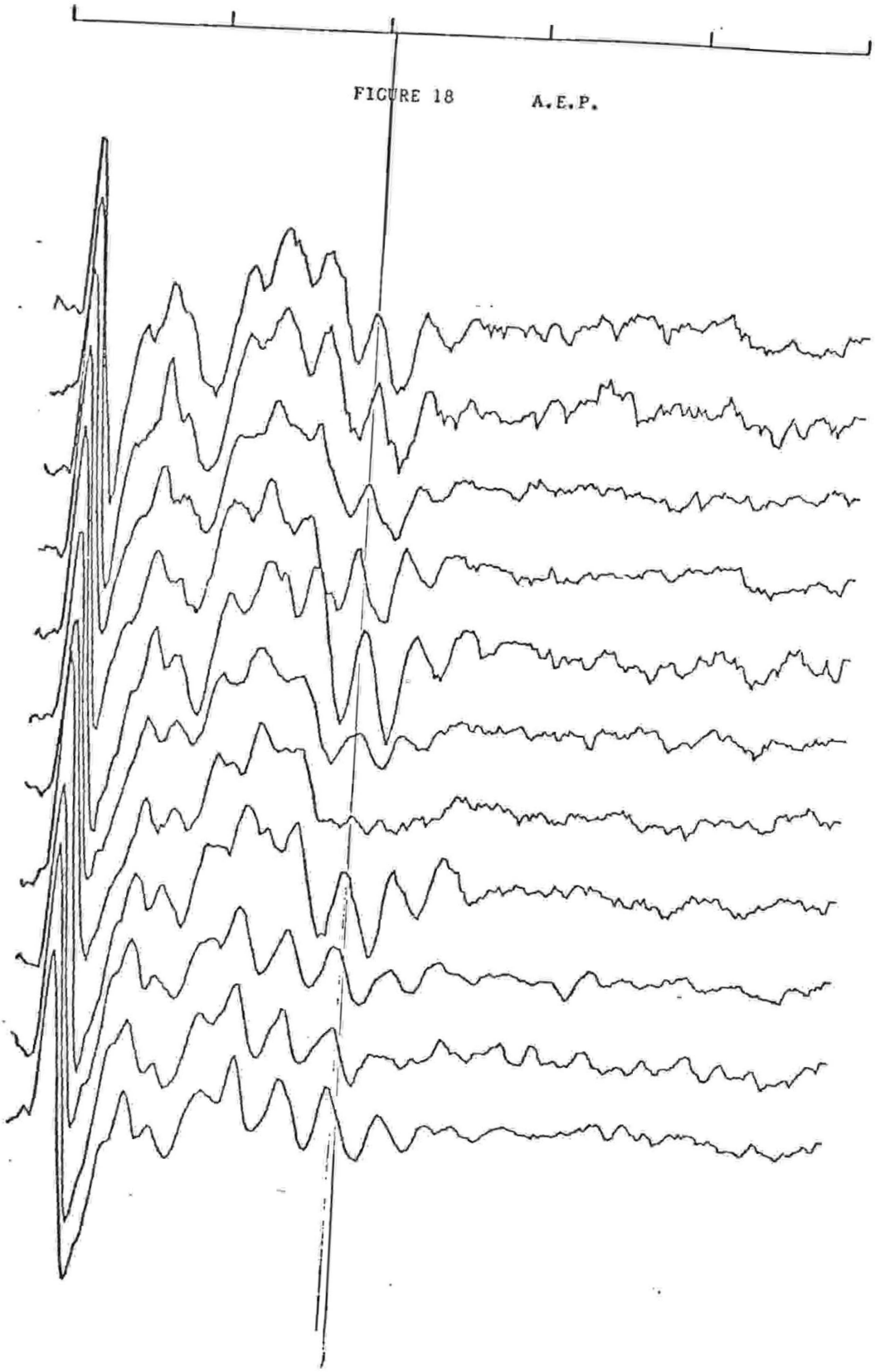

FIGURE 18 A.E.P.

FIGURE 19

Distribution of Criterion Potentials obtained from Animal 10, on nine consecutive sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents number of CPs, each calibration is equal to one CP.

FIGURE 19 D.C.P.

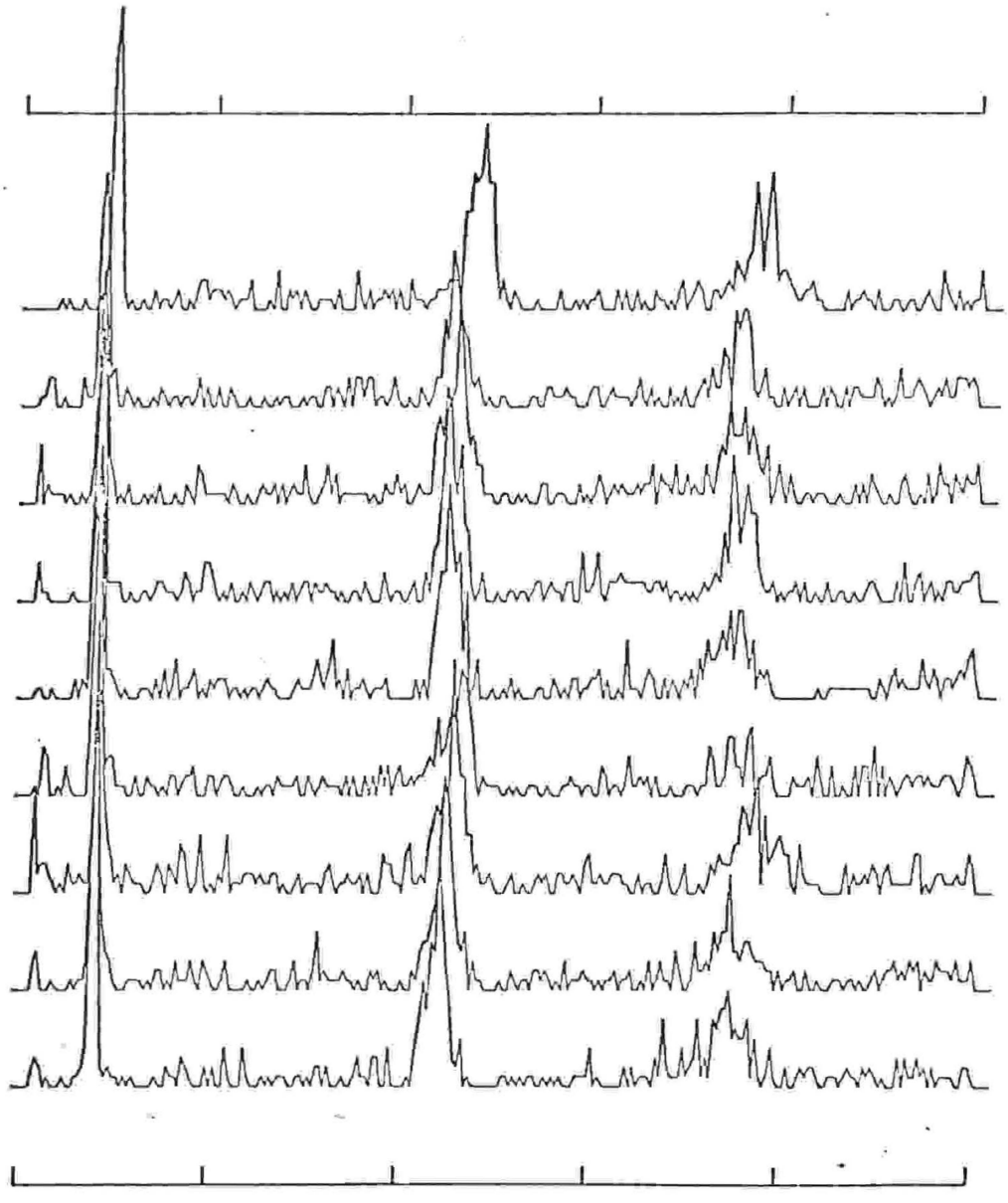

FIGURE 20

Average Evoked Potentials obtained from Animal 10, on nine consecutive sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents microvolts, each calibration is equal to 16 microvolts.

FIGURE 20 A.E.P.

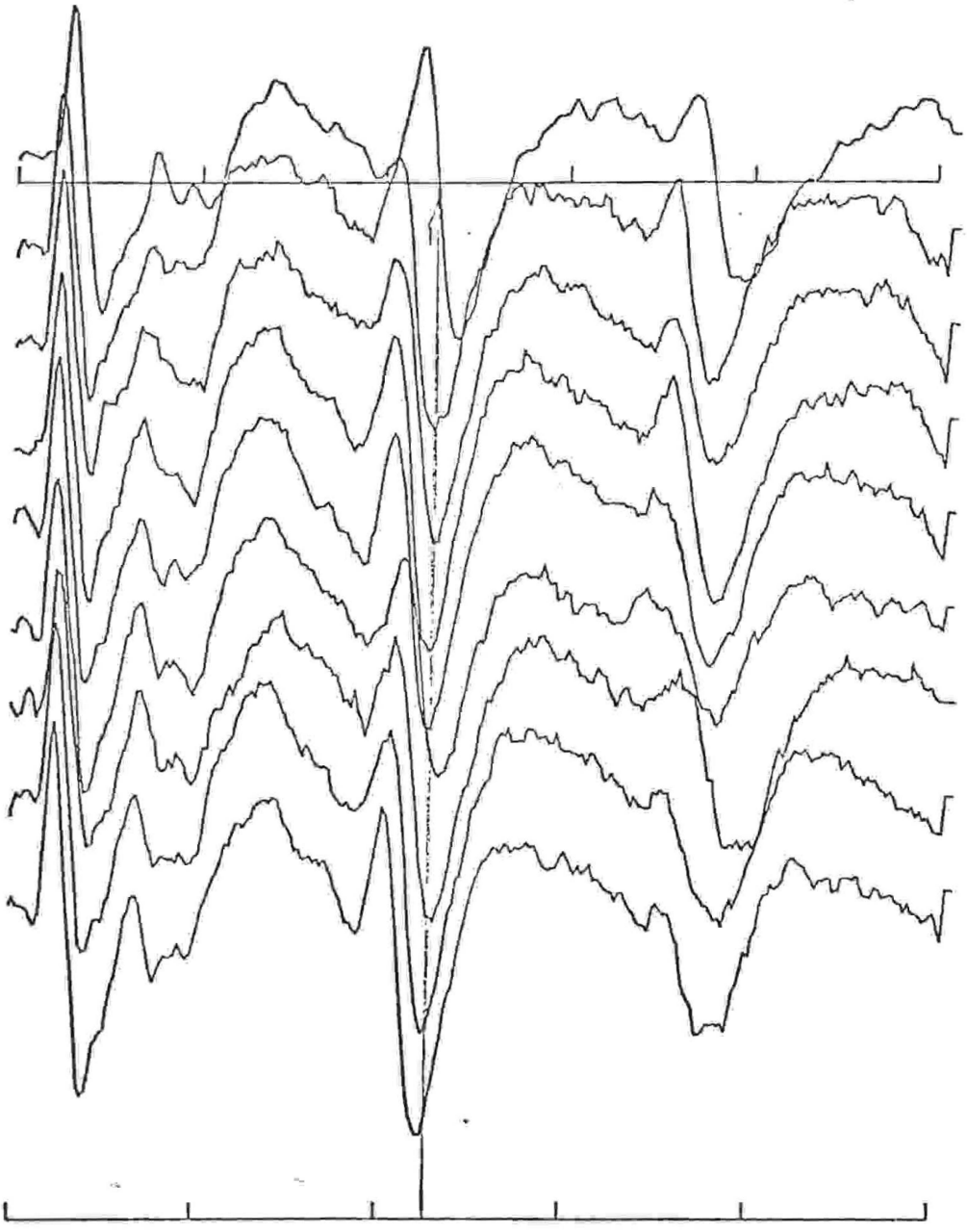

FIGURE 21

Distribution of Criterion Potentials obtained from Animal 11, one eleven consecutive sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents the number of CPs, each calibration is equal to one CP.

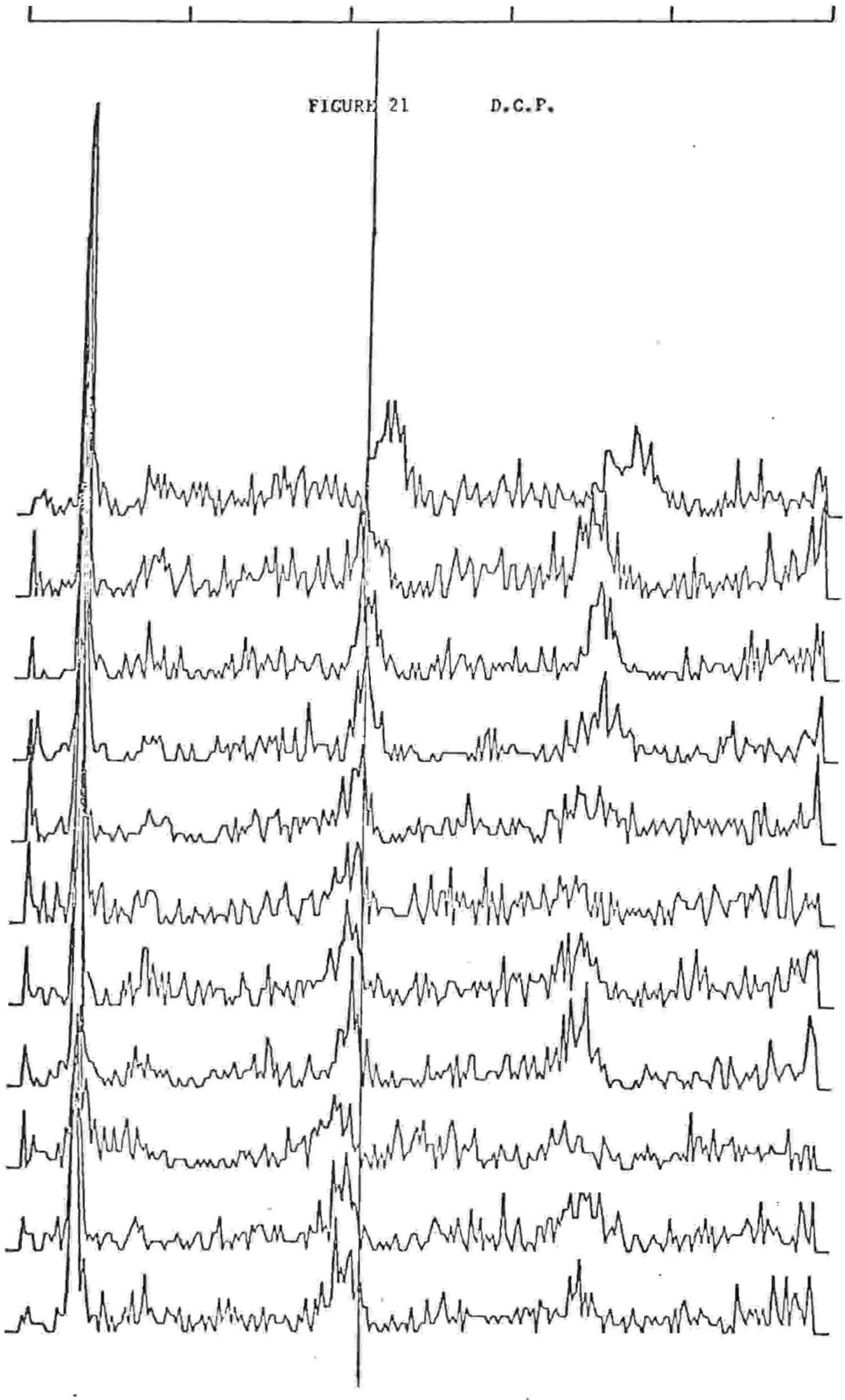

FIGURE 21 D.C.P.

FIGURE 22

Average Evoked Potentials obtained from Animal 11, on eleven consecutive sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents microvolts, negative up, each calibration is equal to 16 microvolts.

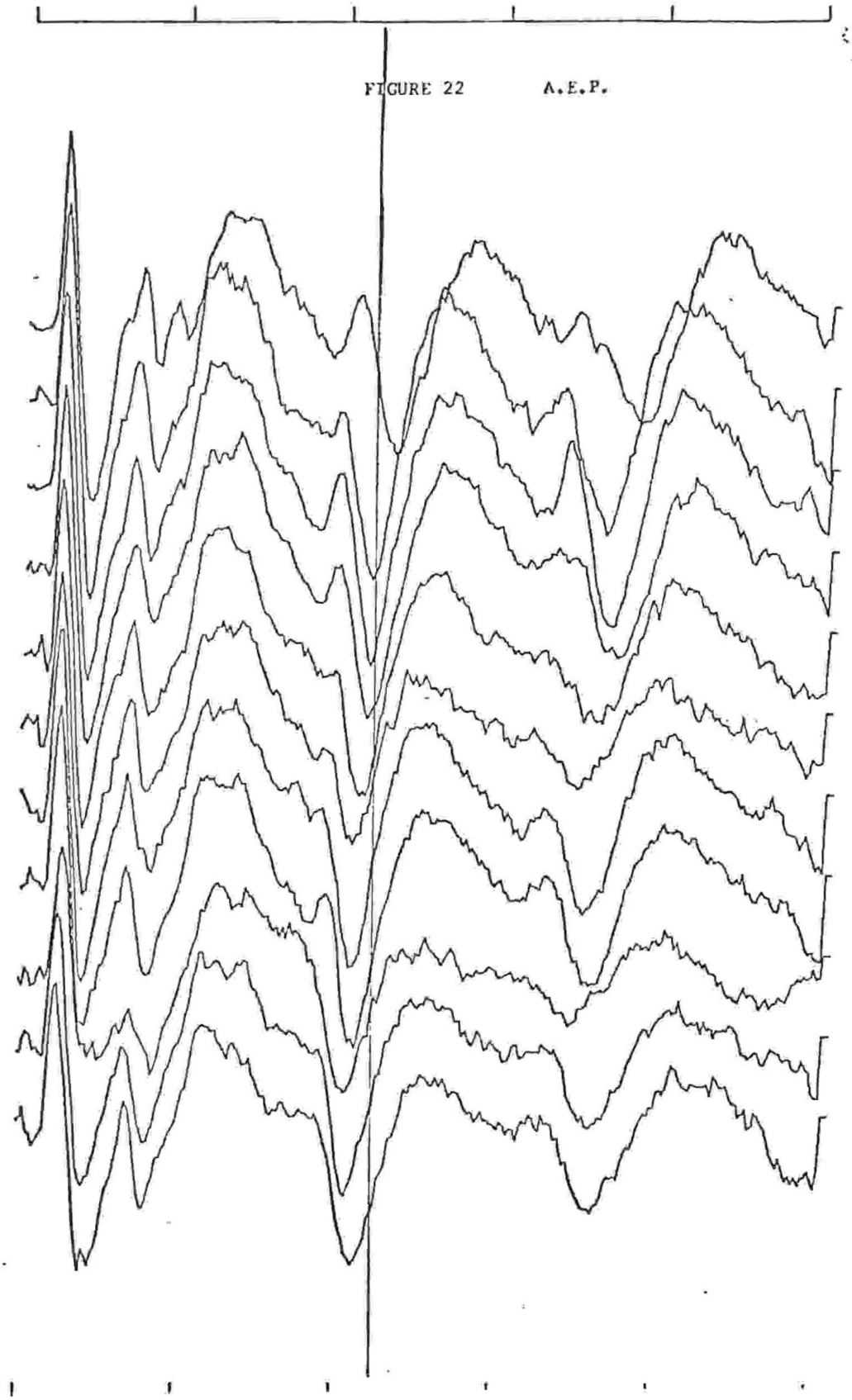

FIGURE 22 A.E.P.

FIGURE 23

Distribution of Criterion Potentials, obtained from Animal 12, on twelve consecutive sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents the number of CPs, each calibration is equal to one CP.

FIGURE 23 D.C.P.

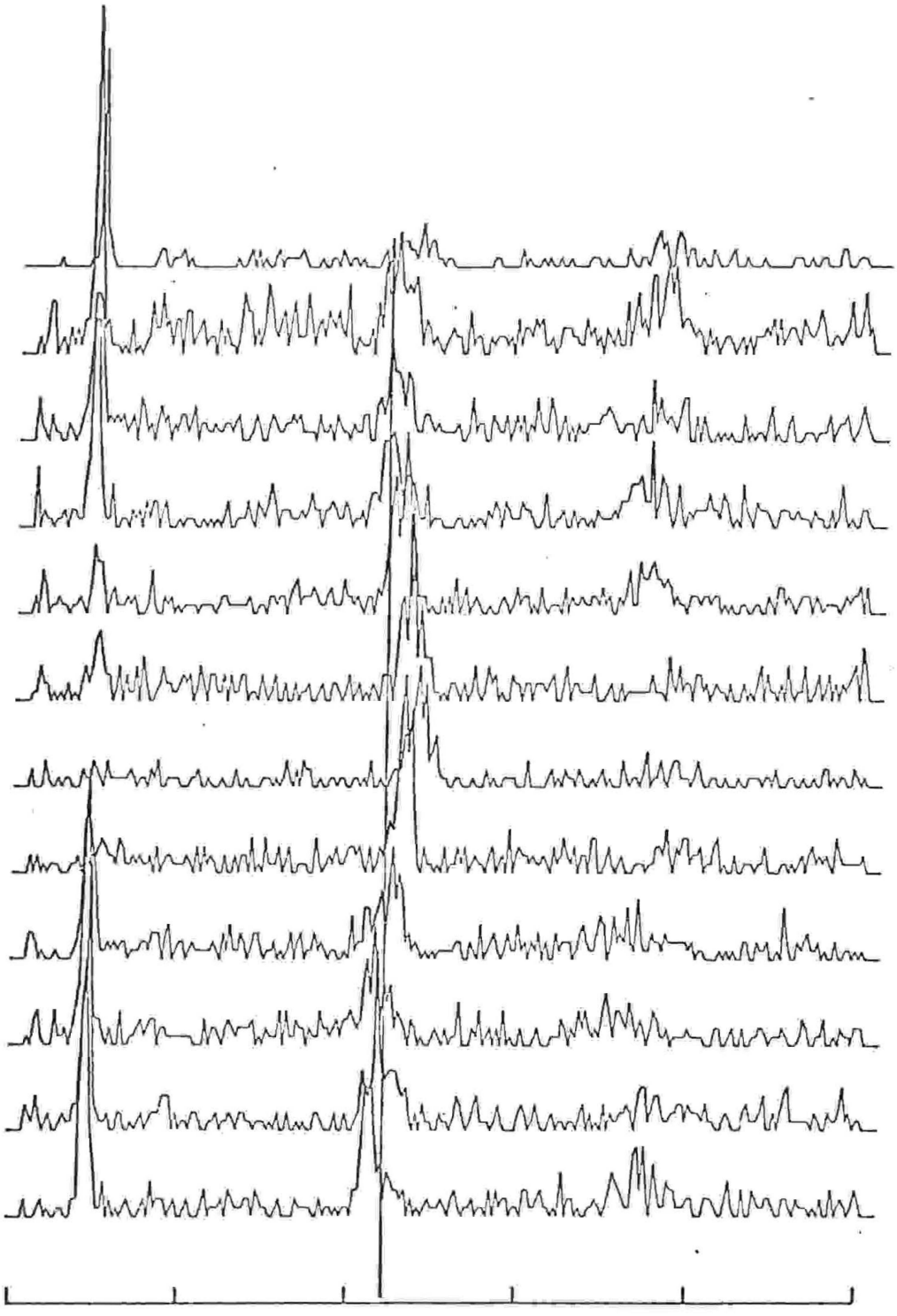

FIGURE 24

Average Evoked Potentials, obtained from Animal 12, on twelve consecutive sessions.

The horizontal axis represents time, each calibration is equal to 100 msec.

The vertical axis represents microvolts, negative up, each calibration is equal to 16 microvolts.

FIGURE 24 A.E.P.

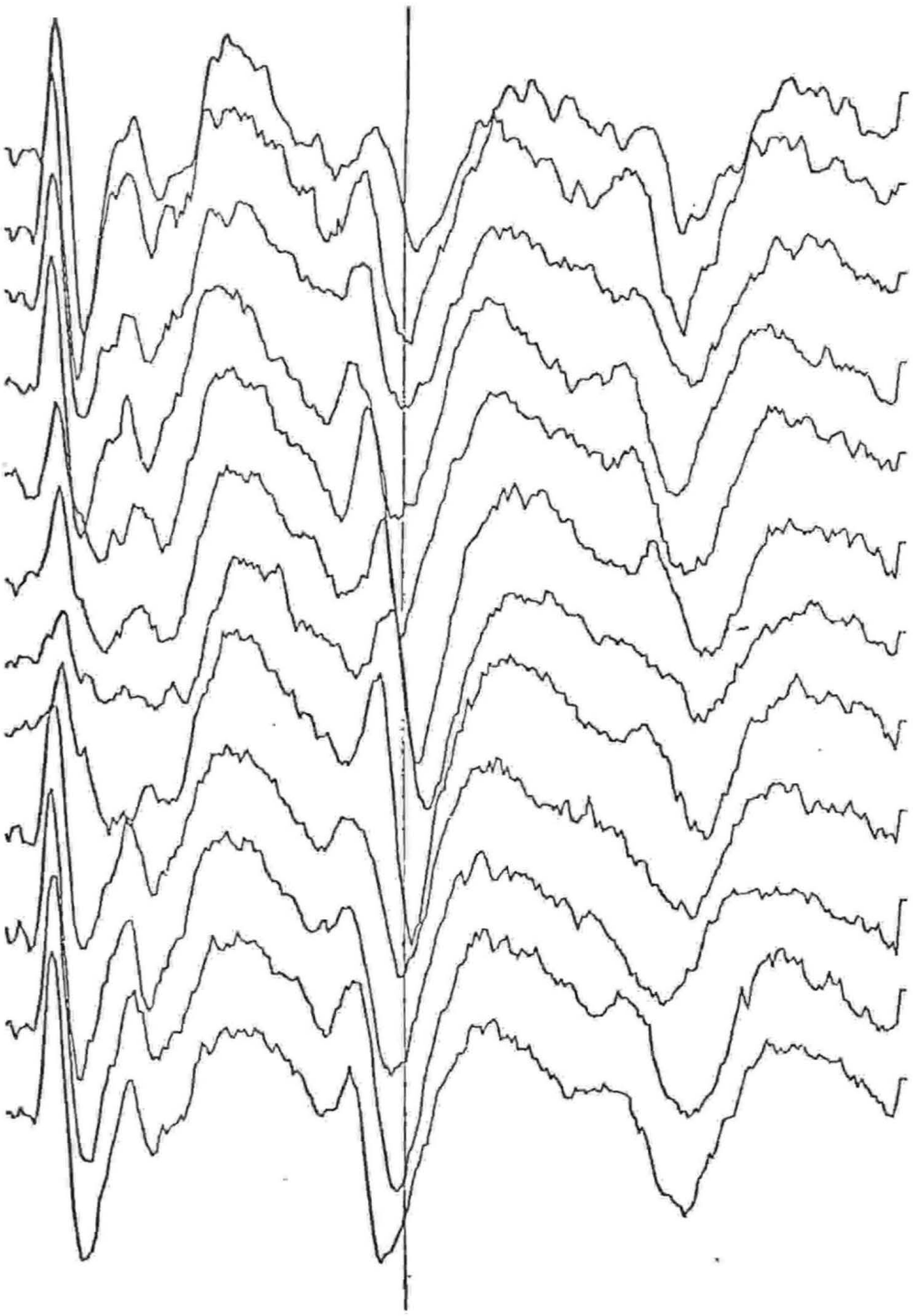

Figure 25

Relative Latency Shift of PL

VITA

Surname: MILLER Given Names: Harvey Adolphe

Place of Birth: Vita, Manitoba

Date of Birth: March 31, 1946

Educational Institutions Attended,
with Dates of Entering and Leaving:

UNITED COLLEGE, WINNIPEG	1964	to	1965
UNIVERSITY OF MANITOBA, WINNIPEG	1965	to	1967
UNIVERSITY OF WINNIPEG, WINNIPEG	1971	to	1972
UNIVERSITY OF VICTORIA, B.C.	1972	to	1977

Degrees:

B.A.	1969	University of Manitoba, Winnipeg

Honours and Awards:

Publications:

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name

June 7, 1977

date