EFFECTS OF PERCEPTUAL ISOLATION ON THE CNS

SOME EFFECTS OF PERCEPTUAL ISOLATION ON THE CENTRAL NERVOUS SYSTEM

by

HERMAN H. SAMSON, B.A.

A Thesis

Submitted to the Faculty of Graduate Studies

in Partial Fulfilment of the Requirements

for the Degree

Master of Arts

McMaster University

May 1966

MASTER OF ARTS (1966) McMASTER UNIVERSITY
(Psychology) Hamilton, Ontario

TITLE: Some effects of perceptual isolation on the central nervous system.

AUTHOR: Herman H. Samson, B.A. (University of Colorado)

SUPERVISOR: Dr. W. Heron

NUMBER OF PAGES: v, 147

SCOPE AND CONTENTS:

The hypothesis that the mechanism underlying the effects of perceptual isolation is the sensitization of the nervous system by "functional" deafferentation was tested by recording evoked potentials from the optic lobe of the pigeon before and after one eye had undergone pattern deprivation.

It was found that before isolation, the second peak of the evoked potential was reduced by background illumination, but after isolation, it was not. There was no clear indication of change in the absolute amplitudes of the potentials after isolation.

These results suggest that an interpretation of the effects of isolation in terms of denervation supersensitivity is oversimplified.

A second finding was that in the normal anesthetized bird, background illumination potentiated the photically evoked potential.

ACKNOWLEDGEMENTS

The author wishes to express his sincere gratitude to Dr. W. Heron for his assistance during this thesis. The help of Dr. G. Smith is also greatly appreciated. Thanks are also due to Drs. Morrison and Venderwolf for their friendly aid during this study.

Special thanks are due Miss J. Bentley for her assistance in the surgery and running of the experiments as well as typing of the manuscript; and, to Mrs. Rebecca Samson for her patience and support.

TABLE OF CONTENTS

TABLE OF CONTENTS (cont.)

INTRODUCTION

The term sensory deprivation has been used to describe conditions ranging from those of controlled experiments involving the complete deprivation of all sensory input to those experienced by the explorer during prolonged voyages across the arctic snows. In general, the term has the meaning of depriving the senses of vision, audition, and somesthesis of their normal environmental input. It has also been used in cases where only one of the senses is deprived, while the others receive "normal" stimulation from the environment.

In general, most of the experimental studies have tried to reduce patterned stimulation of the subject as much as possible. His movement is restricted by having him lie on a bed, or sit in a chair; auditory stimulation is controlled by having him wear ear plugs, or ear phones which emit a masking noise; visual stimulation is reduced by having him wear goggles, and somesthetic stimulation minimized by having him wear cuffs or gloves. There seems to be general agreement among many investigators that when persons are exposed to these conditions for long periods of time (a few days), there are many behavioral changes. Performance on intelligence tests may deteriorate (Scott

et al, 1959; Smith and Lewty, 1959; Davis et al, 1960). Perceptual function may become abnormal (Doane et al, 1959; Grunebaum et al, 1960; Freedman et al, 1961) and hallucinations may occur (Sexton, Heron, and Scott, 1954; Goldberger and Holt, 1958; Cohen et al, 1961). In addition, subjects may show motivational and emotional changes and even find the experimental situation intolerable after a few hours (Solomon et al, 1961). Some idea of the range and extent of the effects can be acquired from the excellent reviews in the literature (Biske, 1961; Kubansky, 1961; Solomon et al, 1961; Kenna, 1962).

As pointed out, sensory deprivation is a very broad term, and a useful distinction can be made between those studies which attempt to prevent any stimulation from reaching the receptors - sensory deprivation - and those which use unpatterned stimulation - perceptual isolation. Curiously, most of the evidence indicates that exposure to strong unpatterned stimulation seems to produce more drastic effects than does complete deprivation, except when radical procedures are followed such as those of Lilly (1956), who immersed his subjects in water. This may happen because there is more spontaneous activity of the receptors during sensory deprivation than during perceptual isolation (Kuffler, 1953; Arduini, 1961, 1963). Of course, it is also possible that the subjects are more

accustomed to sensory deprivation conditions than to those of perceptual isolation (Hebb, 1961); for example, they presumably have more experience of being in darkness than they have of being in diffuse light.

The wide range of phenomena produced by these experimental procedures implies that they must have widespread effect on the nervous system. What these effects are, however, is not clear. Two main theories have been advanced. The first and most obvious, was suggested by Bexton, Heron and Scott (1954) and developed more extensively by Lindsley (1961). It asserted that the effects of isolation were the results of changes in reticular activating system (ARAS). The second theory, advanced by Doane (1955), stated that the effects were due to an increased sensitivity of the nervous system.

The basic argument for the ARAS hypothesis is that since sensory input is reduced, input to the reticular system is reduced. The ARAS no longer exercises its usual regulatory influences on the cortex, which then functions abnormally. The main evidence that the ARAS is involved comes from electroencephalographic studies of isolated subjects. Both Heron (1957, 1961) and Zubek (1964) report that there is a change in the EEG of subjects after isolation. Specifically, slower frequencies appear in the alpha band in recordings from the parieto-occipital area, and there is

an increase in slow wave activity of the temporal lobe. A behavioral correlate of these EEG changes is that when subjects are placed in isolation they tend to sleep excessively during the early stages of the experiment, indicating lack of ARAS activity.

However, there are certain phenomena which cannot be accounted for by the ARAS hypothesis. First, all investigators report that there is an increase in general activity as the period of confinement is extended. One of the common explanations given for leaving the cubicle is that the subject feels too restless to remain. Second, as described later, isolation of a very small area of the skin can have perceptual effects. Though there is a good deal of specificity in the arousal system, it would hardly be expected to be so specific that preventing a small area of the forearm from being stimulated would bring about changes only in this small area, and no other observable changes.

The Doane hypothesis states that isolation produces changes in the nervous system which are similar to those produced by destroying the afferent nerve supply. It is well known that when a structure is surgically isolated, hyperexcitability results, the phenomenon being termed denervation supersensitivity. Doane postulated that sensory deprivation "functionally" denervates the

central nervous system.

There is a good deal of evidence to support Doane's hypothesis. After sensory deprivation there is a tendency for visual acuity to increase, visual after images to persist longer, the phi phenomenon more difficult to abolish, and the spiral after effect to be prolonged (Doane, 1955; Doane, Mahatoo, Heron, and Scott, 1959). In addition, the prevalence of hallucinatory activity and the increase in motor activity are compatible with such an interpretation. As mentioned before, it has been shown that isolating even a small area of skin on the forearm will cause an increased sensitivity to touch (Braunstein, 1957; Heron, 1961; Morrison and Heron, 1962; Afetanas and Zubek, 1963; Zubek, 1964). If Doane's hypothesis is correct, it would imply that the part of the central nervous system receiving input from the deprived sense should be found to be supersensitive following isolation. It is to this problem that the present thesis is addressed, but before turning to the experiment, it is necessary to consider the phenomenon of denervation supersensitivity in some detail.

Denervation Supersensitivity.

Supersensitivity involving denervated peripheral structures was observed by physiologists of the mid-nineteenth century. In 1885, Budge described an experiment in the rabbit, in which the sympathetic nerve supply to

one pupil was cut central to the superior cervical ganglion; and the other cut peripheral to the ganglion. After 48 hours, the postganglionically denervated pupil was more dilated than was the one denervated preganglionically. While both pupils had lost their sympathetic nervous supply, indirect denervation did not have the same effect as cutting the final nervous pathway to the pupil.

The "pseudomotor" phenomenon, another example of muscular contraction without benefit of motor nerve stimulation, was first described by Philipeau and Vulpian in 1863. The tongue of a dog was deprived of its motor nerve supply by section of the hypoglossal nerve. Following degeneration of the distal end, the authors stimulated the chorda tympani, a nerve which contains sensory, secretomotor, and vasomotor fibers, but no motor fibers supplying the tongue. While stimulation of this nerve trunk will not normally affect the tongue muscles, stimulation after deafferentation causes them to contract.

A wide variety of similar phenomena have subsequently been described, and the first partial explanation of the mechanism was given in 1922 when Frank, Nothmann, and Hirshman-Kaufmann demonstrated contraction of chronically denervated skeletal muscle when the drug acetylcholine was applied. Since that experiment, the mechanism of denervation supersensitivity in the periphery has been the

subject of intensive work by numerous physiologists, and is a well documented neurophysiological phenomenon. The work up to 1949 is admirably reviewed by Cannon and Rosenblueth, who contributed greatly to our understanding of the phenomenon (Cannon and Rosenblueth, 1949). Cannon's Law of Denervation best summarizes the understanding of the mechanism at that time"

> When in a series of efferent neurones a unit is destroyed, an increased irritability to chemical agents developes in the isolated structure or structures, the effects being maximal in the part being directly denervated (1949, p. 185).

Thus the pseudomotor type of phenomenon is due to the presence in denervated tissue of the nervous transmitter substance from sources other than the motor nerve terminals. While these transmitter sources are not normally adequate to stimulate the tissue, after denervation the increased sensitivity results in a response.

Subsequent work, reviewed by Thesleff (1960) has demonstrated that the increased sensitivity of denervated tissue is due to a spread of the subsynaptic substance beyond the normal limits of the nerve terminal structure so that in the case of denervated skeletal muscle, for example, the entire cell surface becomes an "end plate" (Axelson and Thesleff, 1959; Katz and Miledi, 1964a, 1964b).

The advances in the understanding of the peripheral mechanism made in the early thirties suggested that a

similar phenomenon might be found in the central nervous system. Thus in 1939, Cannon and Haimovici showed that spinal motor neurones below the level of a hemi-transection of the cord became abnormally sensitive to intra-arterial injections of acetylcholine.

More general effects have been demonstrated by Stavraky (1961), who describes the response of cats with unilateral ablations of parts of the cerebral hemispheres to intra-venous injections of acetylcholine. Such animals display tonic convulsions of the limbs, pupilary dilation, salivation, lacrimation, intestinal motility, and other signs associated with cholinergic activity. The responses are confined to the side contralateral to the ablation and can be evoked by doses of the drug too small to produce visible signs in normal animals. The supersensitivity in these cases developes slowly over a period of six weeks or so and apparently persists indefinitely.

Many other examples of supersensitivity following lesions of the CNS have been described, including responses to other drugs such as adrenaline and methacholine. The effects of depressants and convulsants are more controversial, but in general, denervated tissue gives a stronger response to the drug than normal tissue. (Stavraky, 1961).

Supersensitivity to drugs and electrical stimulation also occur when small portions of the CNS are isolated

from their surrounding neural connections. Isolated slabs of cerebral cortex are electrically silent following operation (Burns, 1951). Response of the slab to electrical stimulation and drugs gradually increases until a single electrical stimulus will provoke a "perpetual" epileptiform afterdischarge (Echlin, Arnett, and Zoll, 1952).

Echlin, Arnett and Zoll (1952), however, feel that the excitability of the slab immediately after isolation cannot be due to denervation supersensitivity. Arguing that supersensitivity depends on degeneration of the afferent neurones, they point out that in the acute slab this degeneration cannot have occured. Thesleff (1960) points out in his review that supersensitivity can occur without degeneration. By using Botulinum toxin, which blocks the release of acetylcholine by the end button, supersensitization can be obtained in skeletal muscle. Therefore, whether or not the initial change in responsiveness of the slab is due to supersensitivity is not clear. Sharpless and Halpern (1962), recording from chronically isolated slab, found that after ten to fourteen days of isolation the slab became hyperreactive to electrical stimulation and showed epileptiform discharges. While they found that the slab would give an epileptiform response immediately after isolation, the intensity of the stimulus necessary to produce the same response after fourteen days was greatly

decreased. A control area in the opposite hemisphere showed no change during the same time. This indicates that while the response of the slab immediately after isolation may or may not be due to sensitization, denervation supersensitivity lowers the response threshold greatly after a ten day period. Similar results on chronically isolated cortex have been reported by Grafstein and Sastry (1957) and by Morrell et al. (1960). Spiegel and his collaborators suggest that cutting the sensory pathways may cause increased sensitivity in structures higher up in the sensory system, which are not as drastically isolated as the slab technique. Thus Spiegel and Szekely (1955) report high voltage spike activity in somesthetic cortex four days after unilateral lesions in the thalamic somesthetic relay nucleus when small doses of metrazol or bulbocapnine were given intravenously. Control records from the somesthetic cortex of the intact hemisphere show no change. They also found that bilateral stimulation of the sciatic nerves produced larger evoked responses. It should be pointed out, however, that their published records of the effects on the evoked responses are not entirely convincing, and leave these results open to debate.

Chavez and Spiegel (1957) found in the cat that after section of the optic tract the activity of the lateral geniculate ganglion was at first reduced, but that

after a period of three weeks was characterized by high voltage waves which were similar in form to those of epileptic discharges. The authors account for these findings in terms of denervation supersensitization.

There is one study in the literature which usually is cited as evidence against the denervation supersensitivity hypothesis. In 1953, Eccles and McIntyre described an experiment in which denervation of a central structure appears to result in a subsequent decrease in responsiveness to the afferent pathway. The authors sectioned a dorsal spinal root in the cat distal to the root ganglion, and after thirty to forty days tested the massed action potential response of motor nerve branches arising from the deafferented segment when a single electrical stimulus was applied to the cut dorsal root. They found little or no monosynaptic response compared with similar test stimuli applied to the freshly cut dorsal root on the opposite side. Tetanic stimulation of the chronically sectioned afferents was found to restore monosynaptic response, and this potentiation lasted longer than post tetanic potentiation observed on the control side. There was also evidence that the response of the deafferented segment to stimulation of adjacent intact dorsal roots was enhanced. They conclude that the disused synapses had become less sensitive to transmitter and that synapses activated from adjacent

roots had undergone a compensatory enhancement of sensitivity. The experiment however, may not be a valid test of denervation supersensitivity. Stavraky (1961) points out that a single test stimulus applied to a cut nerve is not comparable with the usual procedure of applying drugs, so that the results are difficult to fit into the body of data on supersensitivity. He also criticizes the use of barbituate anesthetic which has been shown to depress the sensitivity of denervated tissue more than that of normal tissue (Drake and Stavraky, 1948; Sequint and Stavraky, 1957). Moreover, the results of Eccles and McIntyre's experiment are not necessarily inconsistent with the supersensitivity hypothesis. Since the spinal neurones show an enhanced response to adjacent segment stimulation and have abnormally prolonged post-tetanic potentiation, it is possible that in the single volley test the reduction in the efficiency of transmission of the fiber amputated dorsal root axons is great enough to mask the postulated supersensitivity. The authors admit that they cannot evaluate the influence of the observed decrease in average fiber diameter, which was of the order of ten percent. On the whole then, the evidence that denervation supersensitivity exists in the CNS is overwhelming. In fact, as Stavraky points out in his massive review of the evidence, it is possible that the hyper-responsiveness to drugs exhibited

by animals with motor cortex ablations or upper cord lesions may not be due to the release of inhibitory influences which normally dampen segmental reflex systems, as is generally believed. He feels that the evidence that supersensitivity occurs in deafferented spinal neurones, together with the long time period before these changes in reactivity occur, argue in favour of a mechanism based on genuine supersensitivity similar to that seen in peripheral structures.

Aims of the Thesis.

The literature indicates considerable physiological evidence for the existence of the phenomenon of denervation supersensitivity. There is psychological evidence suggesting that this phenomenon may be involved in the effects produced by perceptual isolation. However, all the physiological work is based on experiments in which denervation has been achieved either by surgical interference or by drugs. This thesis proposes to investigate whether "functional" denervation can affect the sensitivity of the nervous system.

In the experiments that follow, we prevented pattern vision instead of preventing all visual stimulation, since, as pointed out earlier, the behavioral evidence suggests that perceptual isolation has more profound effects than sensory deprivation. The visual system of the pigeon was

used, since anatomical studies suggest that there is complete decussation of the optic nerves at the chiasma, and that the neurones from each eye have direct endings only in the contralateral tectum (Ramon y Cajal, 1943). There is physiological evidence of an ipsilateral response which occurs after complete isolation of the ipsilateral tectum from the rest of the brain except for the optic tract (Rougeul, 1957), and this indicates that the crossing in the optic chiasma is not complete. However, these ipsilateral effects are small, and taken with the anatomical data suggest that there are very few uncrossed fibers.

The bird, therefore, seems a reasonable subject to use for this experiment. By occluding one eye, the contralateral optic tectum should be considerably affected and the ipsilateral one very little. By implanting electrodes in both optic lobes, the responsiveness of the two optic lobes to flashes of light can be tested, before and after one eye is deprived of pattern vision.

METHOD

Subjects.

The subjects were male White King pigeons, ranging in weight from 450 to 600 grams.

Operative Procedures.

Operations were done under Nembutal anesthesia (40 mg/kg). The animals were placed in a Kopf sterotaxic instrument equipped with a chicken adapter. The skull was positioned with reference to three points; the inter-aural line of the ear bars, the separation of the maxillary bone from the premaxillary bone on the beak, and the top of the skull at the midline. By using the distance from the inter-aural line to the beak point (the X distance), and the distance from the top of the skull to the beak (the Y distance), the tangent of a set angle phi (see Fig. 1) could be approximated and the head positioned so that a standard reference plane was achieved for all preparations (the tangent of phi was 1.554, which was an arbitrary choice).

In the acute experiments, a craniotomy over one hemisphere was performed or holes trephined at one of three locations, as appropriate. The electrodes were lowered by

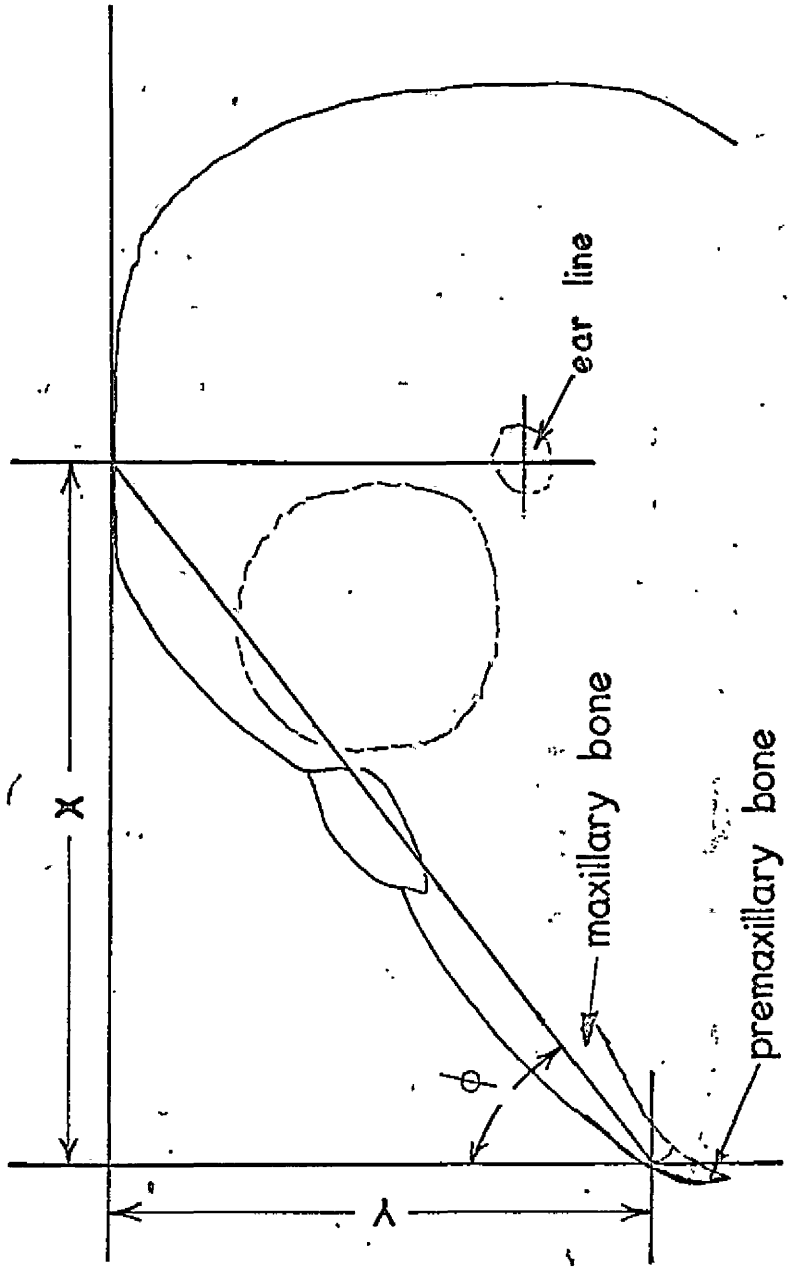


Fig. 1 Illustration of the distances measured for setting the skull in a standard reference plane.

means of the stereotaxic micromanipulator.

Chronic operations were done in the same way, except that all precautions were taken to keep the procedure sterile. After placement, the electrodes were attached to the skull by means of stainless steel jeweler's screws and acrylic dental cement. The wound was closed and plastic rings, as described below, were placed around the animals' eyes. Penicillin (20,000 - 30,000 units) was administered and the animals were given at least ten days to recover before the recording sessions. Additional antibiotics (Penicillin, Albamycin, and Streptomycin) were given over the first four days following the operation.

Isolation and Stimulus Presentation.

The eye was occluded by fitting a disc of translucent plastic in the ring fastened around the orbit. For the first experiments, the type of ring described by Catania (1963) was used. However, it was found that this ring had too small an inside diameter, so that movement of the eye muscles was hindered. Also, the rings were too deep, preventing normal ventilation. As a result, the corneas often became opaque and eye infections frequently developed.

It was found that satisfactory rings could be made from Perspex tubing with an outside diameter of 22 mm, inside diameter of 15 mm, and a height of 3 mm. These

were attached to the animal by dental cement placed on the feathers above and below the orbit. The ring was set away from the head so that air could pass under it (see Fig. 2, a and b). The inside diameter of the rings was larger than the eye orbit, so that movement of the lids or eye muscles was not restricted. Also, since the rings were only 3 mm deep, they did not restrict the visual field to any great extent, and the animals seemed to be able to avoid obstacles and obtain food without any problem.

The rings also provided a base to which the stimulus presentation unit could be attached, thus allowing control of the stimulus. This was essential, since it had been noticed in preliminary experiments that the position of the stimulus was very crucial in determining the amplitude and waveform of the evoked potential; a small movement of the stimulus light would change both the amplitude and the waveform of the response. By using the rings and a coupling system the position of the stimulus light could be kept constant for all recording sessions (see Figs. 3 and 4).

The light flashes used to evoke the potentials were generated by a Grass PS 3 photostimulator unit. The stimulator lamp was modified by placing a funnel, coated inside with aluminum paint, over it, which prevented light scatter

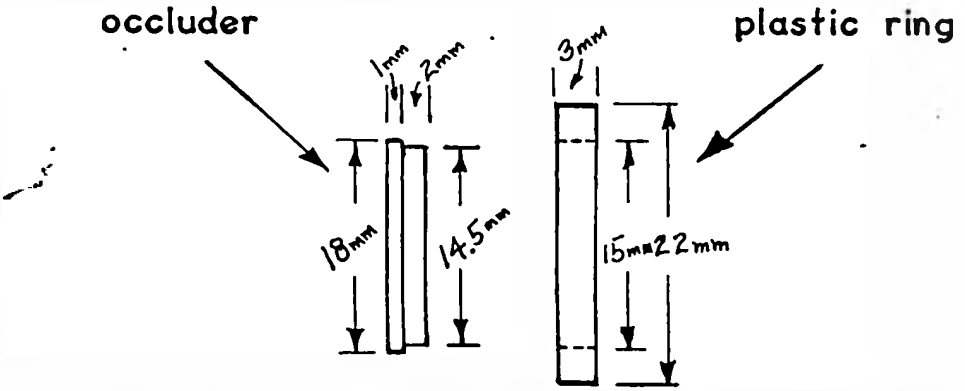


Fig. 2a The dimensions of the plastic ring and occluder and a side view of the birds head with the occluder in place.

Fig. 2b A front and side view of the ring in place on the animal.

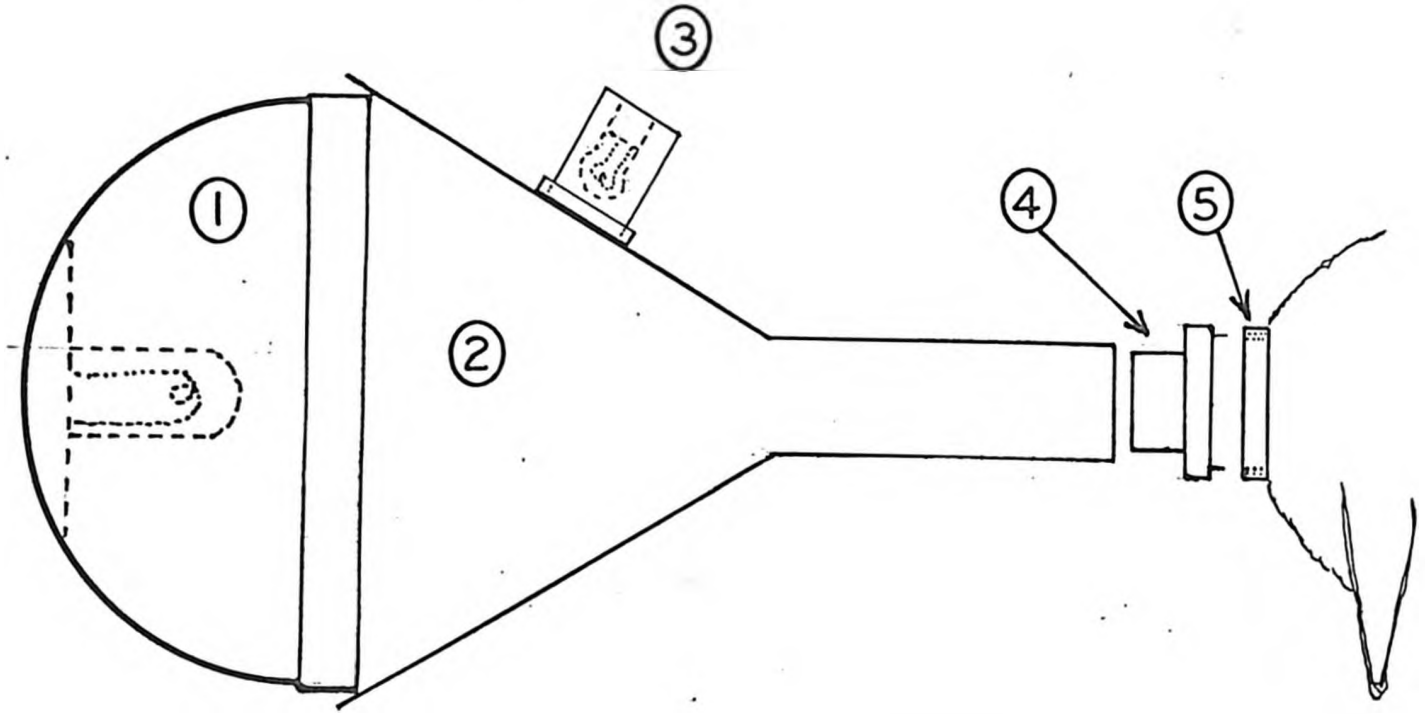


Fig. 3 Schematic diagram of stimulus presentation unit. 1. Flash unit of photostimulator. 2. Funnel placed over the flash unit. 3. Background illumination lights. 4. Adapter and coupling unit. 5. Ring attached to birds head.

a

b

Fig. 4 a. Coupling unit placed on plastic rings.

b. Stimulus presentation unit positioned with coupling unit.

(the intensity of the flash was approximately 1000 ft. lamberts). The small end of the funnel fit the coupling system as shown in the figures.

Controlled background illumination was supplied by four six-volt lamps set in the funnel (see fig. 3), which were run in parallel from a twelve volt battery supply and gave a background intensity of approximately 100 ft. lamberts.

Electrodes.

The electrodes were made from stainless steel insect pins (size 00) which were soldered in miniature connector pins. They were insulated using a modification of the method described by Burch and Myers (1962). The pins were first cleaned of all grease and oil by complete immersion in toluene. They were insulated by slowly immersing and withdrawing them from Insul-X (Insul-X Products Corporation). The electrodes were allowed to dry for at least 24 hours and were tested by passing a small current through the electrode while it was immersed in a salt solution. If there were any breaks in the insulation, small bubbles were seen on the electrode shaft. Any electrode which showed any indication of a break in the insulation was rejected.

Recording Procedure.

All potentials were amplified by 2 Grass P 5 AC amplifiers (amplification - 28K, filters: Low - 7CPS, High - .1KC) and were displayed on a Tektronix 502 oscilloscope. The signals were also fed into a Mnemotron CAT computer which both plotted and printed out the data. The computer was set so that the recording resolution of the amplitude of the potentials was within plus or minus 0.2 microvolts. The analysis time of the computer allowed each bin a storage time interval of 8 msecs. When two channels of the computer were used, the time from the onset of one bin till the onset of the next bin was still 8 msecs., but the data accumulation time for each bin was 4 msecs. This is due to the time sharing mechanism of the computer. With this time analysis setting, independent of the number of channels used, the amount of error in latency determination was plus or minus 4 msecs.

The X-Y recorder was set so that a full excursion of the Y axis could be made in the inter-bin readout interval. The error of measuring the computer X-Y plots is dependent on the amplitude of the individual potential in relation to the amount of magnification of the X-Y recorder and is specified for the individual records. The presentation of the stimulus, triggering of the CAT, and the start of the sweep of the oscilloscope were all

controlled by Tektronix pulse and waveform generators which delivered a pulse every second. The number of stimuli presented were counted with an electronic counter.

In some experiments, photographic records were taken of the oscilloscope traces. This was done with a Grass movie camera run at a speed of 10 mm/second.

Histology.

Both paraffin and freezing techniques were used. For the paraffin sections, the animal was sacrificed and perfused by direct injection of 10 percent formalin into the left ventricle of the heart. The brain was removed and placed in the formalin fixative for two to three weeks. The tissue was dehydrated by the tertiary butal alcohol (TBA) method and embedded in Tissuemat (Fisher Scientific). The paraffin blocks were sectioned at 10 microns and the sections stained with Mallory's Triple stain.

When frozen sections were used, lesions were made at the electrode sites by passing an anodal current of 2 ma. for 15 seconds. Five days after the lesioning procedure the animals were sacrificed and perfused with a solution of 10 percent formalin and one percent potassium ferrocyanide. The brain was removed and placed in the formalin-potassium ferrocyanide mixture for one week. The tissue was then washed with distilled water and placed in

20 percent ethyl alcohol for another week. The sections were cut at 40 microns and stained with Mallory's Triple stain.

PRELIMINARY EXPERIMENTS

Anatomical Locations.

Since a stereotaxic atlas of the pigeon brain has not yet been compiled, it was necessary to determine the position of the optic tectum within the skull. The anesthetized bird's head was placed in the stereotaxic instrument in the position previously described. Monopolar recording, with the indifferent electrode in the frontal bone was used. When necessary, the hemispheres were removed by suction so as to allow measurement of the boundaries of the tectum. Three animals were used in this part of the study.

Results.

The tectum was found to extend from a position 3.5 mm lateral to the midline to 7.5 mm lateral to the midline. Its anterior boundary was 1 mm anterior to the inter-aural line and its posterior boundary was 7 mm posterior to the inter-aural line (see Fig. 5).

To investigate the tectal responses, three skull positions were chosen: an anterior position, 2 mm from the inter-aural line and 4.5 mm lateral to the midline, a medial position 3 mm posterior to the inter-aural line

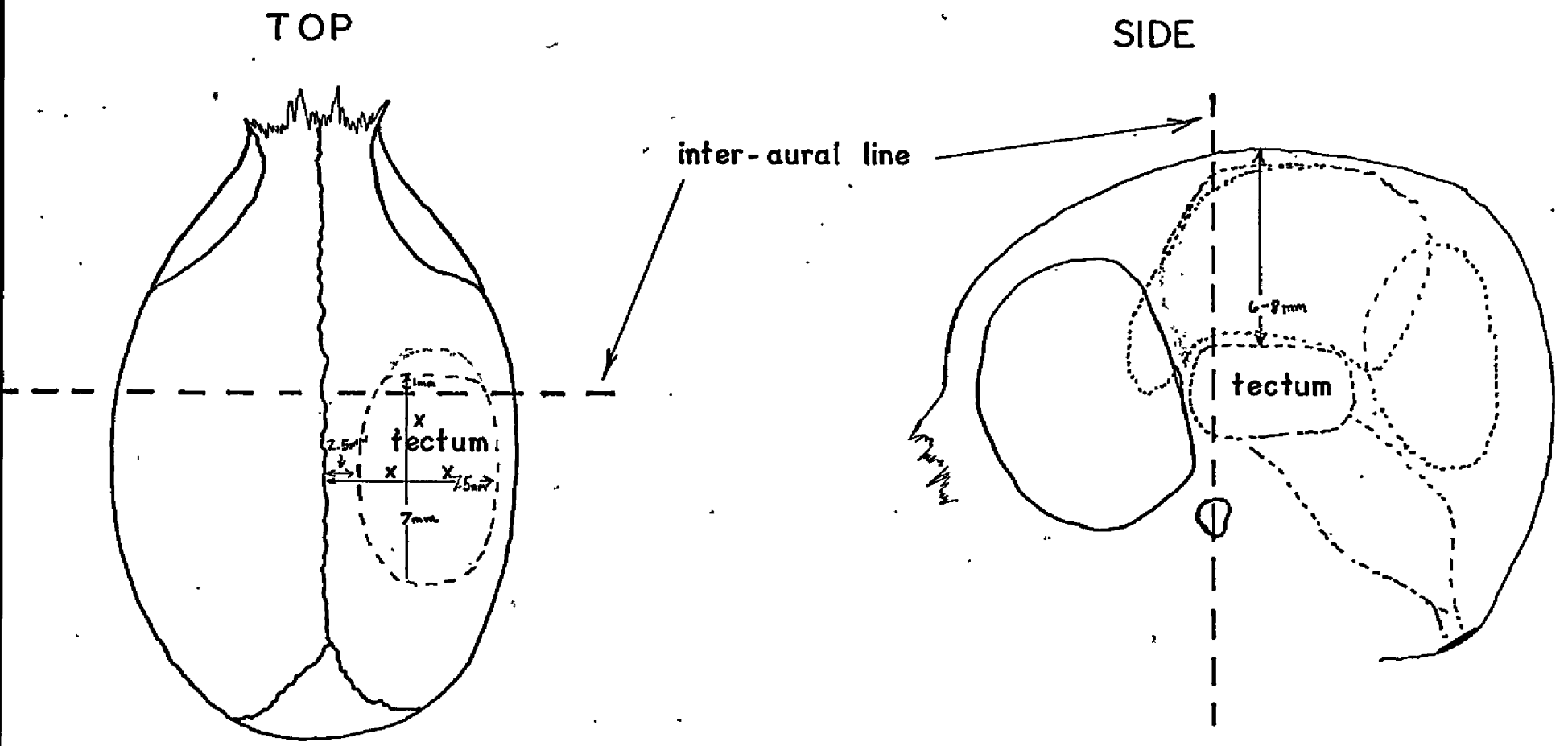


Fig. 5 Illustration of the tectum in relation to the skull.

and 4 mm lateral to the midline, and a lateral position, 3 mm posterior to the inter-aural line and 5 mm lateral to the midline. These three positions were chosen so as to investigate the responses from regions of the tectum which received projections from different parts of the retina (Hamdi and Whitteridge, 1954).

As the electrode was lowered through any of the three holes, the responses to flashes of light presented to the contralateral eye were first recorded 6 to 8 mm from the surface of the hemisphere, becoming maximal at 9 mm. It was a triphasic wave, positive-negative-positive, with a mean latency of 18 msecs. (range 16 to 20 msecs.) (see Fig. 6). Further lowering of the electrode to a depth of 12 to 14 mm from the hemisphere surface resulted in a reversal in polarity of the response and a slight modification in the waveform, but no change in latency (see Fig. 6). The amplitude of the reversed response was maximal at approximately 13 mm. The lateral coordinates were found to give larger responses with a more stable waveform than other electrode locations.

The reversal in potential as the electrode is moved through the various layers of the tectum is very much like that found in the mammalian cortex (Bishop and O'Leary, 1936). Histological examination showed that the electrode was above the level of termination of the optic nerve fibers

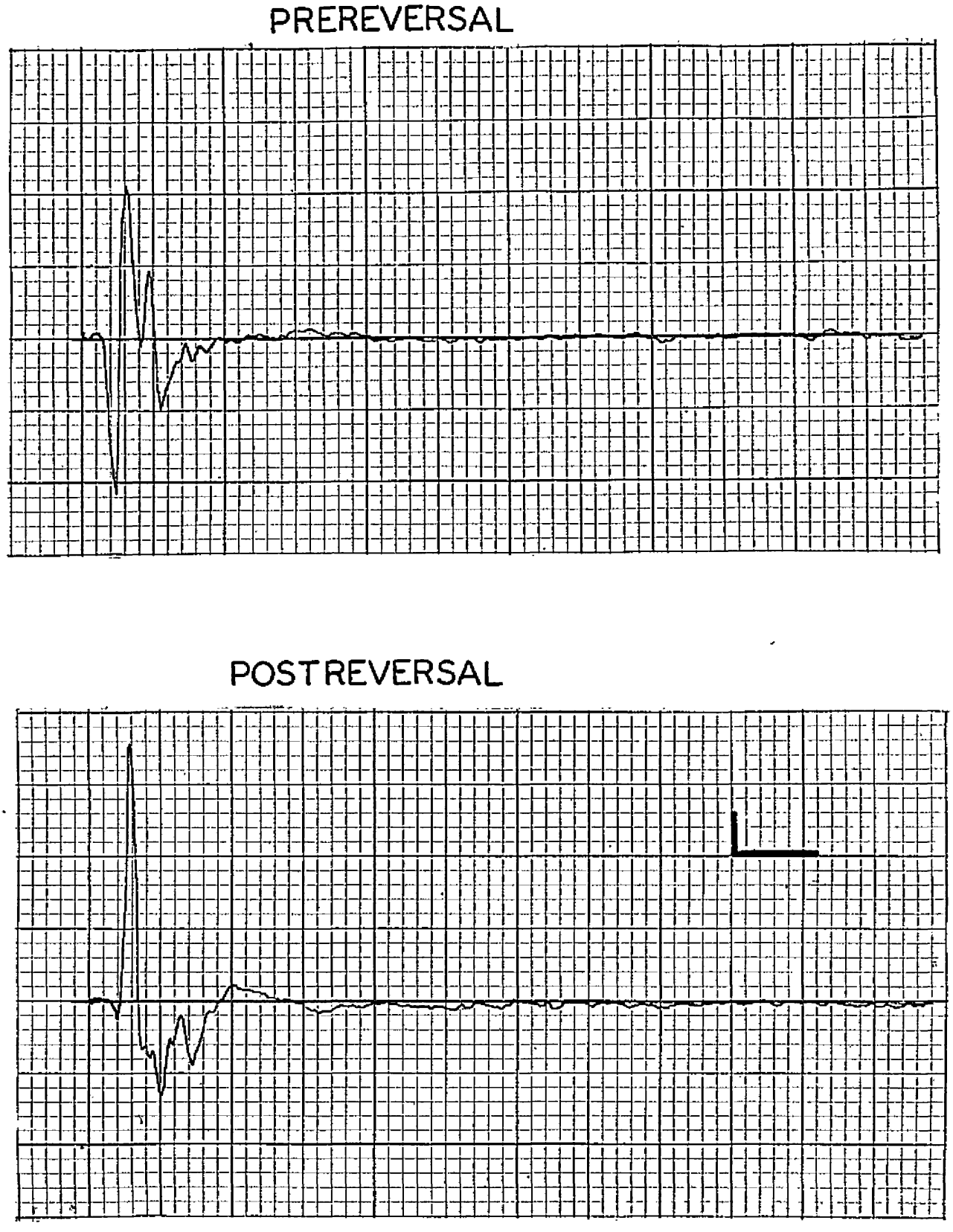


Fig. 6 Tectal response recorded before and after reversal of waveform. (Calibration: 50 microvolts, negative up; 50 msec., lateral electrode position)

while recording the pre-reversal response. After reversal, the electrode was found below the major divisions of the tectal layers (see fig. 7).

Deprivation Effects.

After suitable locations had been found in the tectum, the first study on the effects of perceptual isolation was made. The design was to measure the responses and then isolate one eye, and after a passage of time measure the responses again. If any changes were observed, the responses after the occluders had been removed for some time were to be measured to determine if the tectum would return to the pre-deprivation state.

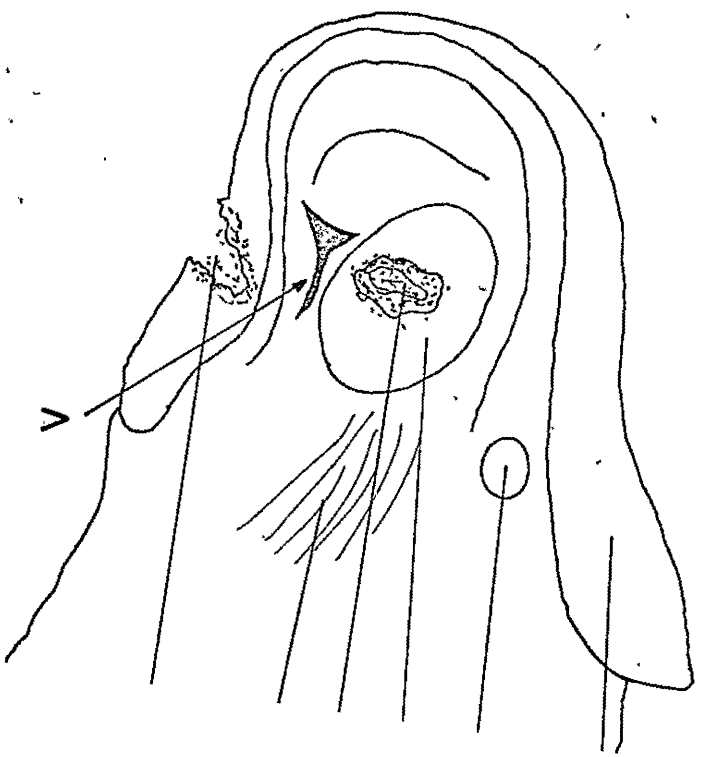
Method.

Chronic bipolar electrodes were implanted bilaterally in five animals. One pole of the electrode was placed in the medial location, and the other in the lateral, producing approximately 2 mm between the tips. Occluders as described by Catania (1962) were used.

One week after the operation, there were two successive days of recording sessions. In each session, one eye was tested with four runs of fifty single flashes, followed by a series of seven runs of fifty double flashes with inter-flash intervals of 10, 20, 40, 50, 70, 90, and 100 msecs., and the time between each run being two minutes.

Fig. 7 Histological location of electrode giving the largest responses before reversal (PRE ET) and after reversal (POST ET) of the waveform (LGN - lateral geniculate nucleus; ON - optic tract; TECT - tectal nucleus; TTT - tectothalamic tract; V - ventricle)

BIRD 2



The same procedure was then carried out on the other eye. After the second pre-deprivation session, one eye was occluded, and on the fifth and seventh days of isolation, DEP sessions were held. The occluders were then removed and the animals allowed seven days of "normal" vision before having similar recordings made on two successive days.

Results.

The double flash procedure failed to reveal any effects of isolation, and no increase in the amplitude of the response was found. In fact, there was a decrease in the amplitude of the evoked potentials obtained from both the isolated and control optic lobes. These results, however, are suspect since, as mentioned previously, use of the Catania type occluders resulted in infection of the eye and cataracts. However, there were some results bearing on the stability of the response which are relevant to the rest of the thesis.

Table one shows the average peak to peak response amplitudes (see Fig. 9) for each of the four single flash runs in the PRE sessions, from birds who had not yet developed any obvious infections. An analysis of variance found no difference in the average amplitudes from one run to the next. Also, as shown in figure eight, there was no change in the waveform of the response.

This experiment made it apparent that a more satis-

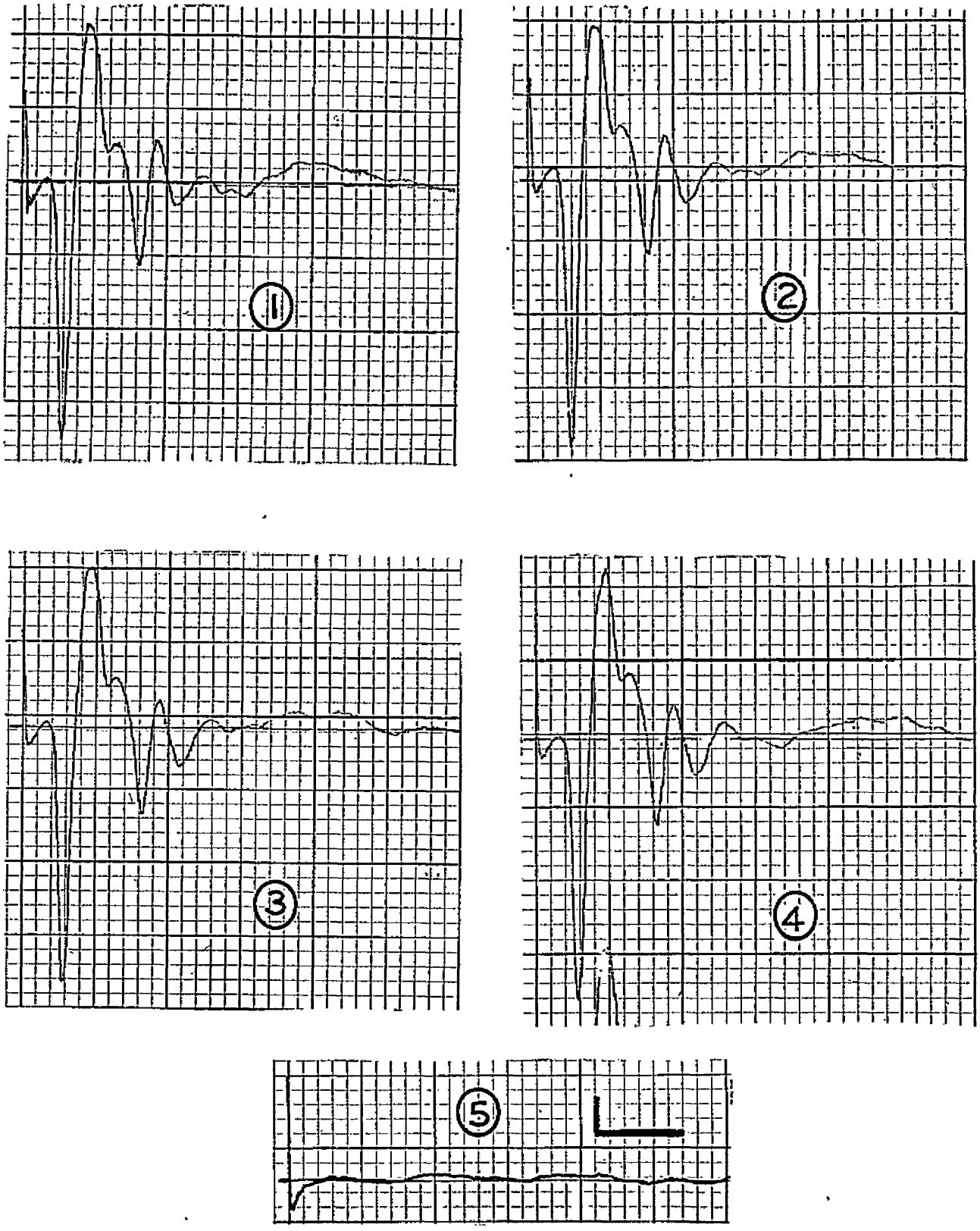


Fig. 8 Computer averages of four runs of fifty flashes per run (1-4). Computer average of 50 responses with no stimuli presented (No Stimulus Control, 5). (Calibration: .50 microvolts, negative down; 50 msec.)

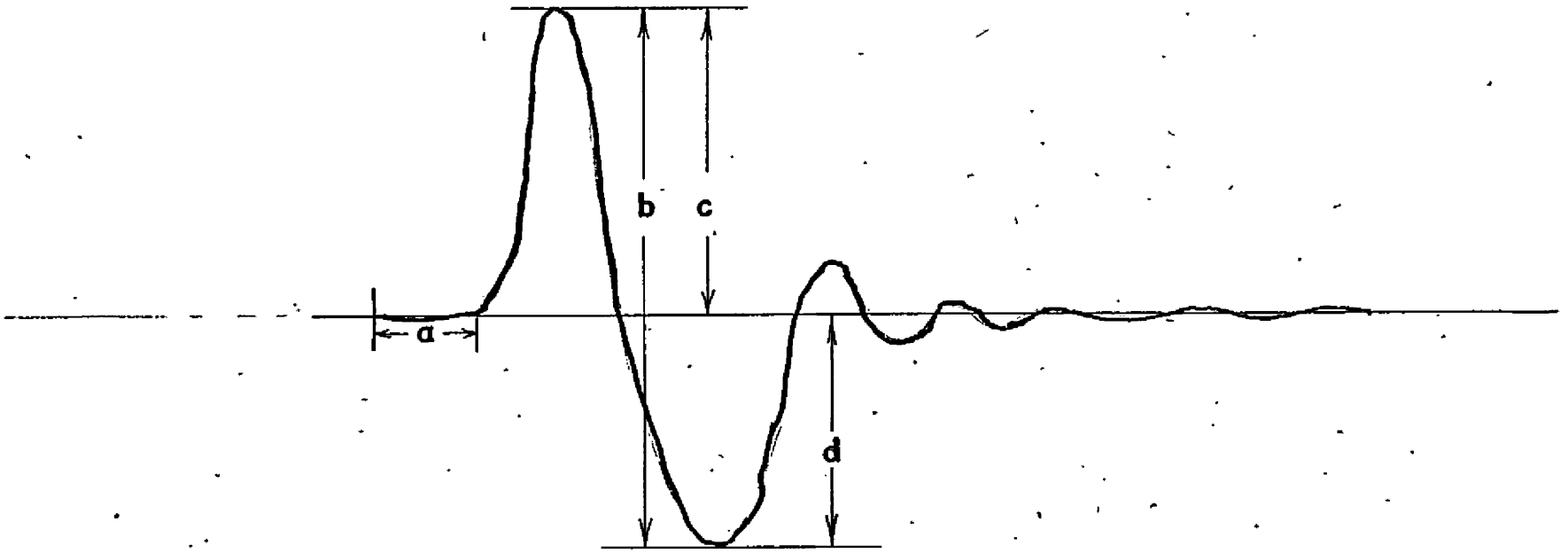


Fig. 9 A "typical" waveform showing the various measurements which were taken for analysis. (a - latency; b - peak to peak amplitude; c - amplitude of the primary (1st) peak; d - amplitude of the 2nd peak)

factory occluder system had to be developed before repeating the procedure. However, it was decided to first investigate a peculiar phenomenon which had been noticed when the electrodes were being implanted.

Table 1

Peak to peak amplitudes of the four single flash tests

run	1	2	3	4
animal				
30	60	59	65	55
42	85	75	77	67
2	432	456	432	436

POTENTIATION EXPERIMENT

Introduction.

In the preliminary experiments it was noticed that turning the room lights on or off during the implantation procedure resulted in a marked change in the amplitude and waveform of the evoked potential; the response appeared to increase in amplitude when the room lights were turned on, a potentiation effect. As the phenomenon of potentiation is controversial, and as the writer is not aware of any examples of potentiation occurring when a light flash is used as a stimulus, it was decided to examine these changes in more detail in acute preparations. Before presenting the procedure and results of these experiments, a brief review of the literature on the potentiation of the evoked response by background illumination is in order.

History of the Potentiation Phenomenon.

The phenomenon of potentiation was first reported for the anesthetized cat by Marshall, Talbot, and Ades in 1943. They found that when a conditioning flash was presented to one eye and followed with a test shock to the contralateral optic nerve, the cortical evoked potential from the shock was enhanced. Chang (1952a) made a more

thorough investigation of the effect and found that when the retina was steadily illuminated, the cortical evoked potential from geniculate stimulation was larger than when there was no retinal illumination. Chang concluded that the potentiation was due to a facilitation effect of retinal illumination on the geniculate, and rejected the possibility that illumination of the retina diminished its inhibitory effects on the geniculate, since the excision of the eye did not result in potentiation.

In 1956, Malis and Kruger repeated the Chang experiment. They used different levels of background illumination and found that the level of background illumination was related to the amount of potentiation seen. As the level of retinal illumination increased, the potentiation increased until a limit was reached, after which no increase in the amplitude of the response was found. They also reported that potentiation was not found over the entire visual receiving area, and that the location for potentiation within the visual cortex was not the same in different cats. Doty (1958) found that diffuse illumination of the retina would potentiate the cortical response from both geniculate and optic nerve stimulation in the monkey and cat.

While all investigators agree that potentiation occurs, the mechanism involved is not clear. A good deal

of evidence supports the view that potentiation occurs because tonic retinal influences are removed by background illumination, especially the work of Arduini and his collaborators (Arduini and Hirao, 1960; Arduini and Goldstein, 1961, 1963; Arduini and Pinneo, 1962, 1963). They found that the cortical evoked response to geniculate stimulation was enhanced both during retinal illumination and retinal ischemia. They also have confirmed the earlier results of Kuffler (1953), Granit (1955), and Barlow, Fitzhugh, and Kuffler (1957), who reported that diffuse retinal illumination decreases the amount of spontaneous activity of the retina. Arduini argues that potentiation occurs because of a decrease in the tonic activity of the retina, and finds the Chang effect when this tonic activity is depressed by background illumination or completely blocked by ischemia. His case is strengthened since, as pointed out earlier, Malis and Kruger found that the degree of potentiation varies directly with the intensity of retinal illumination and Arduini has shown (1963) that the amount of spontaneous activity of the retina is inversely proportional to the amount of background illumination. Arduini points out that Chang's report that excision of the eye did not result in potentiation was not confirmed by Posternak, Fleming, and Evarts (1959), who found potentiation of the cortical response to geniculate stimulation after section of the optic nerve. He argues that the difference in results may

have been due to injury discharges in Chang's preperation.

As Bremer (1961a, 1961b) has pointed out, however, and as Arduini himself agrees (1961), the mechanism of potentiation probably involves some diffuse subcortical systems, especially the reticular activating system. Chang (1952b) reports responses in the auditory cortex evoked by medial geniculate stimulation are enhanced by retinal illumination (though others, e.g., Arduini and Goldstein, 1961, have failed to confirm this). Also, Van Eyck (1963) reports that the vestibular response is similarly enhanced by retinal illumination, and Arduini (Arduini and Hirao, 1960) has reported that olfactory stimulation potentiates the cortical response to lateral geniculate stimulation. These experiments support the idea that nonspecific subcortical mechanisms may be involved.

In all of these studies, retinal illumination potentiates the response to electric shock. None of the investigators have reported enhancement of the response to light flash. It seemed worth while to make a brief study of the observation that background illumination appeared to potentiate the photically evoked response.

Method.

Three pigeons were used in this experiment. The lateral electrode location was explored with a monopolar

electrode, the indifferent electrode being in the midline of the frontal bone. The stimuli were presented to the contralateral eye. In one bird (#41) the series of stimulus presentation was five runs of 50 flashes without background illumination, followed by five runs of 50 flashes with background illumination. In the second bird (#5), five runs of 50 flashes with background were alternated with five runs of 50 flashes without background. In these two animals, the potentials were recorded with the electrodes above the point at which reversal occurred. In the remaining bird (#7), the potentials were recorded after reversal of waveform. For this bird, three runs of 50 flashes per run were given without background illumination. The three runs were followed by three runs of 50 flashes with background illumination. Then, after a ten minute interval, one more run of 50 flashes was taken in the dark. Another ten minute interval was inserted and then a final set of 50 flashes with background illumination was obtained.

Results.

Table two gives the results of the peak to peak amplitudes in microvolts (see Fig. 9), and Figures 10, 11, and 12 show the averaged responses for the three birds. There is no waveform change, and the amplitude of the responses during background illumination is increased.

TABLE TWO

Peak to Peak Amplitudes of the Evoked Potentials with and without background illumination.

Animal #41

NB Run		B Run	
1	120	6	150
2	130	7	160
3	115	8	160
4	110	9	150
5	130	10	158

Animal #5

NB Run		B Run	
1	336	2	392
3	340	4	460
5	400	6	420
7	356	8	460
9	352	10	424

Animal #7

NB Run		B Run	
1	586	4	636
2	577	5	617
3	555	6	630
7	568	8	613

Amplitudes expressed in microvolts; error of measurement: plus or minus 2.5 microvolts.

Fig. 10 The effect of background illumination on the evoked response in bird #41. Monopolar electrode at pre-reversal depth. Numbers 1 - 10 indicate order in which runs of fifty flashes were presented. Inter-run interval two minutes. Calibration: 50 microvolts, negative up; 50 msec.

NO BACKGROUND

BACKGROUND

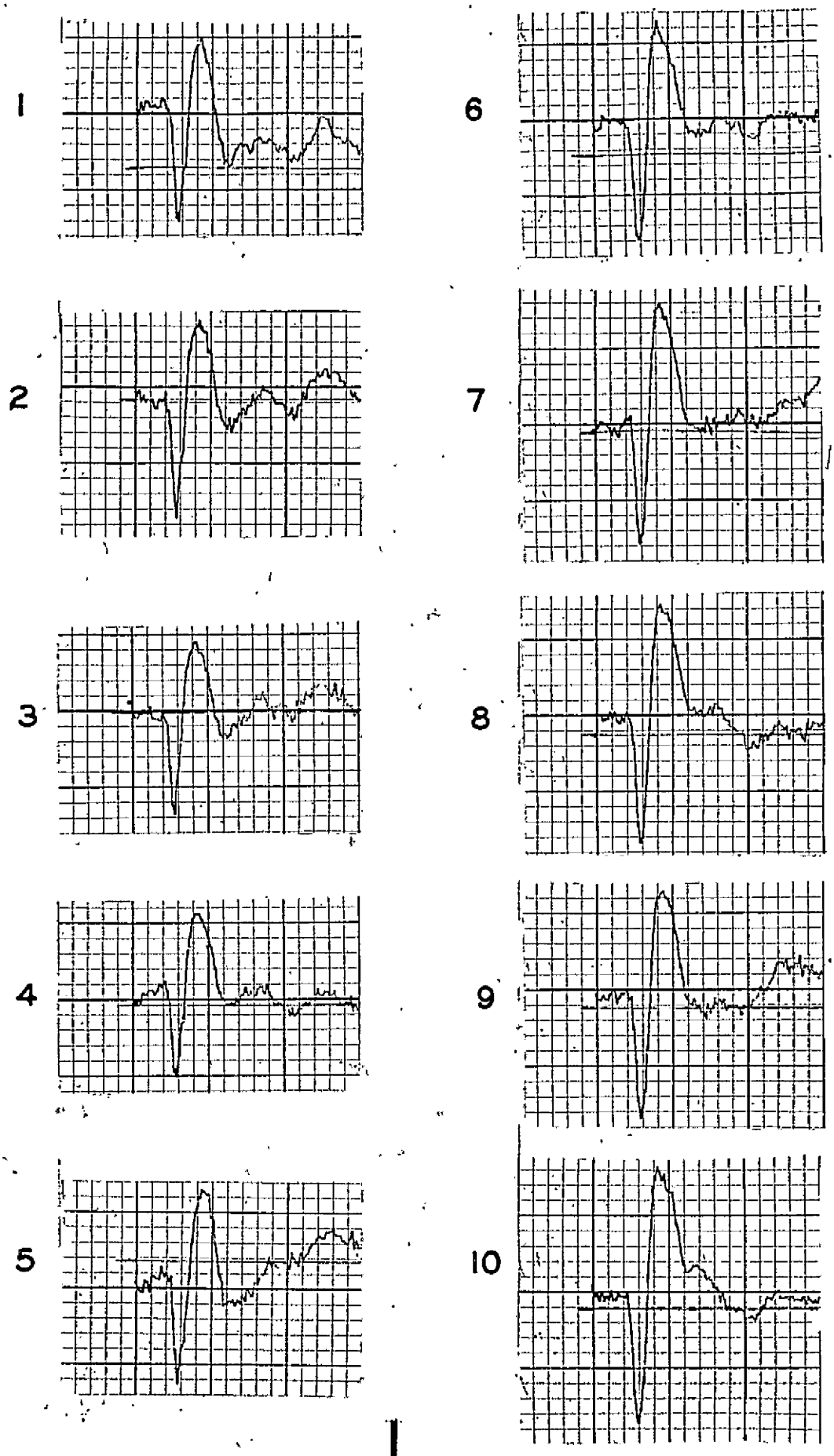
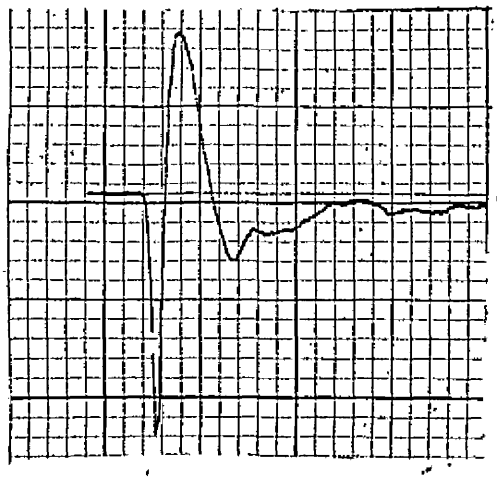


Fig. 11 The effect of background illumination on the evoked response in bird #5. Monopolar electrodes at pre-reversal depth. Numbers 1 – 10 indicate order of runs. Inter-run interval two minutes. Calibration: 100 microvolts, negative up; 50 msec.

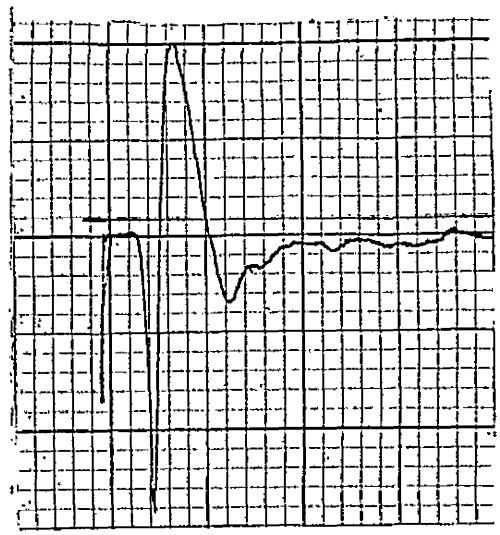
NO BACKGROUND

BACKGROUND

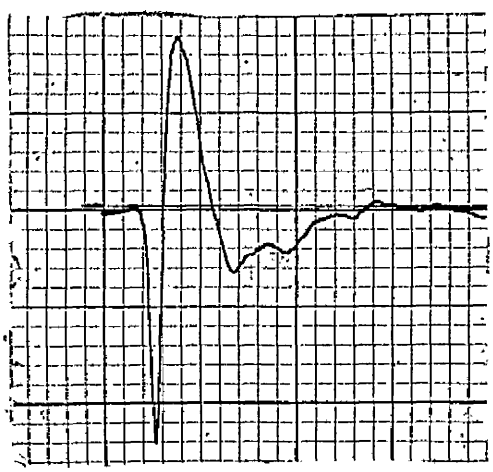
1



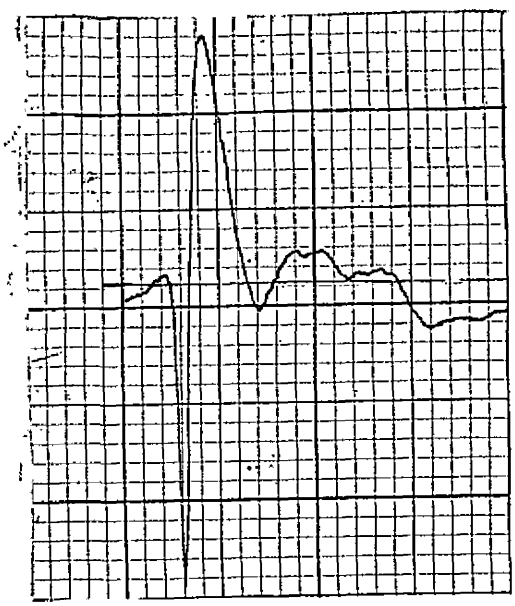
2



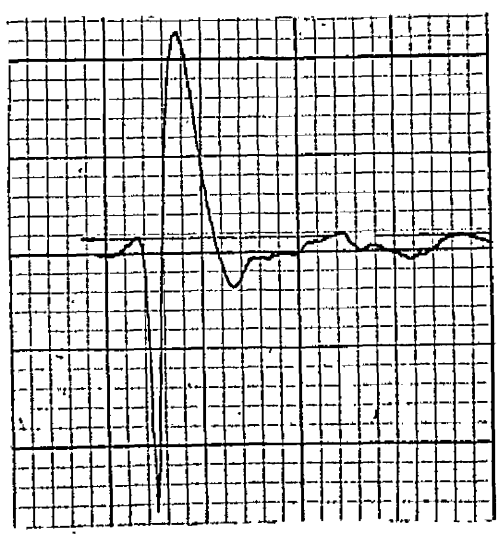
3



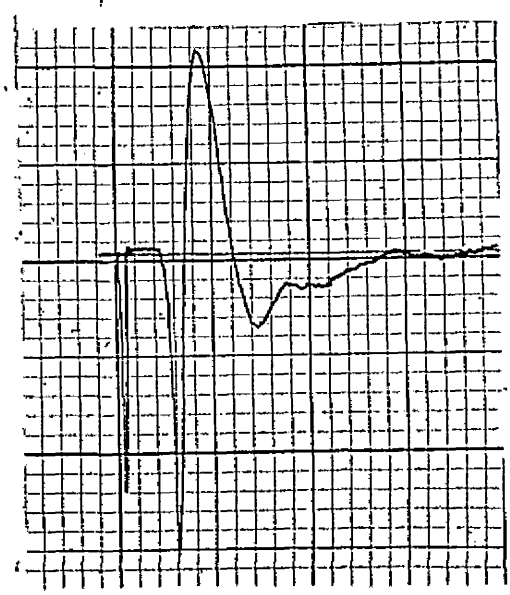
4



5



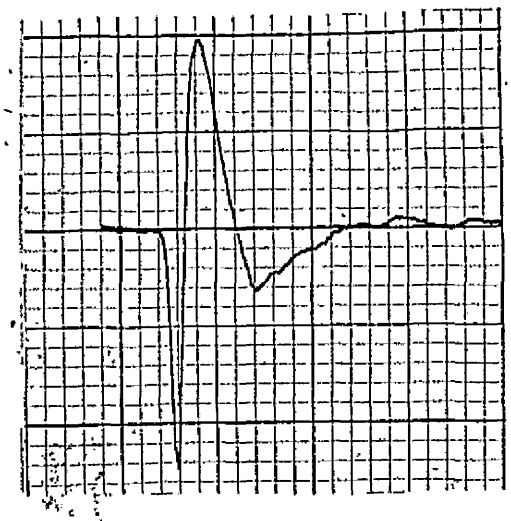
6



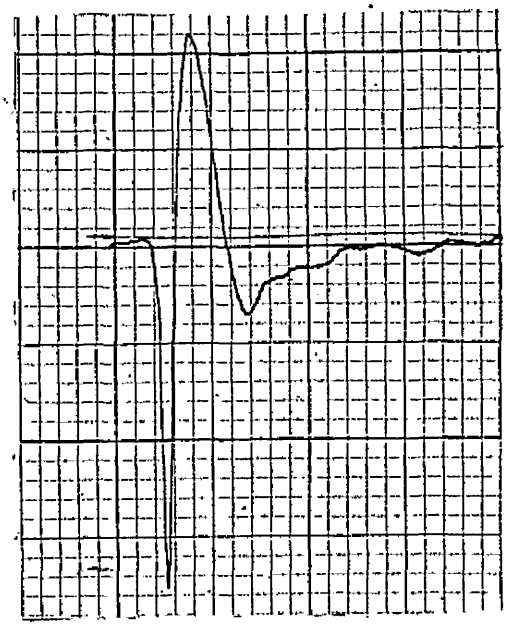
NO BACKGROUND

BACKGROUND

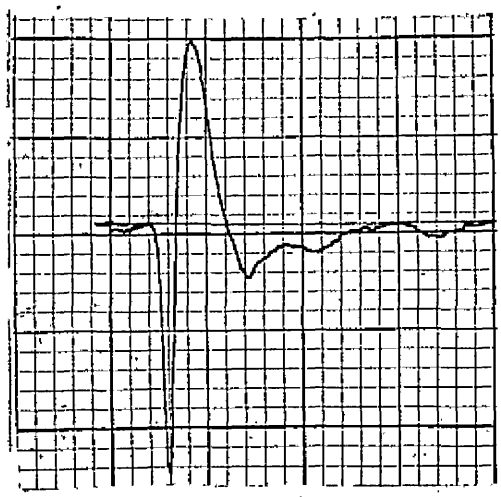
7



8



9



10

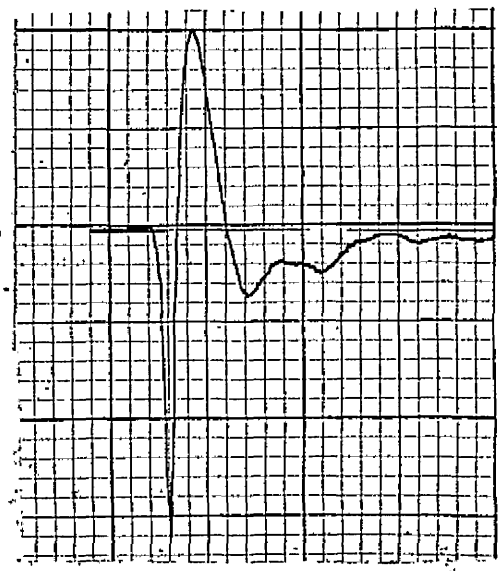
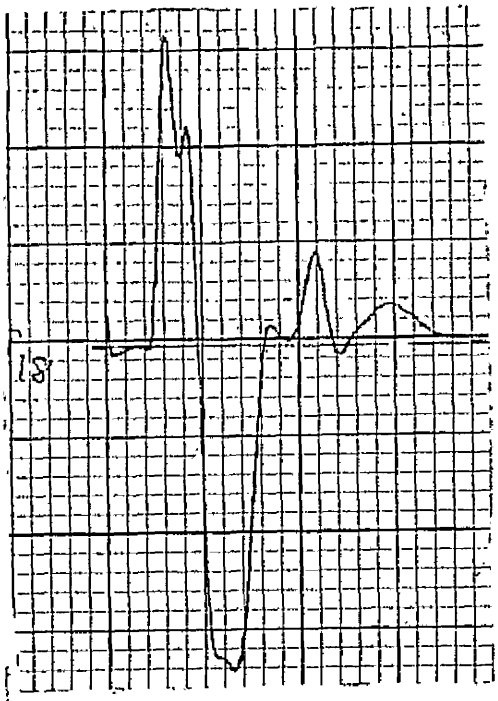


Fig. 12 The effects of background illumination on the evoked response in bird #7. Monopolar electrode at post-reversal depth. Numbers 1 – 8 indicate the order in which the runs were presented. Calibration: 100 microvolts, negative up; 50 msec.

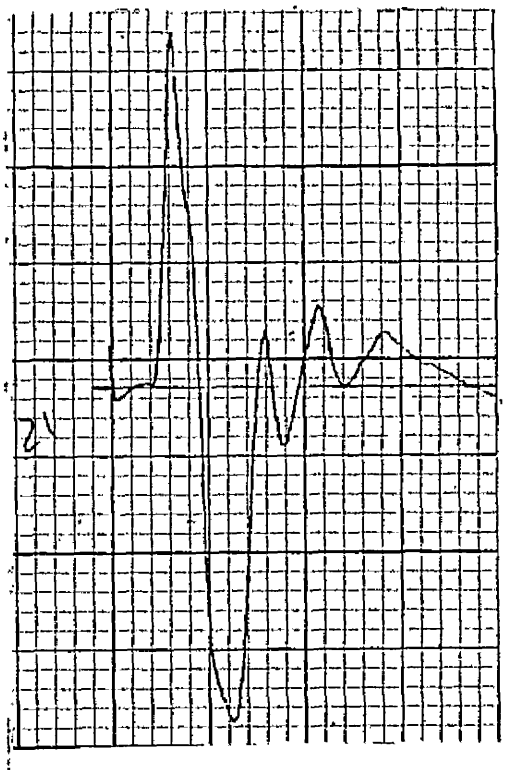
NO BACKGROUND

BACKGROUND

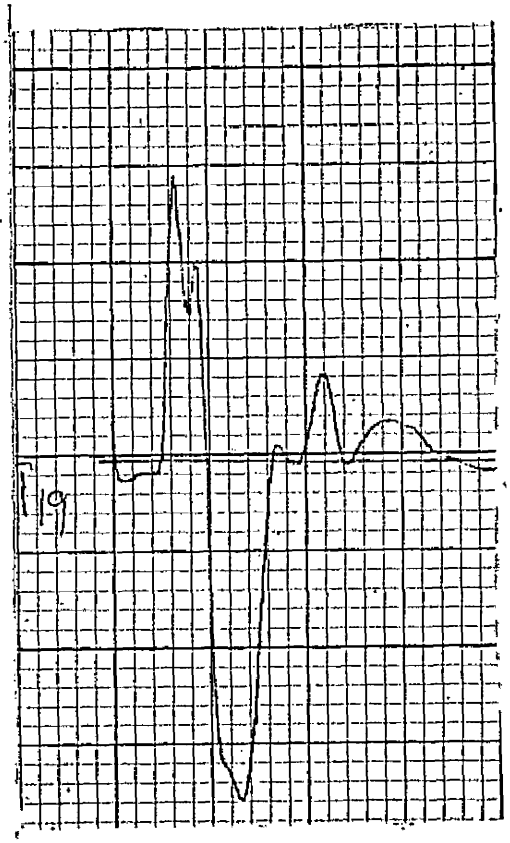
1



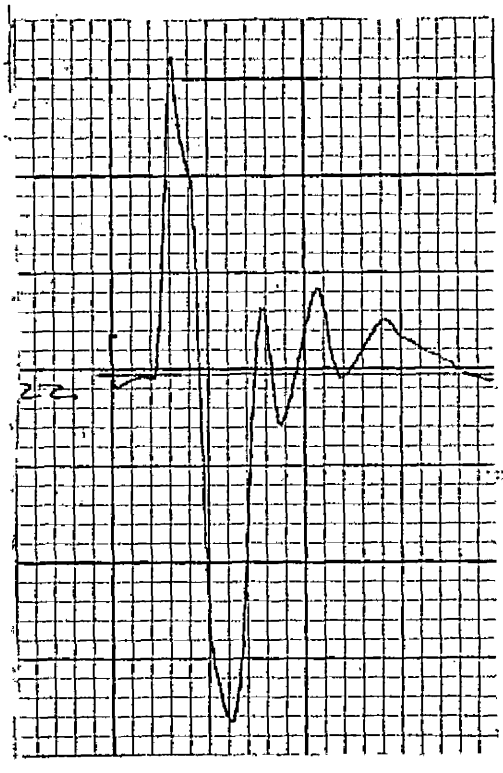
4



2



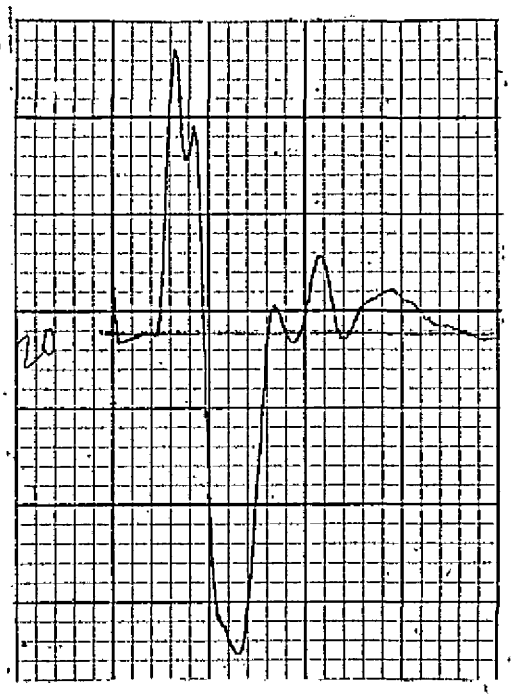
5



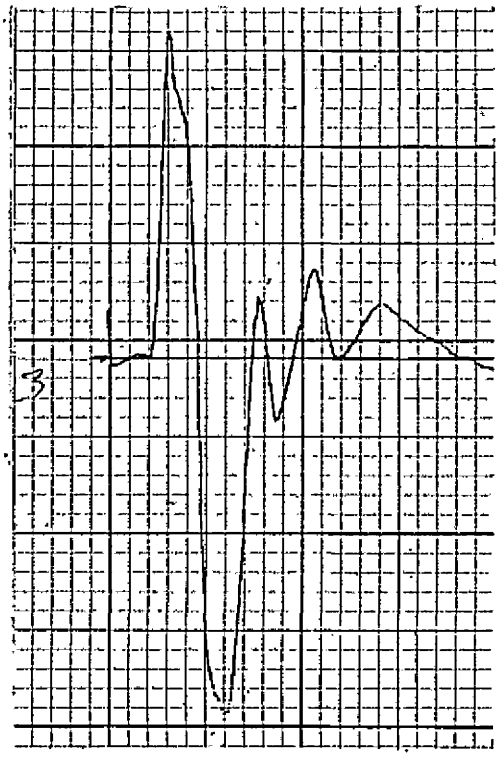
NO BACKGROUND

BACKGROUND

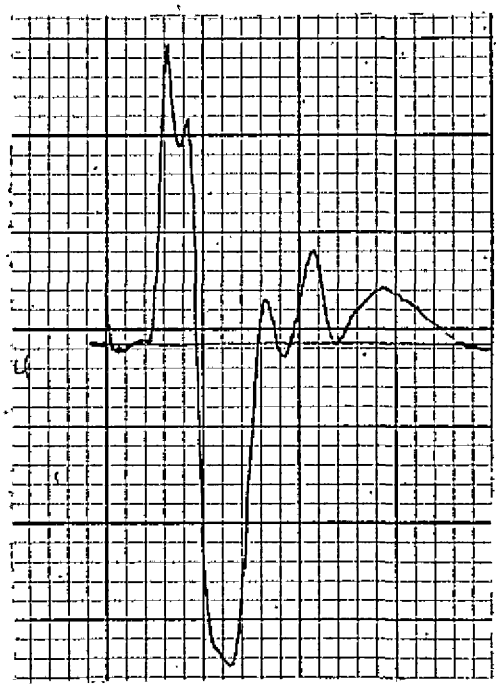
3



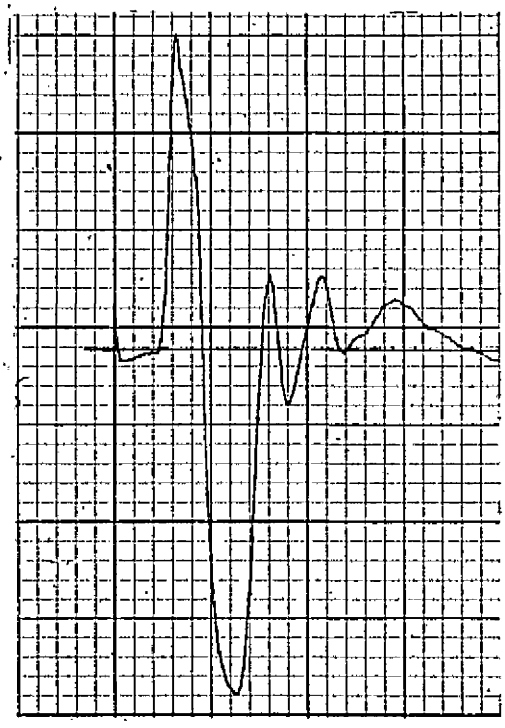
6



7



8



There is only one incidence of overlap between the average response amplitudes of runs in the two conditions.

Discussion.

The results indicate that background illumination potentiates the photically evoked response from some regions in the pigeon's optic tectum. It is surprising that the addition of retinal illumination brings about an increment in the response to the light flash. In the unanesthetized bird, it has been found that dark adaptation greatly enhances the potential, which would seem to be the opposite of the effect observed. It is generally accepted that an increase in retinal illumination brings about decreases in the output of the retina, which should result in a decrease of the cortical evoked response (Arduini, 1963). Bignall and Rutledge (1964) showed that the effects of background illumination on the photically evoked potential in the anesthetized cat's cortex was not an increase in the amplitude of the response, but rather a decrease in the amplitude of the primary and secondary peaks. Similar results have been found for the rat (Samson and Babb, unpublished observation). The results obtained do not easily fit into Arduini's proposal to account for potentiation. They seem to be more in accord with Chang's proposal of some central facilitation produced by background illumination. However, it is not known how the tonic activity of

the bird's retina is affected by background light, nor what effects background illumination would have on the tectal response to optic nerve stimulation. Moreover, since the mammalian and avian visual systems are very different, generalizing from these results to those obtained from mammals is perhaps unjustified.

ISOLATION EXPERIMENT

Since it was not known exactly what the effects of isolation might be, and since the potentiation phenomenon has been found sensitive to changes in neural function, it seemed reasonable to test the response of the isolated tectum to flashes when background illumination was and was not present.

Method.

Four birds were used with chronic monopolar electrodes in the lateral location of the optic lobes at a depth of 12 mm from the skull surface. In birds #1 and #32, the indifferent electrode was in the frontal bone of the skull, and for birds #14 and #93 it was placed into the ipsilateral hemisphere at a depth of 1 mm. The new occluders, as previously described, were used.

On each test session, the bird was brought from its home cage (which was brightly lighted by a 12", 112v fluorescent bulb) to the experimental room, and the adapter unit was placed on the plastic ringe. The stimulus presentation unit was coupled to the right eye and a light filter was placed over the left adapter unit to prevent stimulation of the left eye. A ten minute interval preceded the testing so that the bird could get used to the

experimental situation and dark adapt. Two days of pre-testing (PRE) were given. Each day two runs of fifty flashes per run were presented to each eye. The first run was given without, the second with, background illumination. After the PRE sessions, one eye was occluded for seven days, with the deprivation sessions occurring on the sixth and seventh days of the isolation period. The recording procedure was identical to the PRE sessions, except that on the second day an occluder was placed over the control eye two hours before the recording session so as to control for possible effects of dark adaptation in the experimental eye. Both occluders were removed when the stimulus adapter unit was fixed to the rings. After the last isolation session, the animals were given a seven day period of normal vision and the post deprivation sessions (POST) recorded on the sixth and seventh days. One week after the POST sessions, a second post session (POST-POST) was given to three birds.

After this, two of the birds were run through the experiment in the same way, except that the eye which had previously served as the control was now isolated.

Both computer averages and photographic records were made of the responses of the contralateral tectum. For analysis, the film was projected through an enlarger onto a grid marked off in 2.5 mm squares. As there was

a calibration mark on each film, the raw amplitude scores could be converted into microvolts. The latency and amplitude of the first peak and amplitude of the second peak were measured (see Fig. 9) from the film records (the error of measurement is dependent on the amplitude of the potential and is given in the appropriate tables). Information about waveform changes of the responses was obtained from the computer averages.

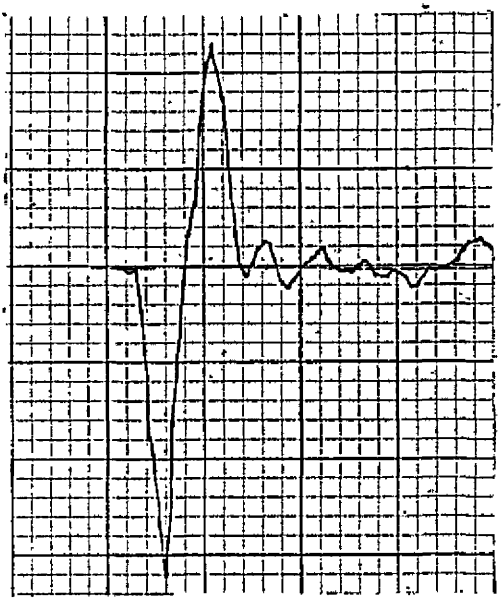
Results.

Figures 13 and 14 give the computer averages of the responses obtained during the PRE, DEP, and POST sessions for bird 14 (Appendix 1 shows the records from the isolated optic lobe of the other three birds). The response consists of a rapid negative deflection, followed by one or a series of positive deflections. Inspection of the PRE records shows that background illumination does affect the response. There is an obvious decrease in the amplitude of the second peak and a small decrease in the amplitude of the first peak when background illumination is introduced. This is the opposite of the effect that background illumination had on the responses of the anesthetized bird. The DEP records show that isolation greatly reduced the effect of background illumination in the experimental eye. The responses during background illumination resemble those when no background is present. A similar but smaller ef-

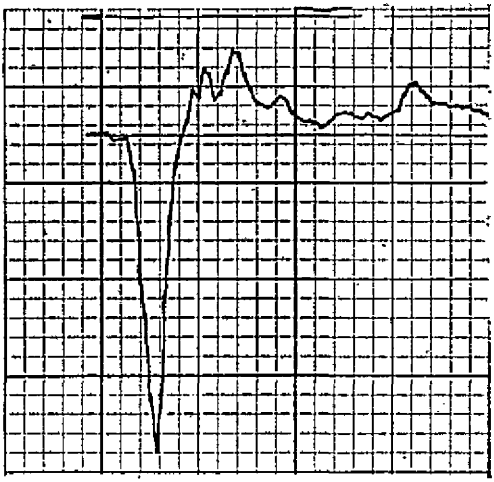
Fig. 13 Bird 14, averaged responses of the isolated tectum. Calibration: 100 microvolts, negative down; 50 msec.

PRE

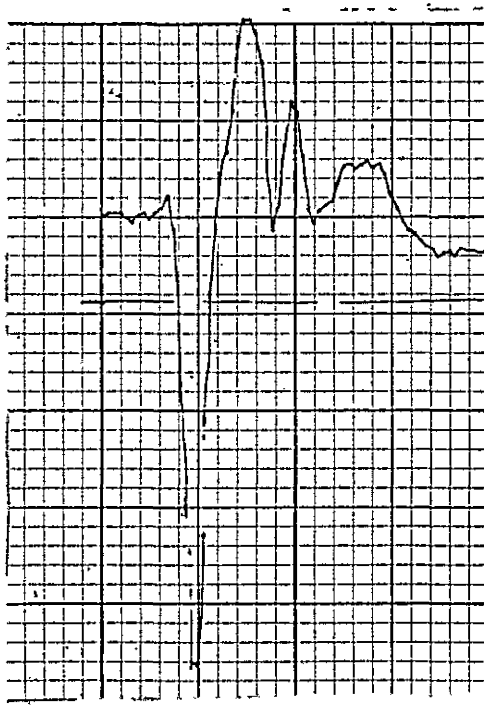
NO BACKGROUND



BACKGROUND

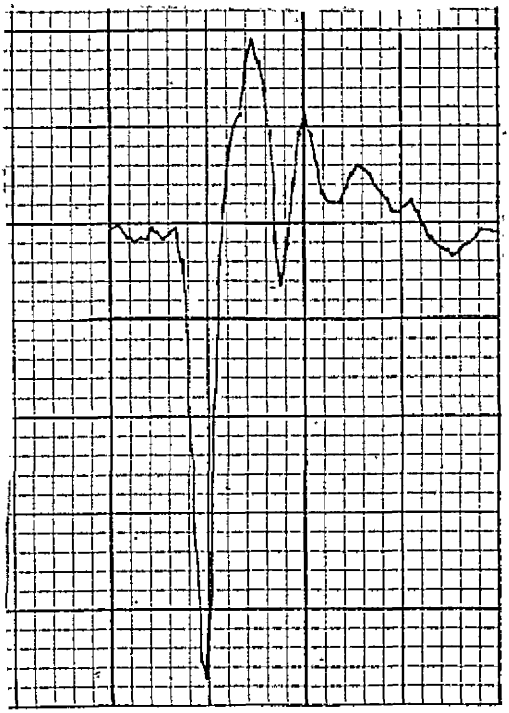


NO BACKGROUND



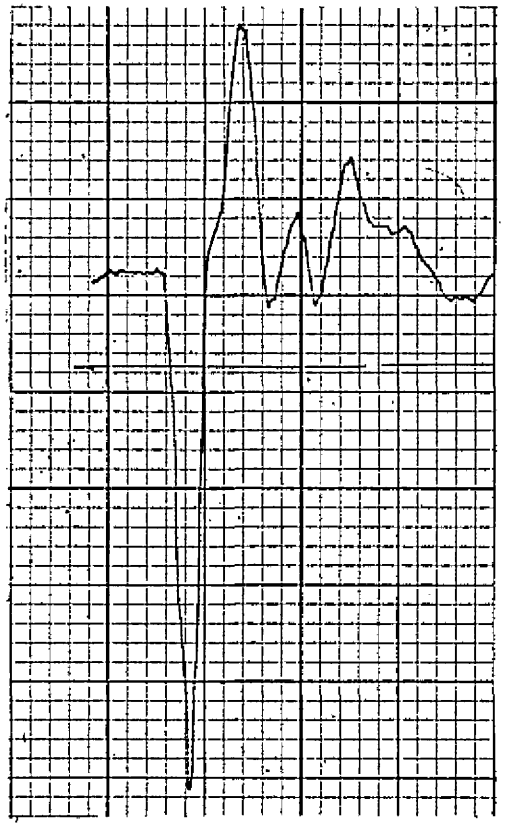
DEP

BACKGROUND



POST

NO BACKGROUND



BACKGROUND

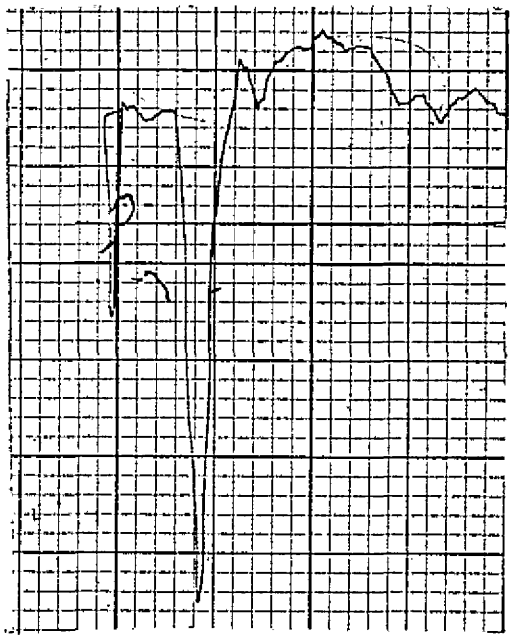
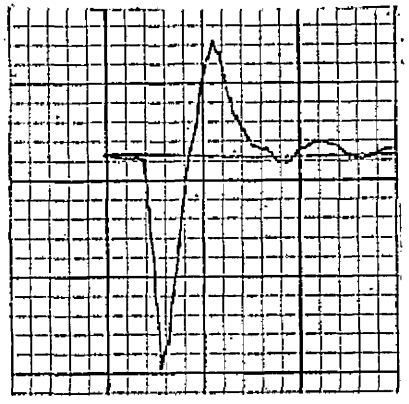


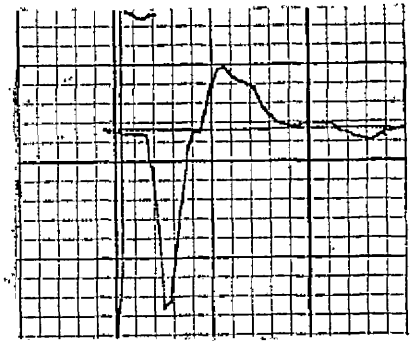
Fig. 14 Bird 14, averaged responses of the control tectum. Calibration: 100 microvolts, negative down; 50 msec.

PRE

NO BACKGROUND

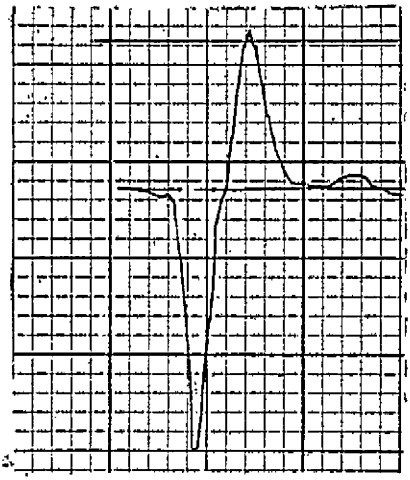


BACKGROUND

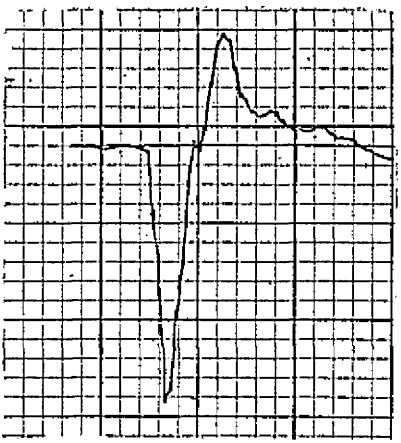


DEP

NO BACKGROUND

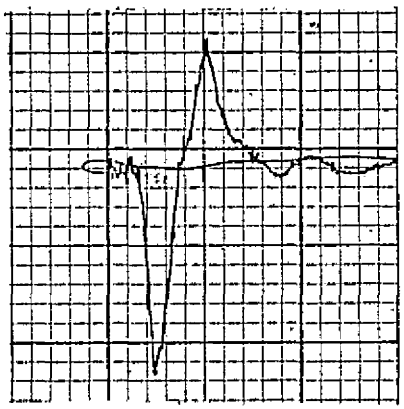


BACKGROUND

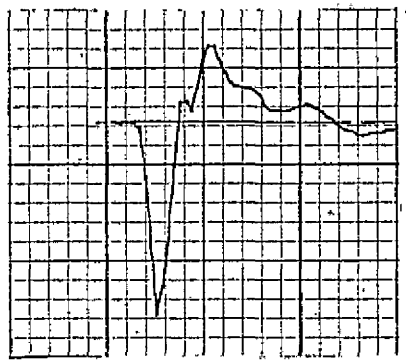


POST

NO BACKGROUND



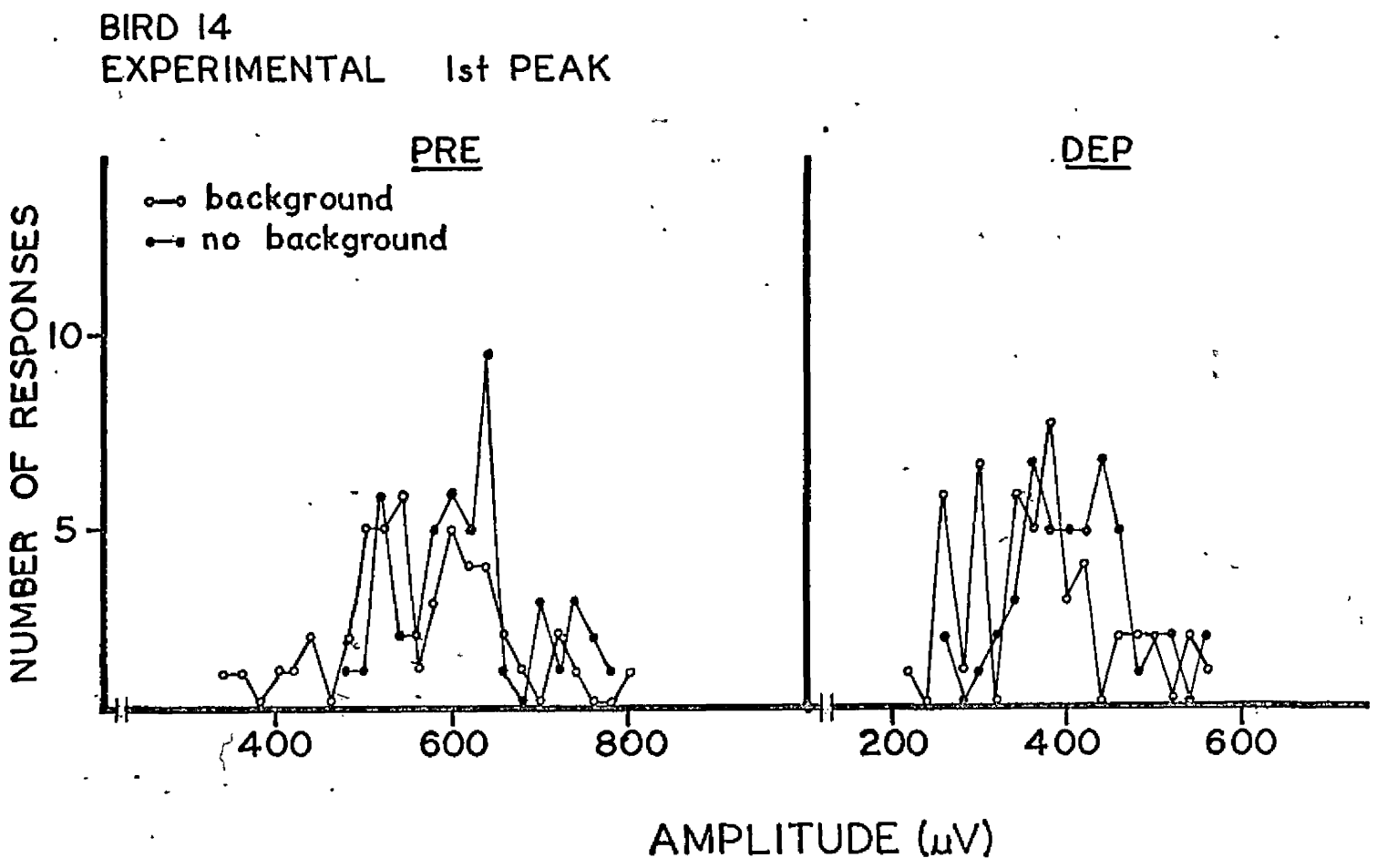
BACKGROUND



fect also seems to occur in the control tectum during the DEP sessions. The POST records show that after six days of normal vision, the second peak is again depressed by background illumination. There seems to be no increase in the amplitudes of the first peak in the no background condition after isolation as might be expected from the denervation supersensitivity hypothesis.

The film records were measured to clarify the computer results. The amplitude of the first negative peak (first peak) and the first positive peak (second peak) were determined (The averaged responses for bird #32 in the background condition of the PRE and POST sessions (Appendix 1) suggest that the second peak disappeared, but in the films it was always clearly defined, though its latency varied.). Figures 15 and 16 show the distribution of the amplitudes (in microvolts) of the first peak of the response of one bird (the distributions for the other animals are in appendix 2). The responses with and without background illumination are compared under the PRE, DEP, POST, and POST-POST conditions. These curves indicate no change in the amplitudes for the control tectum, and show that in the experimental tectum there is a decrease in the variability of the response in the DEP condition and a reduction in amplitude in the POST sessions. The distributions indicate that responses from the control tectum also vary less during the DEP

Fig. 15 Frequency distribution of amplitudes of first peak of responses from the isolated tectum.


BIRD 14
EXPERIMENTAL 1st PEAK
PRE
DEP
background
no background
NUMBER OF RESPONSES
10
5
400
600
800
200
400
600
AMPLITUDE (μV)

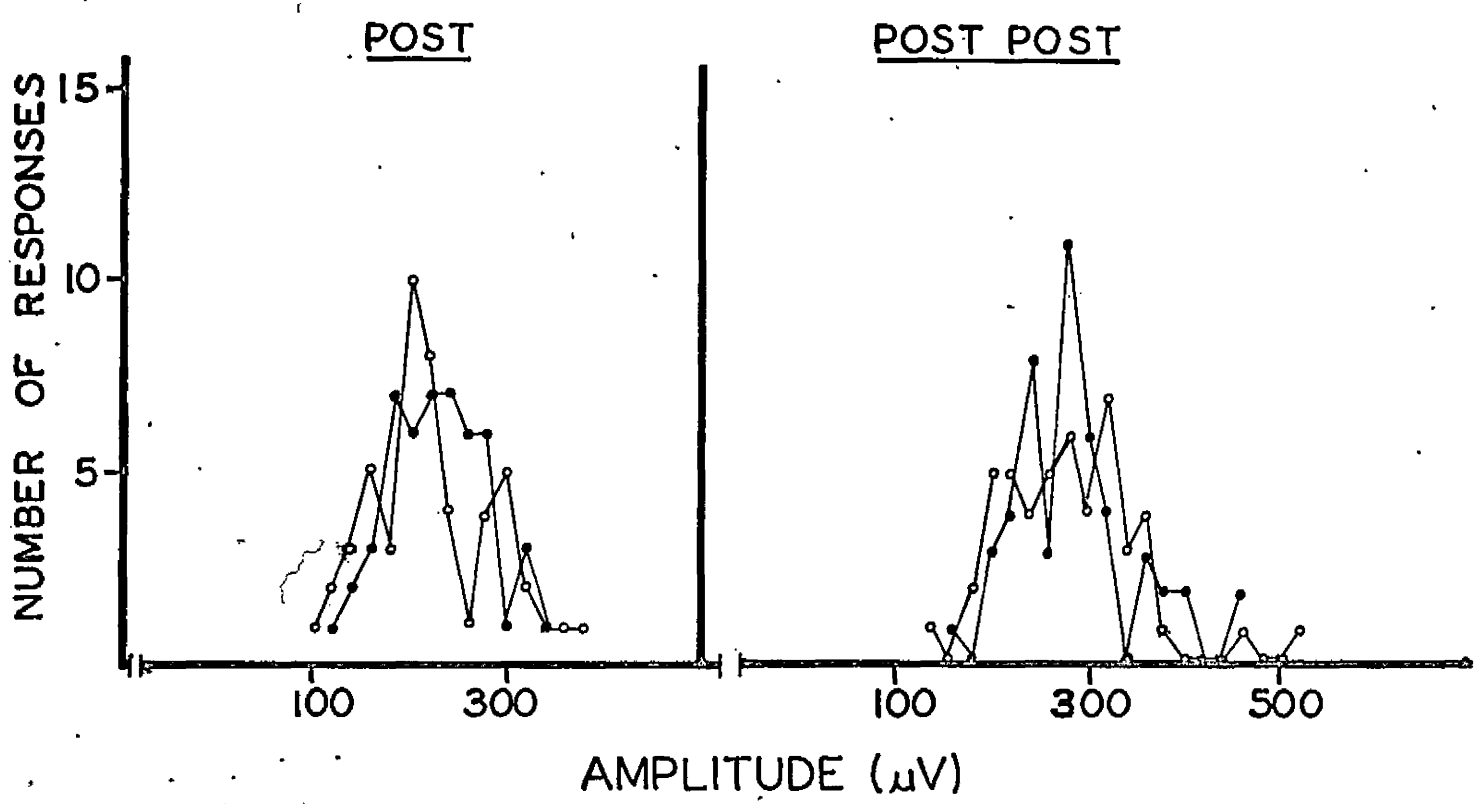
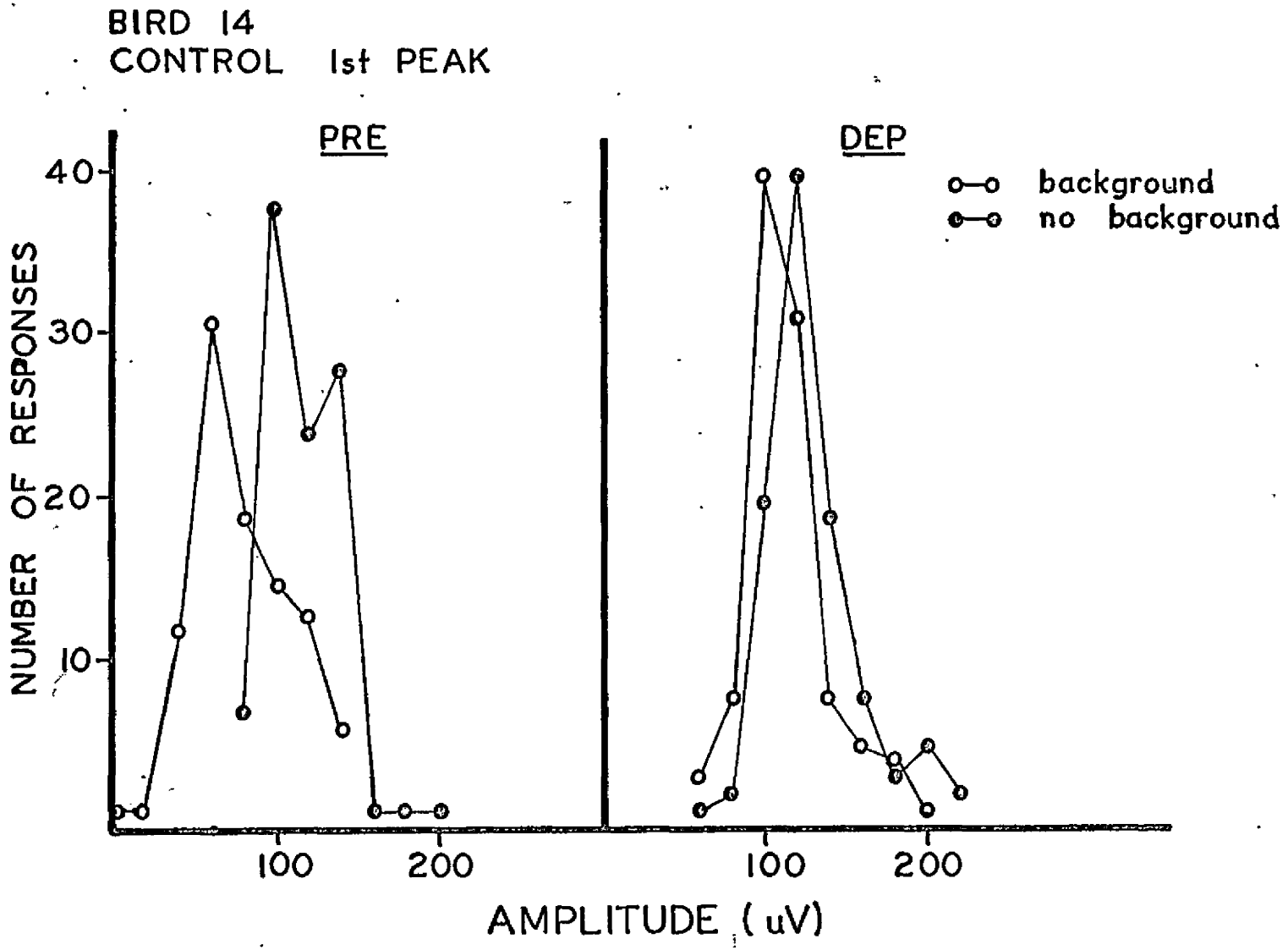
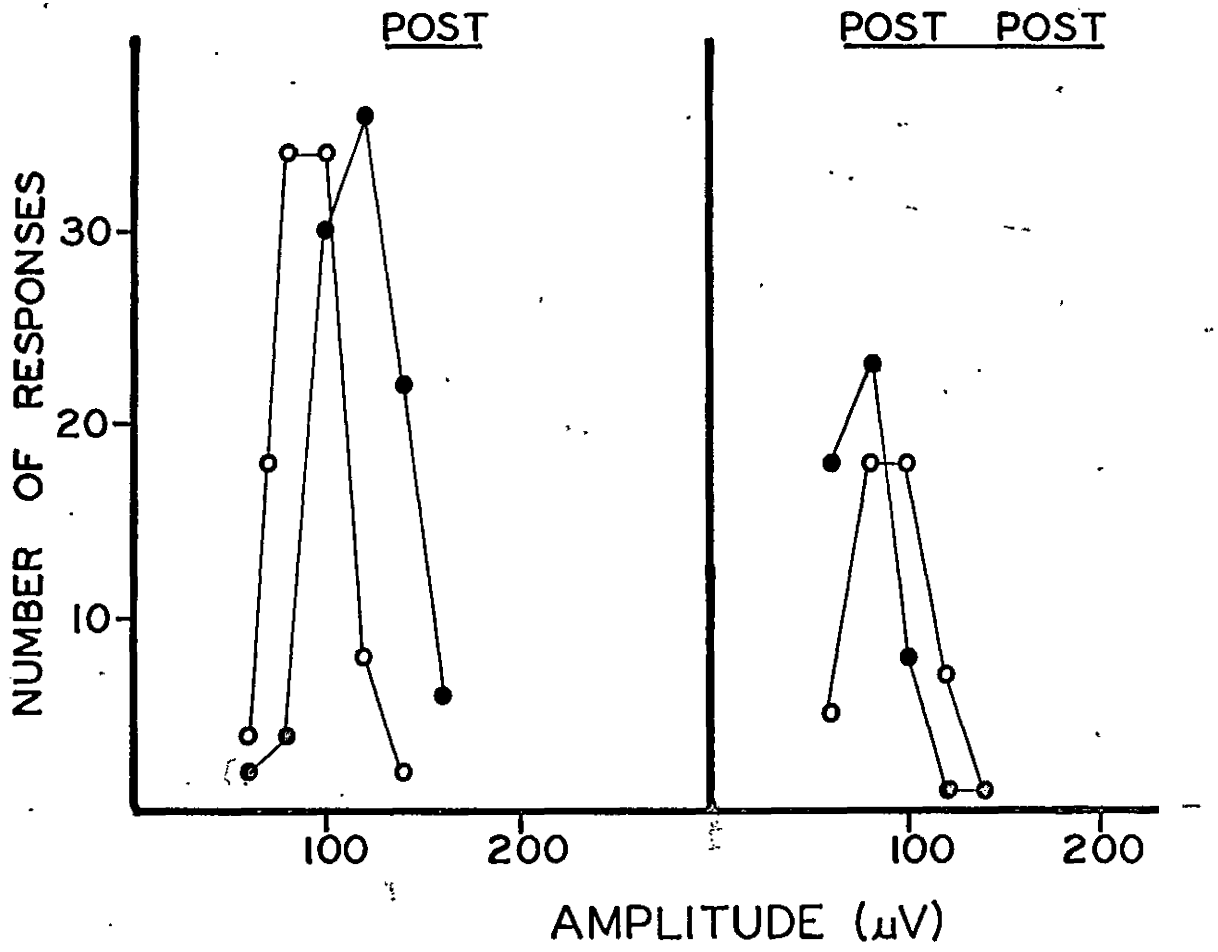

POST
POST POST
NUMBER OF RESPONSES
15
10
5
100
300
100
300
500
AMPLITUDE (μV)

Fig. 16 Frequency distribution of amplitudes of first peak of responses from the control tectum.


BIRD 14
CONTROL 1st PEAK
PRE
DEP
background
no background
NUMBER OF RESPONSES
40
30
20
10
100
200
100
200
AMPLITUDE (uV)


POST
POST POST
NUMBER OF RESPONSES
30
20
10
100
200
100
200
AMPLITUDE (μV)

sessions than in the PRE and POST sessions.

Table three presents the mean amplitudes of the primary peaks. An analysis of variance of these means for the control tectum shows that there is no significant difference between the PRE, DEP, and POST conditions. Further, background illumination does not significantly change the amplitude of response. The POST-POST scores are not included in this analysis as they were only available for three of the four animals. The analysis of variance for the experimental eye showed a significant difference ($f=5.63$ for 16 and 3 d.f.) between the PRE, DEP, and POST trials, but no difference between the amplitudes of responses with and without background. A Scheffe analysis showed that the PRE and DEP trials were not significantly different, but that both were larger than the POST scores.

In figures 17 and 18, the frequency plots for the amplitudes of the second peak of the responses from one bird (the plots for the others are in appendix 3) are shown. Examination of the curves shows that in the DEP sessions, the distribution of the second peak amplitudes of the responses from the isolated tectum with background illumination is very similar to the distribution of the amplitudes without background illumination. Also, during isolation, the variability of the amplitudes of the response is reduced. Using the means shown in table four, an analysis of variance on the control data showed that there was no

TABLE THREE

Amplitudes (in microvolts) of the primary peak

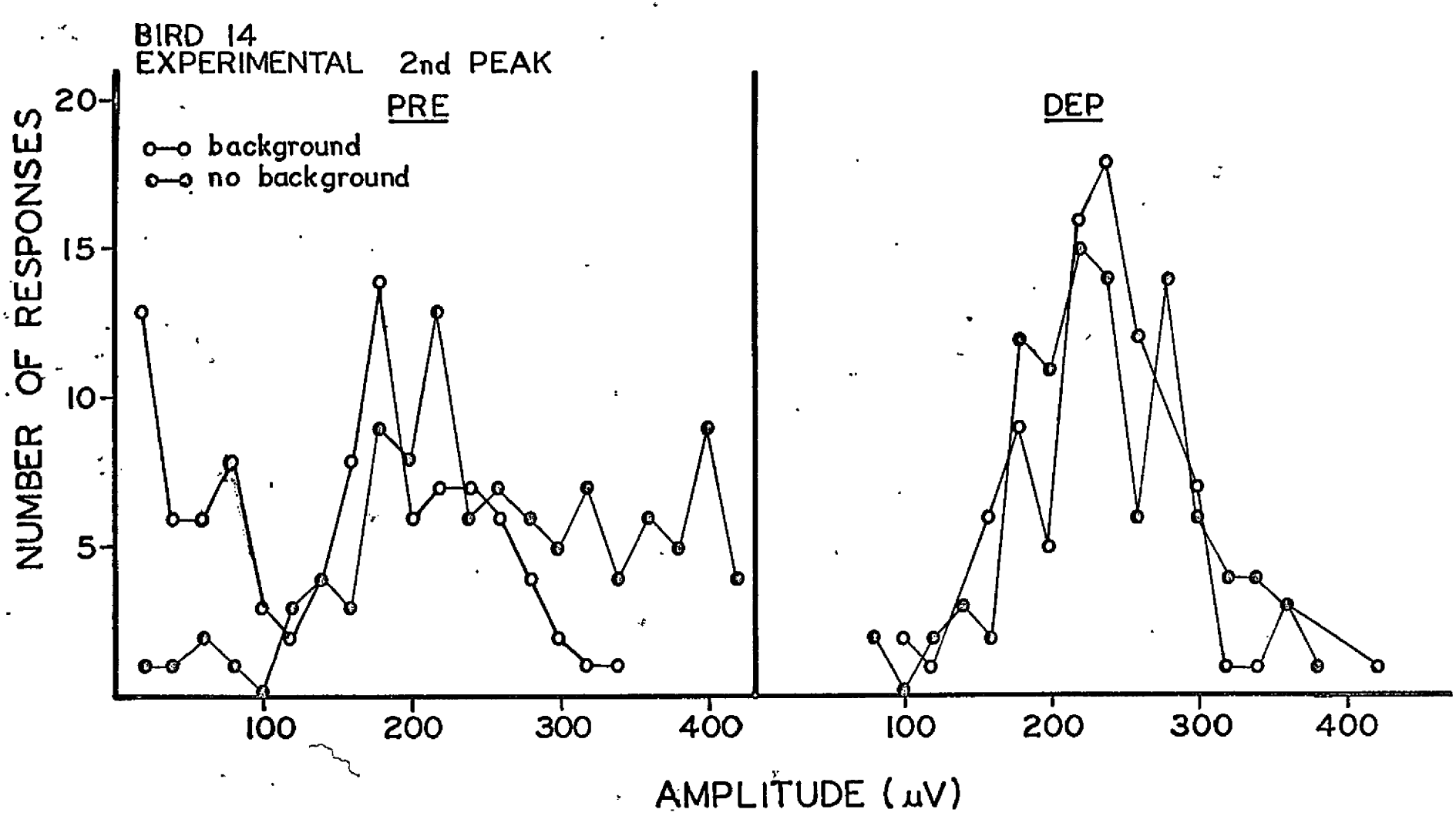
EXPERIMENTAL

	PRE		DEP		POST		MEASUREMENT
Bird	NB	B	NB	B	NB	B	ERROR
32	457	395	452	424	406	356	+ or -8
14	406	381	348	359	214	225	+ or -5
93	123	112	108	123	98	101	+ or -3
1	290	238	309	316	240	143	+ or -4.5

CONTROL

	PRE		DEP		POST		MEASUREMENT
Bird	NB	B	NB	B	NB	B	ERROR
32	239	211	294	255	308	235	+ or -3
14	80	75	62	62	90	70	+ or -2.5
93	125	112	140	125	127	117	+ or -3
1	384	384	378	362	370	306	+ or -5

Fig. 17 The amplitude distributions for the second peak of responses of the isolated tectum.


BIRD 14
EXPERIMENTAL 2nd PEAK
PRE
DEP
o—o background
o—o no background
NUMBER OF RESPONSES
20
15
10
5
100
200
300
400
AMPLITUDE (μV)

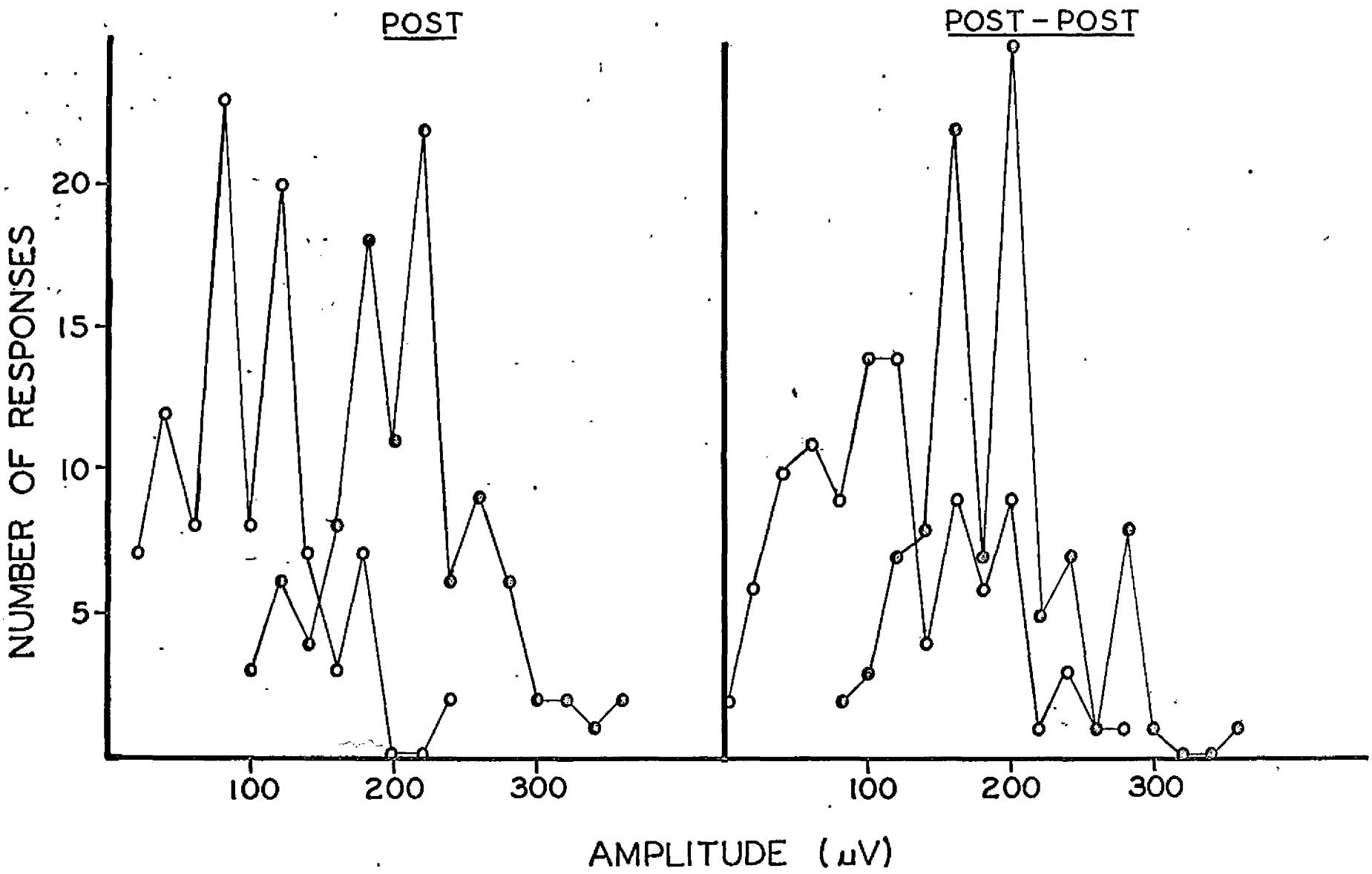
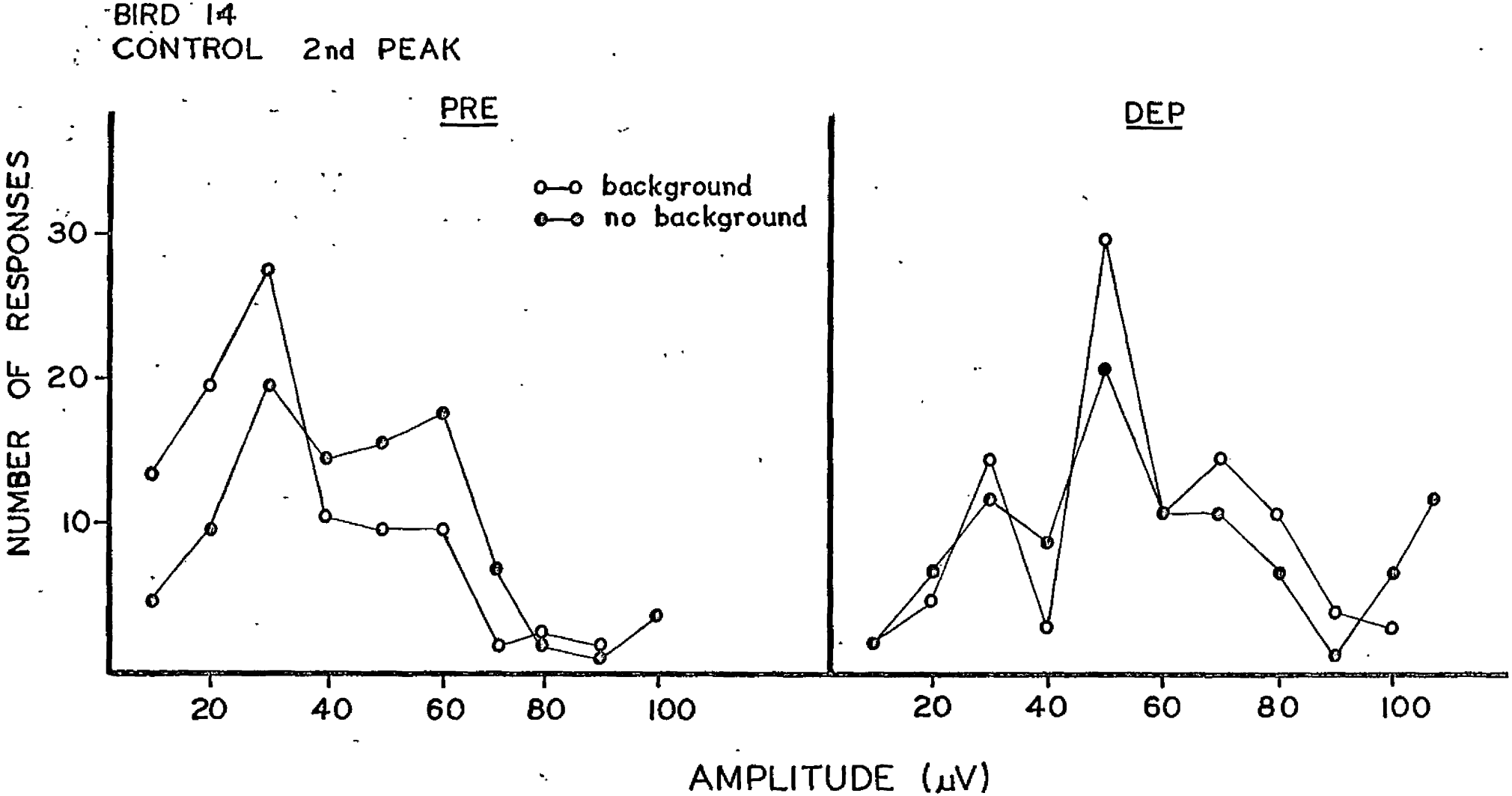

POST
POST - POST
NUMBER OF RESPONSES
20
15
10
5
100
200
300
100
200
300
AMPLITUDE (μV)

Fig. 18 The frequency plot for the amplitude of the second peak of responses from the control tectum.


BIRD 14
CONTROL 2nd PEAK
PRE
DEP
background
no background
NUMBER OF RESPONSES
30
20
10
20
40
60
80
100
AMPLITUDE (μV)

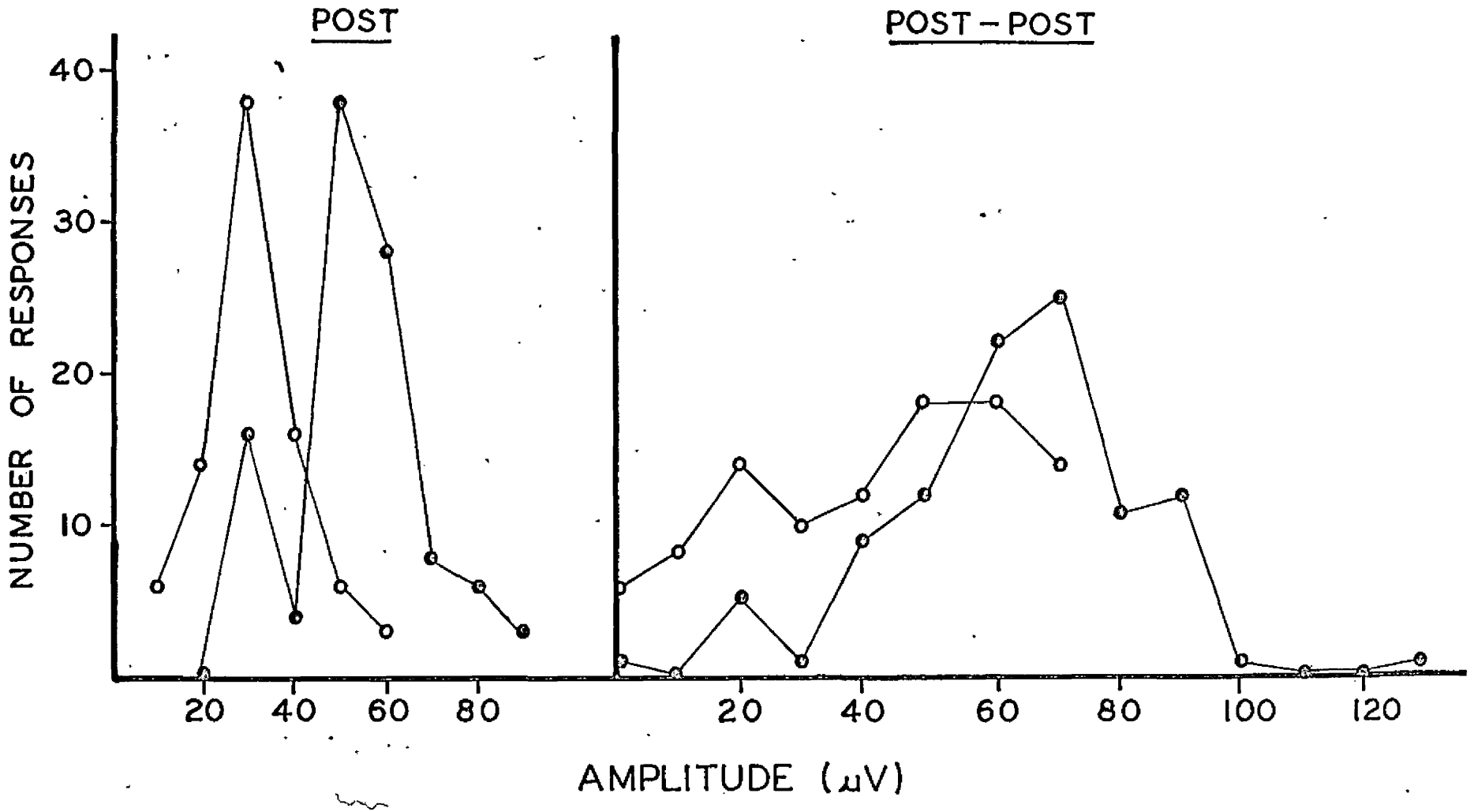

POST
POST – POST
NUMBER OF RESPONSES
40
30
20
10
20
40
60
80
20
40
60
80
100
120
AMPLITUDE (μV)

TABLE FOUR

Amplitudes (in microvolts) of the second peak

EXPERIMENTAL

	PRE		DEP		POST		MEASUREMENT
Bird	NB	B	NB	B	NB	B	ERROR
14	232	124	229	224	197	86	+ or -2
1	263	213	281	277	185	142	+ or -2.5
93	60	42	60	63	62	50	+ or -2.5
32	198	119	261	261	214	120	+ or -5

CONTROL

	PRE		DEP		POST		MEASUREMENT
Bird	NB	B	NB	B	NB	B	ERROR
14	48	35	61	50	55	33	+ or -2
1	258	170	242	206	246	156	+ or -2
93	22	18	26	20	27	20	+ or -1.5
32	106	65	135	72	144	44	+ or -3.5

significant difference between the PRE, DEP, and POST conditions, and no significant difference between the amplitudes of the potentials with and without background illumination. An analysis of variance on the means of the experimental eye (see table four) shows a significant difference ($F=6.3$, 2 and 6 d.f.) between the PRE, DEP, and POST conditions. The mean difference between the amplitudes of the potentials with background is not significantly different from those without background illumination. A Scheffe analysis finds the DEP scores are significantly larger than the PRE or POST, and the PRE are larger than the POST. To clarify this data, the mean amplitudes of the responses with background were expressed as percentages of the mean amplitude without background.

Table five shows the percent scores for both the first and second peaks. An analysis of variance shows a significant difference for both the first and second peaks (first peak $F=2.94$ for 5 and 15 d.f.; second peak $F=8.50$ for 5 and 15 d.f.) between the PRE, DEP, and POST scores. A Scheffe comparison is given in table six. It can be seen immediately that the experimental DEP scores for both first and second peaks are significantly larger than any of the other scores. These results again show that the major effect of isolation is on the second peak response during background illumination.

TABLE FIVE

Percentage scores (Mean amplitude with background/mean amplitude without background).

Primary Peak

	Experimental			Control		
Bird	PRE	DEP	POST	PRE	DEP	POST
32	86	94	88	88	87	76
14	94	103	105	93	100	78
93	91	114	103	90	89	92
1	82	102	60	83	96	82

Second Peak

	Experimental			Control		
Bird	PRE	DEP	POST	PRE	DEP	POST
32	53	98	44	73	82	60
14	81	98	77	66	85	63
93	70	105	81	81	77	74
1	60	94	56	61	53	31

TABLE SIX

Scheffe analysis for the results of the analysis of variance on the difference scores of table five.

Primary Peak

	PRE*	DEP*	POST*	PRE	DEP	POST
PRE*	-	60	3	1	19	25
DEP*		-	57	59	41	85
POST*			-	2	16	28
PRE				-	18	26
DEP					-	44
POST						-

C greater than 20 is significant

Second Peak

	PRE*	DEP*	POST*	PRE	DEP	POST
PRE*	-	131	6	17	33	36
DEP*		-	137	114	98	167
POST*			-	28	39	30
PRE				-	16	53
DEP					-	69
POST						-

C greater than 46 is significant

* Experimental tectum

The mean latencies for the onset of the primary response in the PRE, DEP, and POST conditions were found to be between 15 and 20 msecs. The resolution of the analysis was not fine enough to determine small changes in latency and therefore we cannot say what effects deprivation had upon the latencies of the response.

The two animals which were put through the procedure again, after reversing the experimental and control eyes, showed the same changes (see table seven) as in the original run. Again, during isolation, background illumination had less effect on second peak of the deprived tectum.

The occluder which was placed over the control eye two hours before the second deprivation trial showed that dark adaptation was not a factor. An analysis of the response amplitudes of the two deprivation sessions (one with the occluder, the other without) revealed no differences.

During the experiment, pupil size was not controlled. To test the possibility that pupil size could be involved with the result, responses to light flashes with and without background illumination were collected before and after local application of atropine sulphate (2%) on the eye. The results are shown in the computer averages in Fig. 19. The response shows a general increase in amplitude but the effect of background illumination is the same; the amplitude of the second peak is decreased.

TABLE SEVEN

Mean amplitudes of the second peak of responses of two birds retested with the control and isolated tectums reversed.

Isolated Tectum

	PRE		DEP		POST	
Bird	NB	B	NB	B	NB	B
14	69	42	48	48	65	34
1	213	155	130	113	200	152

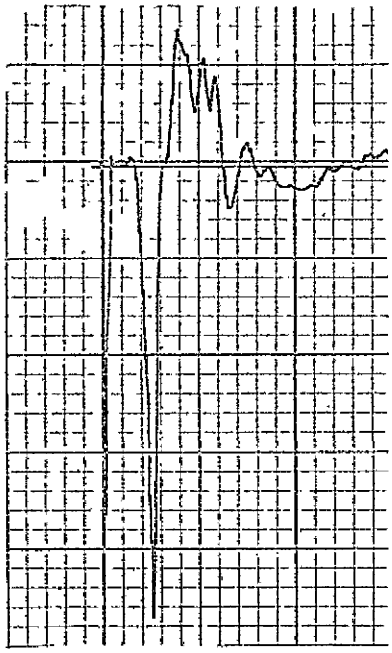
Control Tectum

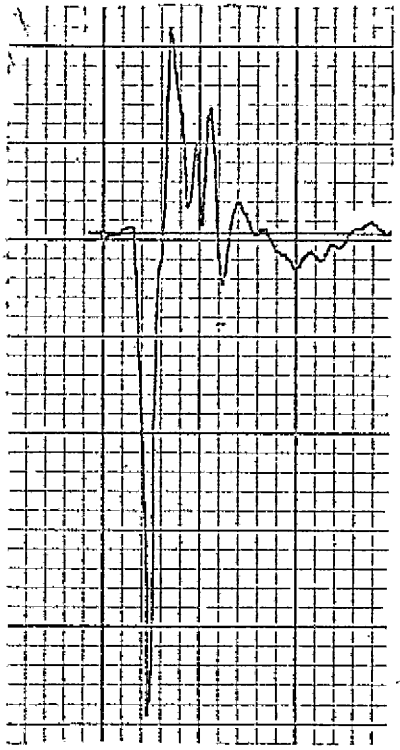
	PRE		DEP		POST	
Bird	NB	B	NB	B	NB	B
14	185	112	187	105	222	138
1	233	204	162	142	190	163

Fig. 19 Effects of atropine on the photically evoked response, with and without background illumination.

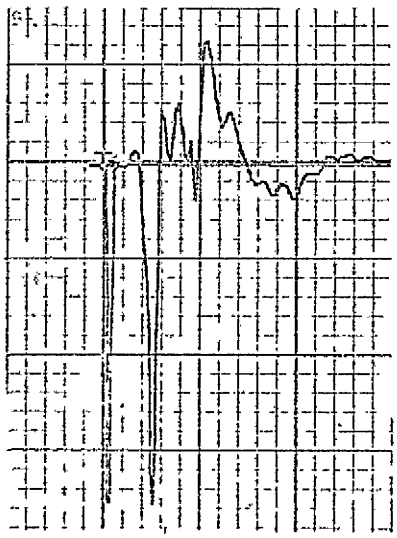
BEFORE AFTER

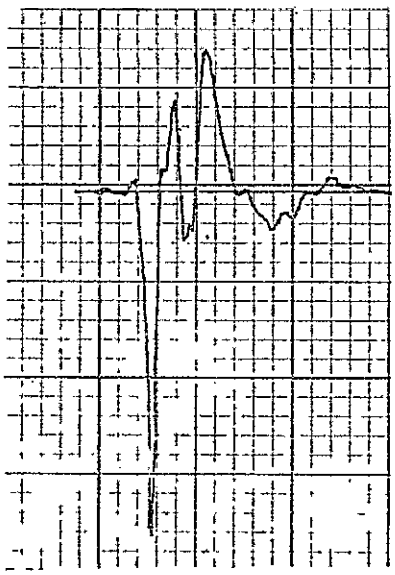
NO BACKGROUND





BACKGROUND

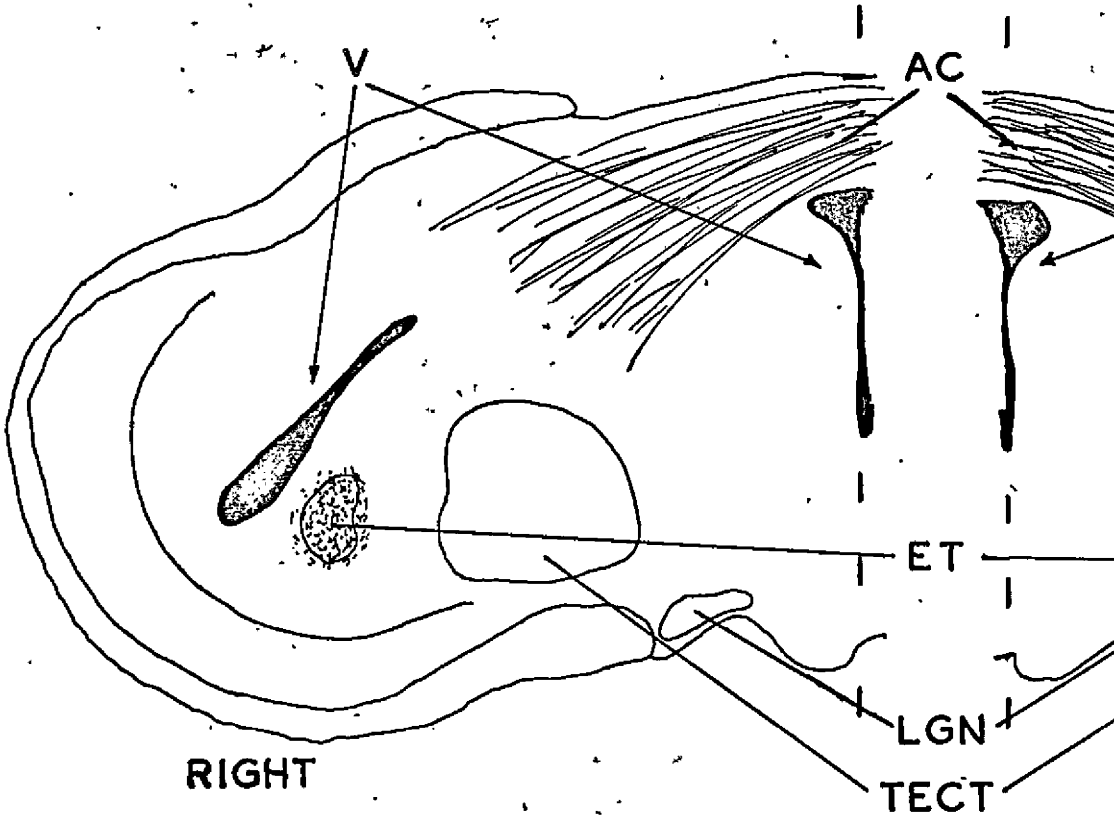




The histological location of the electrodes are shown in Figs. 20, 21, 22, and 23. In all but one bird (#93), the electrodes were found to be approximately within the same area, either in the tectal nucleus or the tectothalamic tract. In the other bird, one electrode would appear to have been shorted out above the tectum, accounting for the small potentials which were recorded from that site.

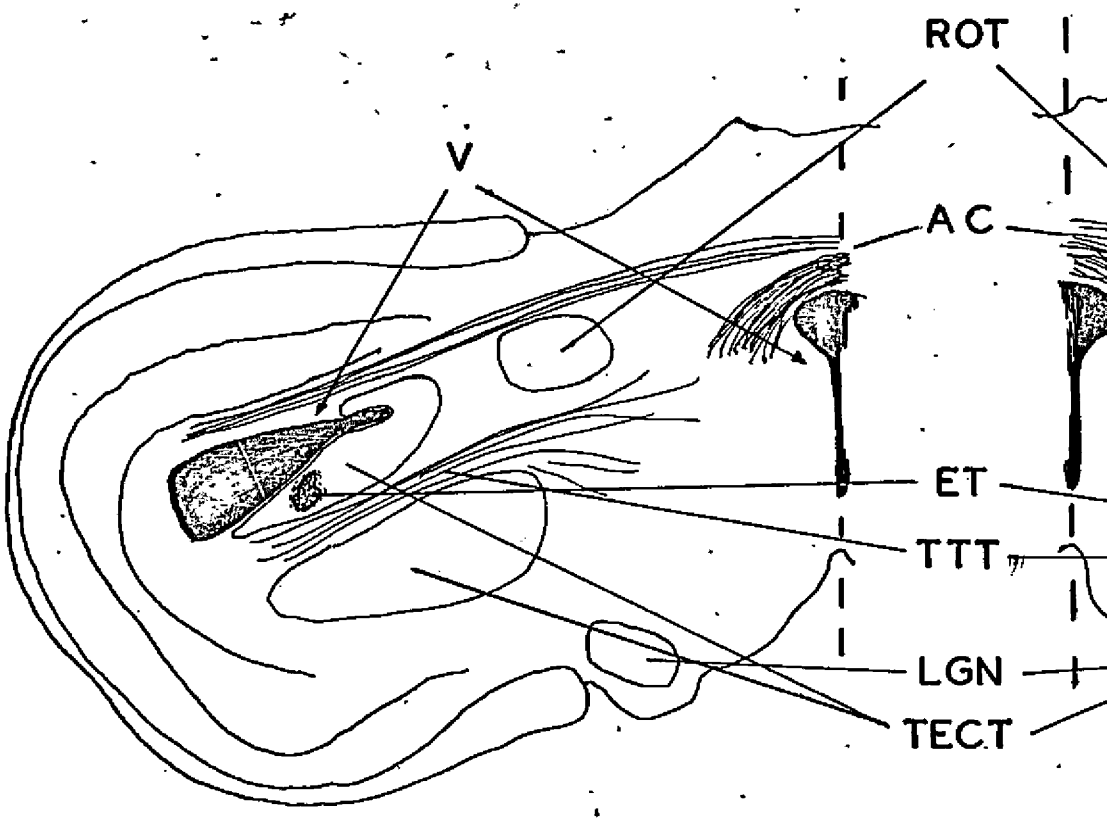
Figs. 20 to 23 Histological location of electrodes in isolation experiment. (AC - anterior commisure; ET - electrode; LGN - lateral geniculate nucleus; ROT - nucleus Rotundus; TECT - tectal nucleus; TTT - tectal-thalamic tract; V - ventricle; X13.4)

BIRD 32



V
LEFT

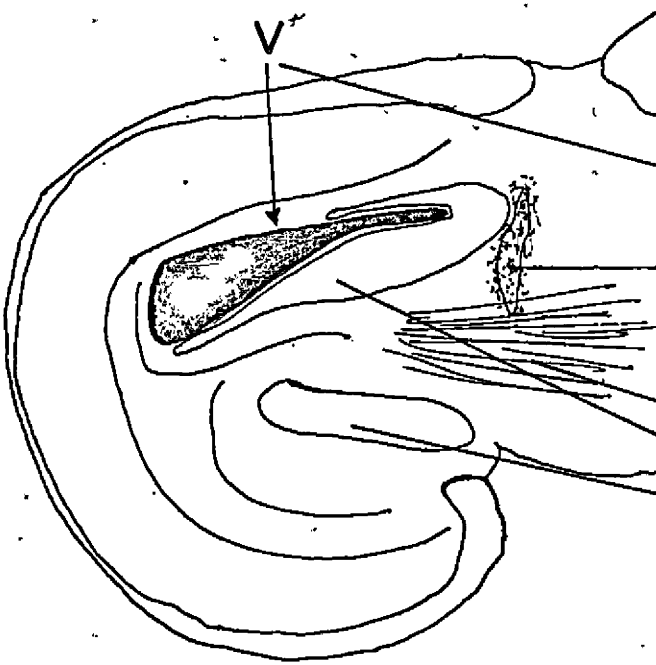
BIRD I



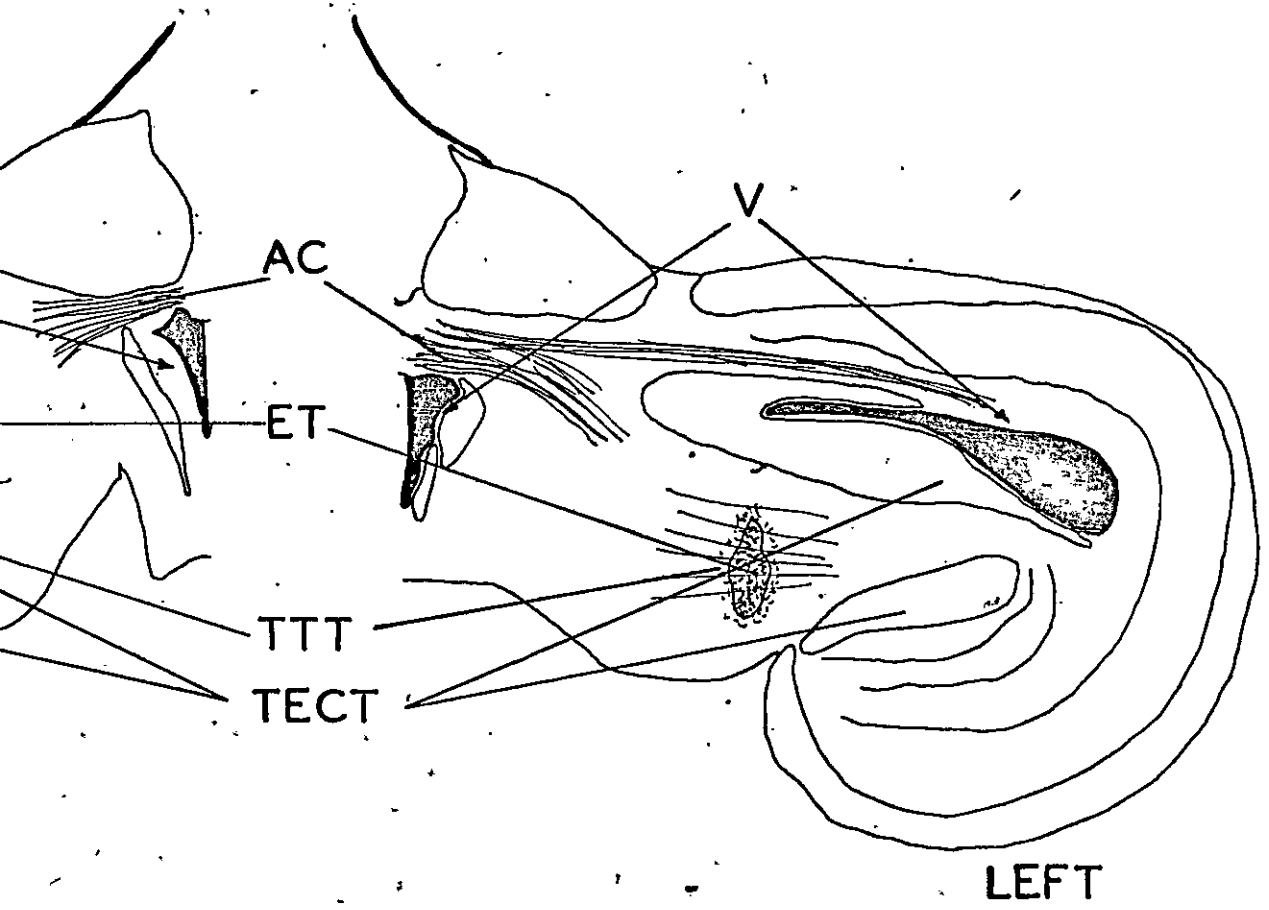
RIGHT

LEFT

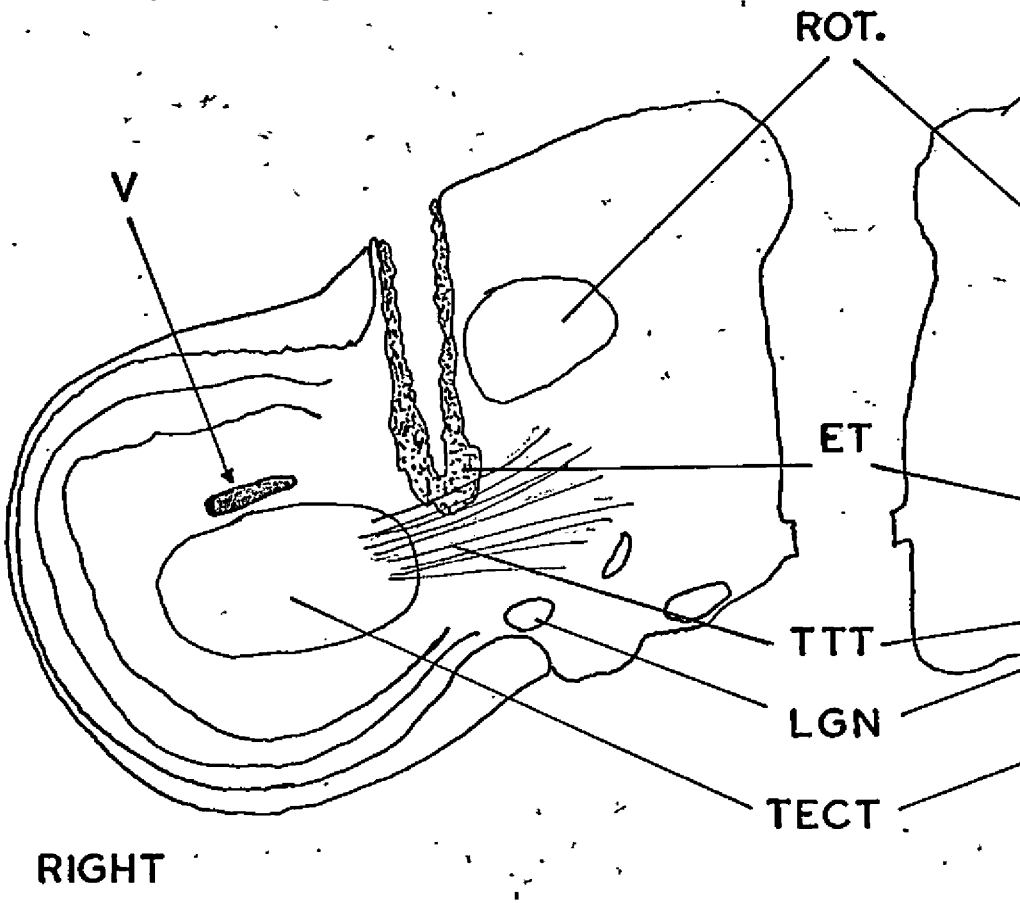
BIRD 14



RIGHT


V
AC
ET
TTT
TECT
LEFT

BIRD 93



V
LEFT

DISCUSSION

There were three major findings of the experiment:
(1) After seven days of isolation, background illumination does not depress the amplitude of the photically evoked response, as it does in the normal bird.
(2) Isolation did not have a consistent effect on the absolute amplitude of the response.
(3) In the normal bird, under barbituate anesthesia, background illumination potentiates the photically evoked response.

It seems unlikely that factors other than deprivation are responsible for the first result. Obvious factors such as dark adaptation are ruled out by the control measures, and in any case it has been observed that in the normal pigeon, background illumination depresses the positive peaks whether the bird is dark adapted or not. Similarly, the fact that the results were obtained during the deprivation sessions, and not before or after, rules out any possibility that changes in the properties of the electrode could be responsible. Further, since the two birds in which the procedure was reversed (so that the eye which was previously isolated now served as the control) showed the same effects, it seems unlikely that order effects

from the admittedly inelegant procedure of always testing the right eye first can have been important.

We are unable, unfortunately, to state definitely whether the effects observed were due to peripheral or central factors. However, there are some reasons for believing that they were central. Occluding the eye produced no changes in the cornea or retina which could be detected with an ophthalmoscope (though of course, there are many changes in the retina which might not be seen by this method). Similarly, although pupil size was not controlled, we do not believe that the results can be explained by saying that deprivation caused the mechanism responsible for pupillary contraction to fail, and that the larger pupil which resulted was responsible for the greater amplitudes of the response when there was background light. First, inspection of the birds' eyes did not indicate that there was any abnormality of the pupillary reflex of the deprived eye, and second, when the pupil is maximally dilated by atropine, the response of the normal bird is depressed by background light (see Fig. 19).

Finally, it can be seen from the averaged responses that there is a tendency for the second peak of the responses from the control to be relatively larger when background illumination is present during the DEP sessions

than it is when the background light is on in either the PRE or POST sessions. It is true that this effect is seen in only two birds (32 and 14), but this may be due to the position of the electrodes.

The data do not indicate that isolation had any effects on the absolute amplitudes of the responses when there was no background illumination. What changes there were can probably be attributed to changes in the electrodes, or to fluctuations in the state of the bird. It will be remembered, however, that a significant decrease in amplitude was found during the POST sessions. All birds showed a decrease, but the decrease of bird #14 (which showed the greatest decrease) is probably due to alterations in the properties of the electrode, since the response amplitude remained low for two weeks after the POST tests. Similarly, the responses of bird #93 continued to decline over the next two week period. The response amplitude of bird #1 returned to the PRE level, but the POST changes in this case are relatively small. Thus, on the whole, there is no reason to believe that the decrease in amplitude observed in the POST sessions is related to the isolation procedure.

Theoretical Implications.

The stimulus used was not maximal since in some preliminary experiments we varied the intensity of the

flash in steps until it was roughly twice the intensity used in the isolation study, and found that the amplitude of the evoked potential continued to increase. Hence, the fact that amplitude of the response did not increase after deprivation does not fit in well with what would be predicted from the denervation supersensitivity hypothesis. Granted that most studies which demonstrate the phenomenon have involved measuring the threshold of the response (and consequently it might have been more sensible to have measured the response threshold to light flash in this experiment), the experiments of Spiegel and his co-workers would lead us to expect an amplitude increase.

What other mechanisms might be involved? It is obvious that the results might be due to the failure of some inhibitory mechanism, and such a hypothesis is particularly attractive since it would also account for some of the effects which have been observed in humans (the lowered touch threshold, for instance). Such inhibition could involve the primary pathways, or the reticular system, or both. The fact that potentiation does not occur in the awake bird, but that it does both in the nembutalized bird and in the deprived tectum (birds 1, 14, and 93), does suggest that the reticular system may be involved. Of course, explanations in terms other than inhibition are possible, and we do not at present have the evidence to

decide which explanation is most adequate. However, the effect we have observed seems to be a powerful one, and by analyzing its basis in future experiments it should be possible to get some understanding of what happens in the nervous system during perceptual isolation.

Further Studies.

This study is essentially a preliminary one, and does not really adequately test the denervation supersensitivity hypothesis of the physiological bases of perceptual isolation. By repeating the study, with tests of temporal occlusion and threshold, a better test of the denervation supersensitivity hypothesis could be made.

The following questions will have to be at least partially answered before the results observed can be explained.

1. What is the mechanism by which background illumination affects the photically evoked response both in the normal and anesthetized bird? Is the effect similar to retinal tonic decreases as proposed by Arduini or is there more of an excitation effect as proposed by Chang?
2. What is the time course for the isolation phenomenon as found in this study and how long does the effect last?
3. What mechanism is deprivation affecting? is it a change in the inhibitory systems and/or the ARAS?
4. Will pattern stimuli result in a different evoked po-

tential than a non-patterned flash? What will isolation do to the patterned flash response?

5. Will total deprivation of vision have the same effect as pattern isolation?

SUMMARY

The effects of perceptual isolation on photically evoked potentials in the optic tectum were studied. It was found that:

1. Continuous retinal illumination affects the photically evoked potential. Generally, a marked decrease in the amplitude of the response was found when there was background illumination. Under barbituate anesthesia, background illumination did not depress the response, but was found to potentiate it.

2. Perceptual isolation of one eye greatly changes the effects of background illumination on the evoked response. Before deprivation the response was reduced by background illumination, after seven days of isolation background illumination had little effect. Following the period of deprivation, the response returned to normal seven days after the removal of the occluder.

REFERENCES

Afatanas, M. and J. P. Zubek Effects of prolonged isolation of the skin on cutaneous sensitivity. Percept. Mot. Skills, 1963, 16, 565 - 571.

Arduini, A. Influence of visual deafferentation and of continuous retinal illumination on the excitability of geniculate neurons. In: R. Jung and H. Kornhuber (eds.) Neurophysiologie und psychophysik des visuellen systems. Goettingen, Springer, 1961, pp. 117 - 125.

Arduini, A. The tonic discharge of the retina and its central effects. In: G. Moruzzi et al (eds.) Brain mechenisms, Progress in brain research. v. 1, London, Elsevier, 1963, pp. 184 - 206.

Arduini, A. and T. Hirao Enhancement of evoked responses in the visual system during reversible retinal activation. Arch. ital. Biol., 1960, 98, 182 - 205.

Arduini, A. and M. H. Goldstein Enhancement of cortical responses to shocks delivered to lateral geniculate body. Localization and mechanism of the effects. Arch. ital. Biol., 1961, 99, 397 - 412.

Arduini, A. and L. Pinneo Properties of the retina in response to steady illumination. Arch. ital. Biol.,

1962, 100, 425 - 448.

Arduini, A. and L. Pinneo The tonic activity of the lateral geniculate nucleus in dark and light adaptation. Arch. ital. Biol., 1963, 101, 493 - 507.

Axelsson, J. and S. Thesleff A study of supersensitivity in denervated mammalian skeletal muscle. J. Physiol., 1959, 147, 178 - 193.

Barlow, H. B., R. Fitzhugh, and S. W. Kuffler Change of organization in the receptive fields of the cat's retina during dark adaptation. J. Physiol., 1957, 137, 338 - 354.

Bexton, W. H., W. Heron, and T. H. Scott Effects of decreased variation in the sensory environment. Canad. J. Psychol., 1954, 8, 70 - 76.

Bignall, K. E., and L. T. Rutledge Origin of a photically evoked afterdischarge in cat visual cortex. J. Neurophysiol., 1964, 27, 1048 - 1062.

Bishop, G. H. and J. O'Leary Components of the electrical response of the optic cortex of the rabbit. Am. J. Physiol., 1936, 117, 292 - 308.

Braunstein, P. Effects of local skin deprivation on cutaneous detectability. Unpublished B.A. Thesis, McGill Univer., 1957.

Bremer, F. In the discussion of A. Arduini Influences of visual deafferentation and of continuous retinal

illumination on the excitability of geniculate neurons. In: R. Jung and H. Kornhuber (eds) Neurophysiologie und psychophysik des visuellen systems. Goettingen, Springer, 1961a, p. 116.

Bremer, F. Neurogenic influences on evoked potentials. In: W. A. Rosenblith (ed.) Sensory communication. New York, Wiley, 1961b, pp. 675 - 698.

Budge, J. L. Uber die bewegung der iris: für physiologen und artze. Braunschweig, Vieweg, 1885.

Burch, P. G. and R. D. Myers A simple method for reliably coating intracranial electrodes. J. exp. Anal. Behav., 1962, 5, 96.

Burns, B. D. Some properties of the isolated cerebral cortex in the unanaesthetized cat. J. Physiol., 1951, 112, 156 - 175.

Cannon, W. B. and H. Haimovici The sensitization of motoneurones by partial 'denervation'. Am. J. Physiol., 1939, 126, 731 - 740.

Cannon, W. B. and A. Rosenblueth The supersensitivity of denervated structures: a law of denervation. New York, MacMillan, 1949.

Catania, A. C. Techniques for the control of monocular and binocular viewing in the pigeon. J. exp. Anal. Behav., 1963, 6, 631.

Chang, H. T. Cortical response to stimulation of lateral geniculate body and the potentiation thereof by

continuous illumination of the retina. J. Neurophysiol., 1952a, 15, 5 - 26.

Chang, H. T. Functional organization of central visual pathways. Res. Publ. Ass. nerv. ment. Dis., 1952b, 30, 430 - 453.

Chavez, M. and E. A. Spiegel The functional state of sensory nuclei following deafferentation. Confin. neurol., 1957, 17, 144.

Cohen, S. I., A. J. Silverman, B. Bressler, and B. Shmavonian Problems in isolation studies. In: P. Solomon et al (eds.) Sensory deprivation. Cambridge, Harvard Univ. Press, 1961.

Davis, J. M., W. F. McCourt, and P. Solomon Effect of visual stimulation on hallucinations and other mental experience during sensory deprivation. Amer. J. Psychiat., 1960, 116, 889.

Doane, B. K. Changes in visual function with perceptual isolation. Unpublished Ph.D. Thesis, McGill Univer., 1955.

Doane, B. K., W. Mahatoo, W. Heron, and T. H. Scott Changes in perceptual function after isolation. Canad. J. Psychol., 1959, 13, 210.

Doty, R. W. Potentials evoked in cat cerebral cortex by diffuse and punctiform photic stimuli. J. Neurophysiol., 1958, 21, 437 - 464.

Drake, C. G. and G. W. Stavraky The effect of convulsant agents on partially isolated neurons of the central nervous system. Fed. Proc., 1948, 7, 29.

Eccles, J. C. and A. K. McIntyre The effects of disuse and of activity on mammalian spinal reflexes. J. Physiol., 1953, 121, 492 - 516.

Echlin, F. A., V. Arnett, and J. Zoll Paroxysmal high voltage discharges from the isolated and partially isolated human and animal cortex. Electroenceph. clin. Neurophysiol., 1952, 4, 147.

Fiske, D. W. Effects of monotonous and restricted stimulation. In: D. W. Fiske and S. Maddi (eds.) The functions of varied experience. Homewood, Dorsey Press, 1961, pp. 106 - 144.

Frank, E., N. M. Nothmann, and H. Hirschman-Kaufmann Uber die "tonische" kontraktion des quergestreiften saugetiermuskels nach ausschaltung des motorischen nervan. Pfluger's Arch. ges. Physiol., 1922, 197, 270 - 287.

Freedman, S. J., H. U. Grunebaum, and M. Greenblatt Perceptual and cognitive changes in sensory deprivation. In: P. Solomon et al (eds.) Sensory deprivation. Cambridge, Harvard Univ. Press, 1961.

Goldberger, L. and R. R. Holt Experimental interference with reality contact (perceptual isolation): method and group results. J. nerv. men. Dis.,

1958, 127, 99.

Grafstein, B. and P. B. Sastry Some preliminary electrophysiological studies on chronic neuronally isolated cerebral cortex. Electroenceph. clin. Neurophysiol., 1957, 9, 723 - 725.

Granit, R. Receptors and sensory perception. New Haven, Yale Univ. Press, 1955.

Grunebaum, H. U., S. J. Freedman, and M. Greenblatt Sensory deprivation and personality. Amer. J. Psychiat., 1960, 116, 878.

Hamdi, J. A. and D. Whitteridge The representation of the retina on the optic tectum of the pigeon. Quart. J. exp. Physiol., 1954, 39, 111 - 139.

Hebb, D. O. In the discussion of a symposium - Sensory deprivation: facts in search of a theory. J. nerv. men. Dis., 1961, 132, 40.

Heron, W. The pathology of boredom. Sci. Amer., 1957, 204, 54 - 60.

Heron, W. Cognitive and physiological effects of perceptual isolation. In: P. Solomon et al (eds.) Sensory deprivation. Cambridge, Harvard Univ. Press, 1961.

Katz, B. and R. Miledi Further observations on the distribution of acetylcholine-reactive sites in skeletal muscle. J. Physiol., 1964a, 170, 379 - 388.

Katz, B. and R. Miledi The development of acetylcholine sensitivity in nerve-free segments of skeletal muscle. J. Physiol., 1964b, 170, 389 - 396.

Kenna, J. C. Sensory deprivation: a critical review and explanatory models. Proc. Roy. Soc. Med., 1962, 55, 1005 - 1010.

Kubzanski, P. E. The effects of reduced environmental stimulation on human behavior: a review. In: A. Biderman and H. Zimmer (eds.) The manipulation of human behavior: the case for interrogation. New York, Wiley, 1961, pp. 51 - 95.

Kuffler, S. W. Discharge patterns and functional organization of the mammalian retina. J. Neurophysiol., 1953, 16, 37 - 68.

Lilly, J. C. Mental effects of reduction of ordinary levels of physical stimuli on intact healthy persons. Psychiat. Res. Rep., 1956, 5, 1.

Lindsley, D. B. Common factors in sensory deprivation, sensory distortion, and sensory overload. In: P. Solomon et al (eds.) Sensory deprivation. Cambridge, Harvard Univ. Press, 1961.

Malis, L. T. and L. Kruger Multiple response and excitability of cat's visual cortex. J. Neurophysiol., 1956, 19, 172 - 186.

Marshall, W. H., S. H. Talbot, and H. W. Ades Cortical

responses of the unanesthetized cat to photic and electrical afferent stimulation. J. Neurophysiol., 1943, 6, 1 - 15.

Morrell, F. et al. Excitability of the mirror focus. Electroenceph. clin. Neurophysiol., 1960, 12, 241.

Morrison, R. and W. Heron The effects of localized skin deprivation on cutaneous detectability. Paper read at Eastern Psychol. Ass., Atlantic City, May, 1962.

Philipeaux, J. M. and A. Vulpian Note sur une modification physiologique qui se produit dans le nerf lingual par suite de l'abolition temporaire de la motricita dans le nerf hypoglosse du meme cote. Compt. rend. acad. sci. Paris, 1863, 56, 1009 - 1011.

Posternak, J. M., T. C. Fleming, and E. V. Evarts Effect of interruption of the visual pathway on the response to geniculate stimulation. Science, 1959, 129, 39.

Ramon y Cajal, P. Lobulos opticos de las aves. Trab. Inst. Cajal Invest. biol., 1943, 35, 3 - 20.

Rougeul, A. Exploration oscillographique de la voie visuelle du pigeon. Paris, Foulon, 1957.

Scott, T. H., W. H. Bexton, W. Heron, and B. K. Doane Cognitive effects of perceptual isolation. Canad. J. Psychol., 1959, 13, 200.

Seguin, J. J. and G. W. Stavraky The effects of barbituates

on partially isolated regions of the central nervous system. Canad. J. Biochem. Physiol., 1957, 35, 667 - 680.

Sharpless, S. K. and L. M. Halpern The electrical excitability of chronically isolated cortex by means of permanently implanted electrodes. Electroenceph. clin. Neurophysiol., 1962, 14, 244.

Smith, S. and W. Lewty Perceptual isolation using a silent room. Lancet II, 1959, 342.

Solomon, P. et al. (eds.) Sensory deprivation. Cambridge, Harvard Univ. Press, 1961.

Spiegel, E. A. and E. G. Szekely Supersensitivity of the cortex following partial deafferentation (lesion of the posterior thalamic nuclei). Electroenceph. clin. Neurophysiol., 1955, 7, 375 - 381.

Stavraky, E. W. Supersensitivity following lesions of the nervous system. Toronto, Univ. of Toronto Press, 1961.

Thesleff, S. Effects of motor innervation on the chemical sensitivity of skeletal muscle. Physiol. Revs., 1960, 40, 734 - 752.

van Eyck, M. Etude electromyographique des reflexes labyrinthiques cervicaux du Pigeon. Acta otolaryng. Stockh., 1953, 43, 300 - 310.

Zubek, J. P. Behavioral and EEG changes following 14 days

of perceptual isolation. Psychon. Sci., 1964, 1, 57.

Zubek, J. P. Effects of prolonged sensory and perceptual deprivation. Brit. med. Bull., 1964, 20, 38.

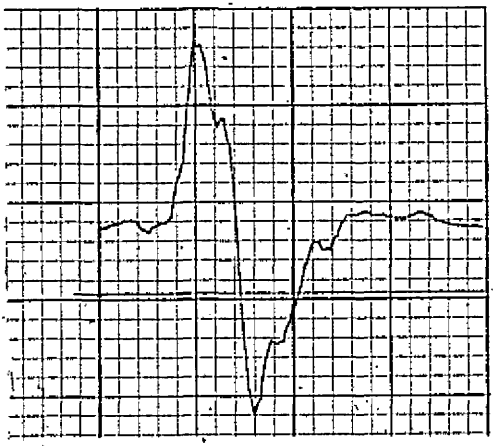
APPENDIX 1

The computer averages for the evoked responses recorded in the PRE, DEP, and POST sessions both with and without background illumination. (Experimental eye only; Calibrations: given for the individual figures)

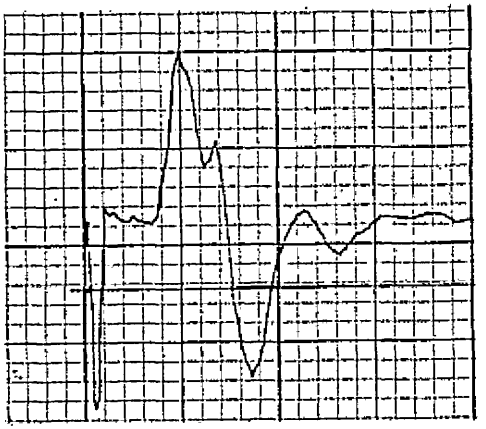
Bird 1 Calibration: 100 microvolts, PRE and DEP negative up; POST negative down; 50 msec.

PRE

NO BACKGROUND

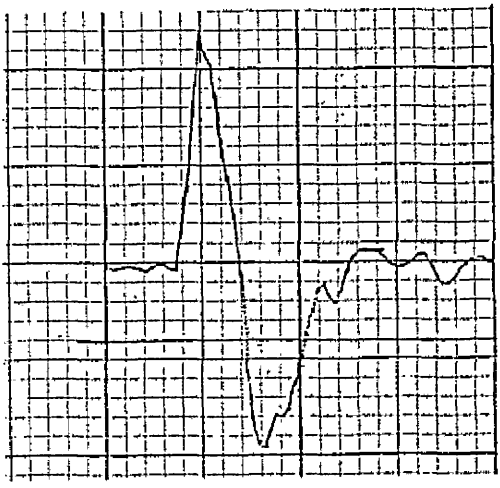


BACKGROUND

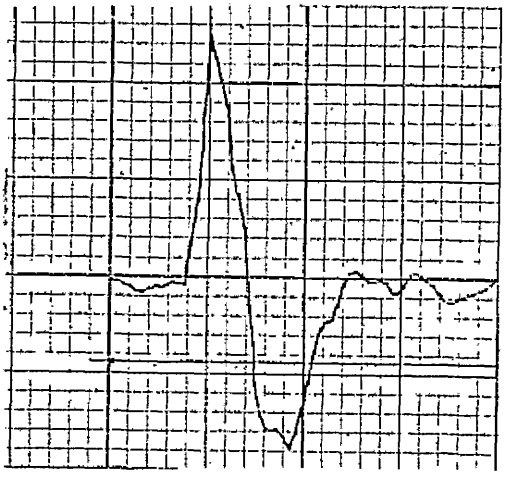


DEP

NO BACKGROUND

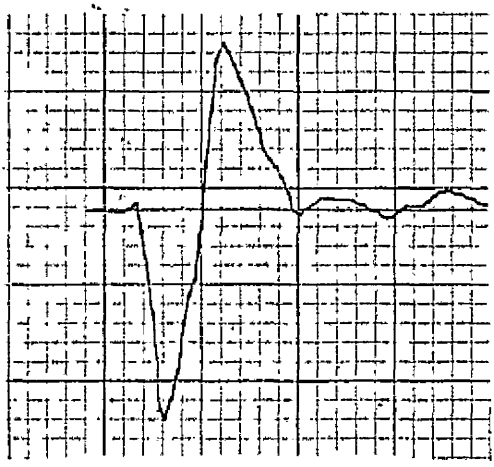


BACKGROUND

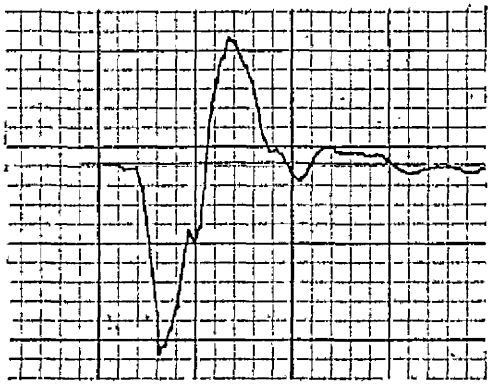


POST

NO BACKGROUND



BACKGROUND

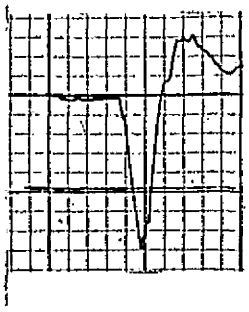


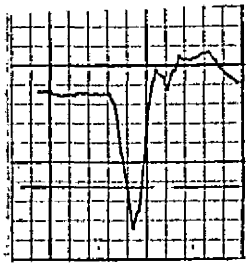
Bird 93 Calibrations: 50 microvolts, negative down;
50 msec.

NO BACKGROUND

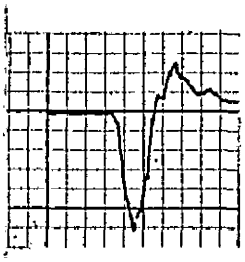
BACKGROUND

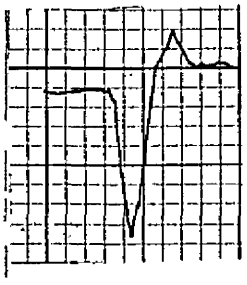
PRE



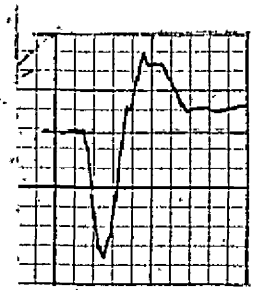


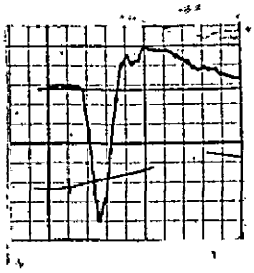
DEP





POST

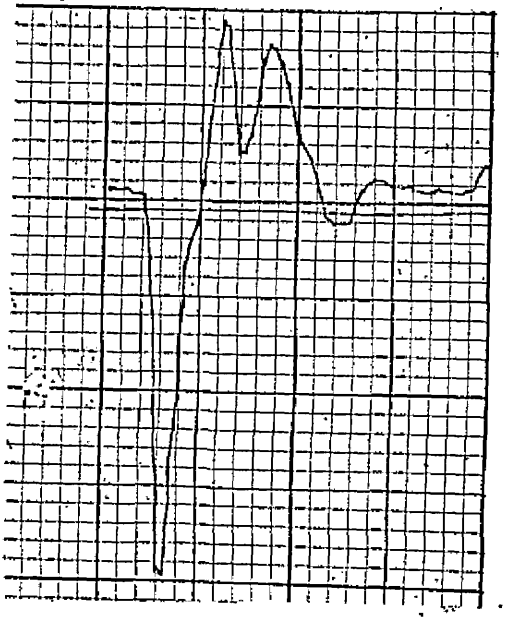




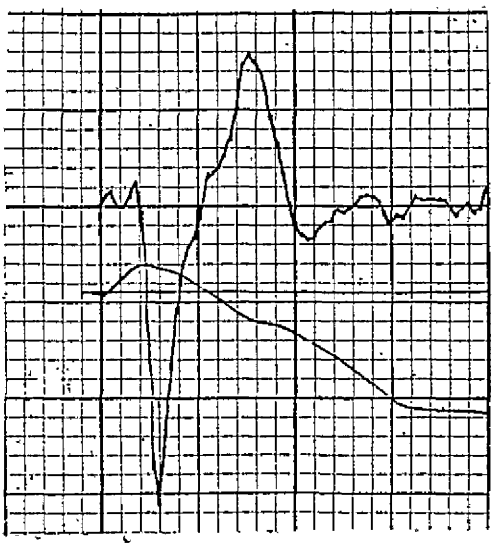
Bird 32 Calibrations: 100 microvolts, negative down; 50 msec.

PRE

NO BACKGROUND

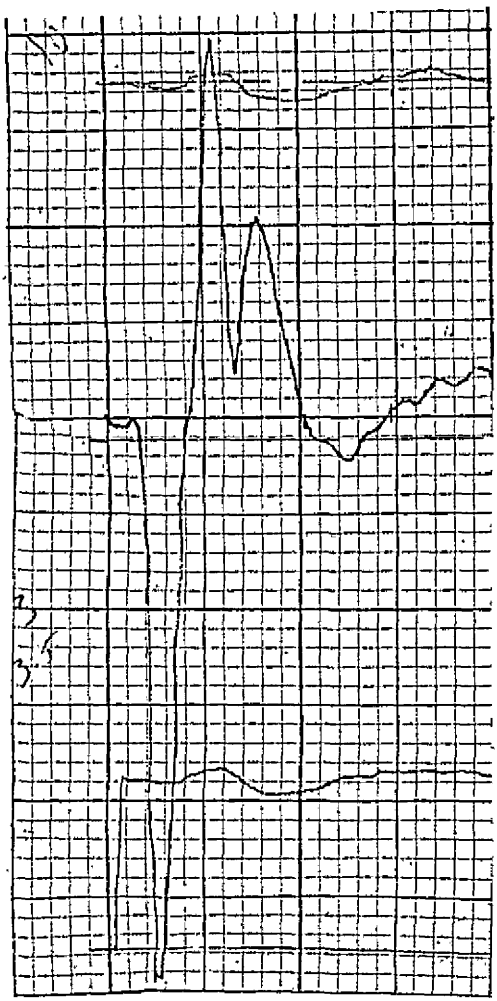


BACKGROUND

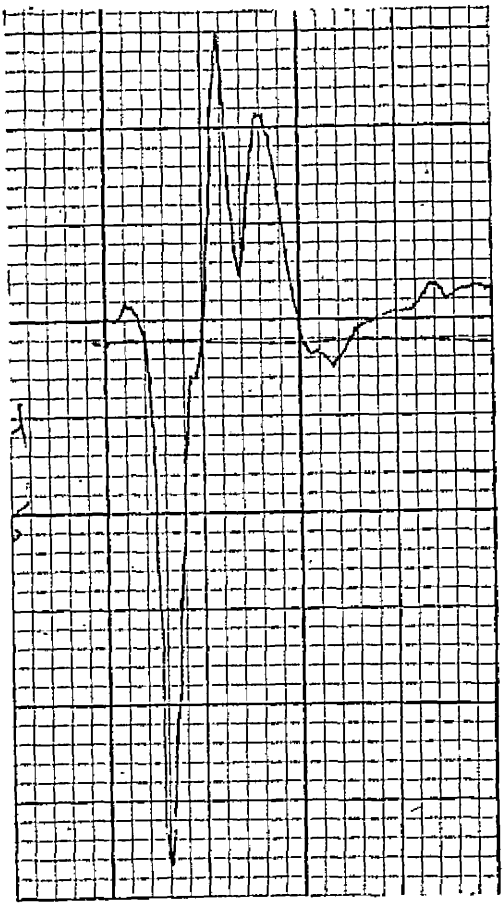


DEP

NO BACKGROUND

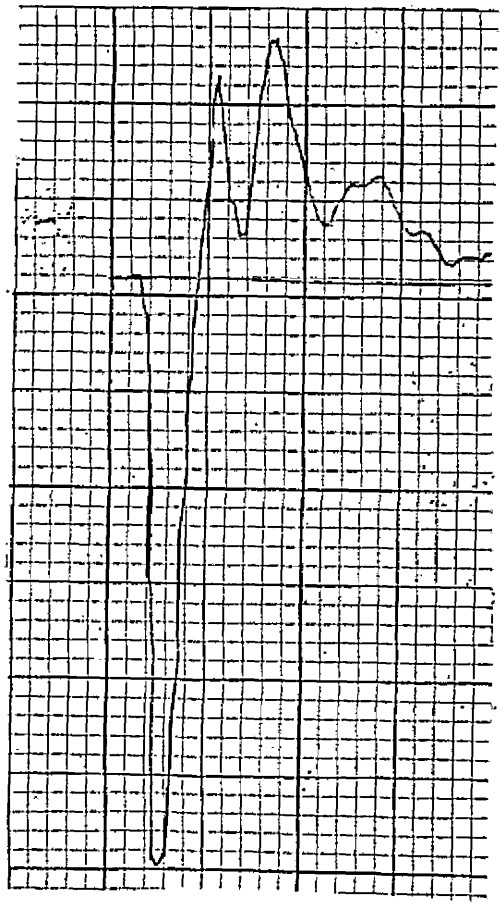


BACKGROUND

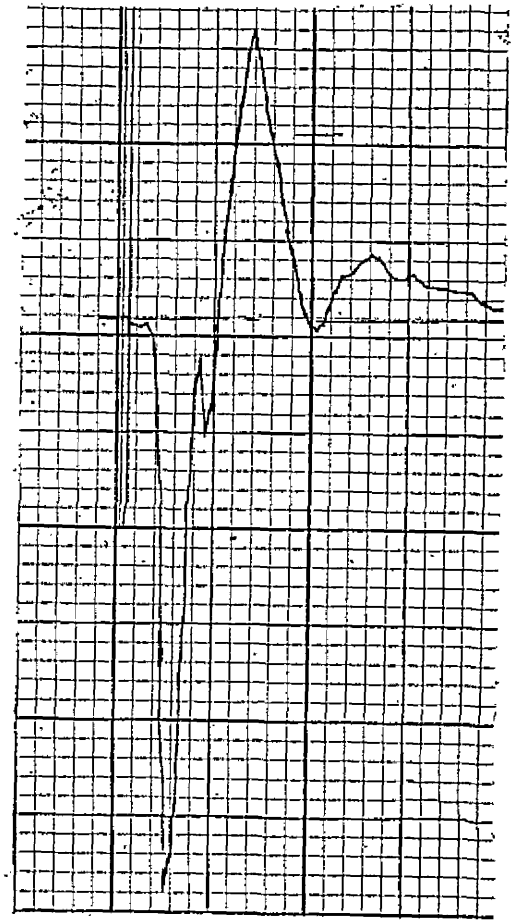


POST

NO BACKGROUND

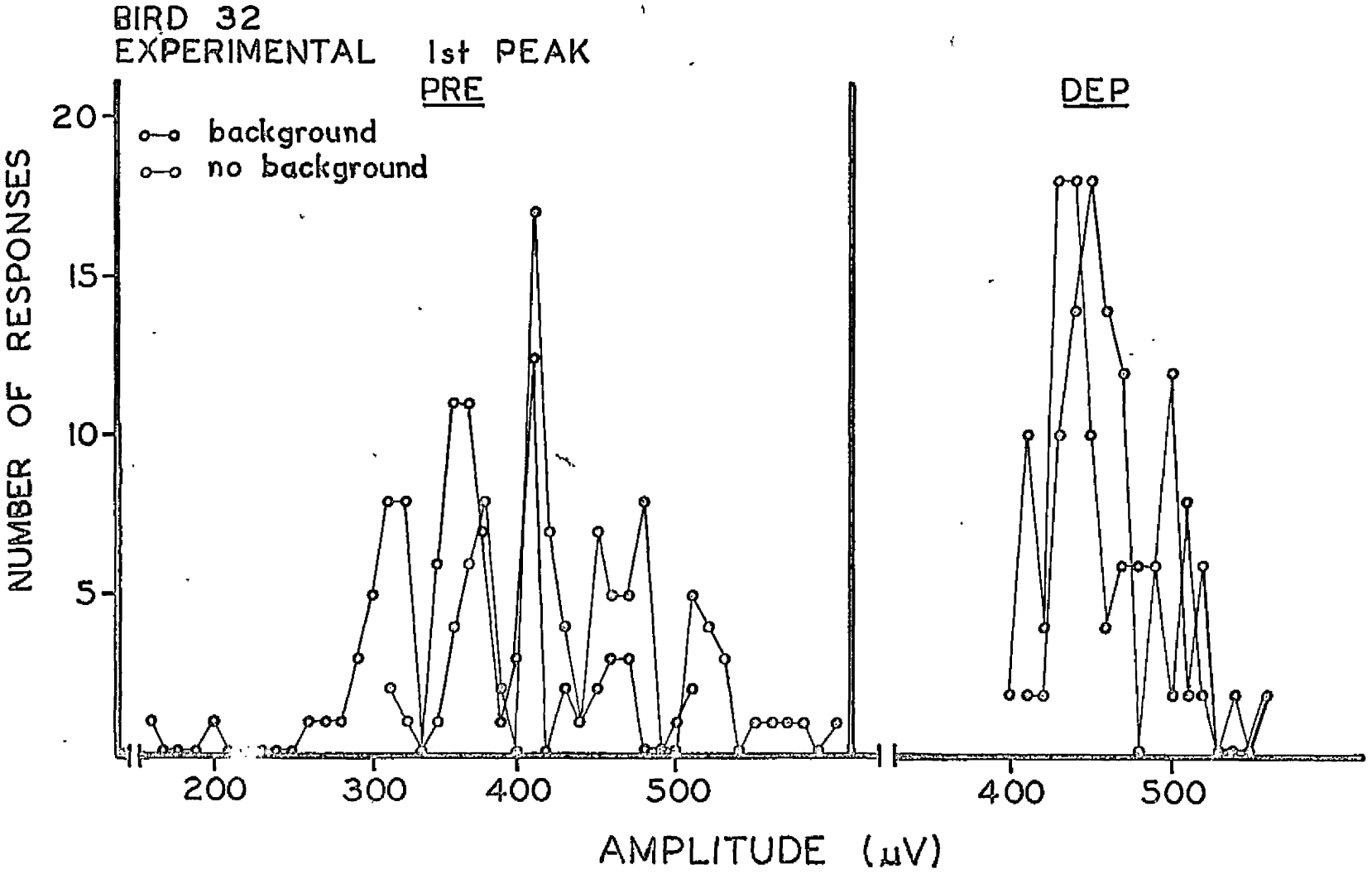


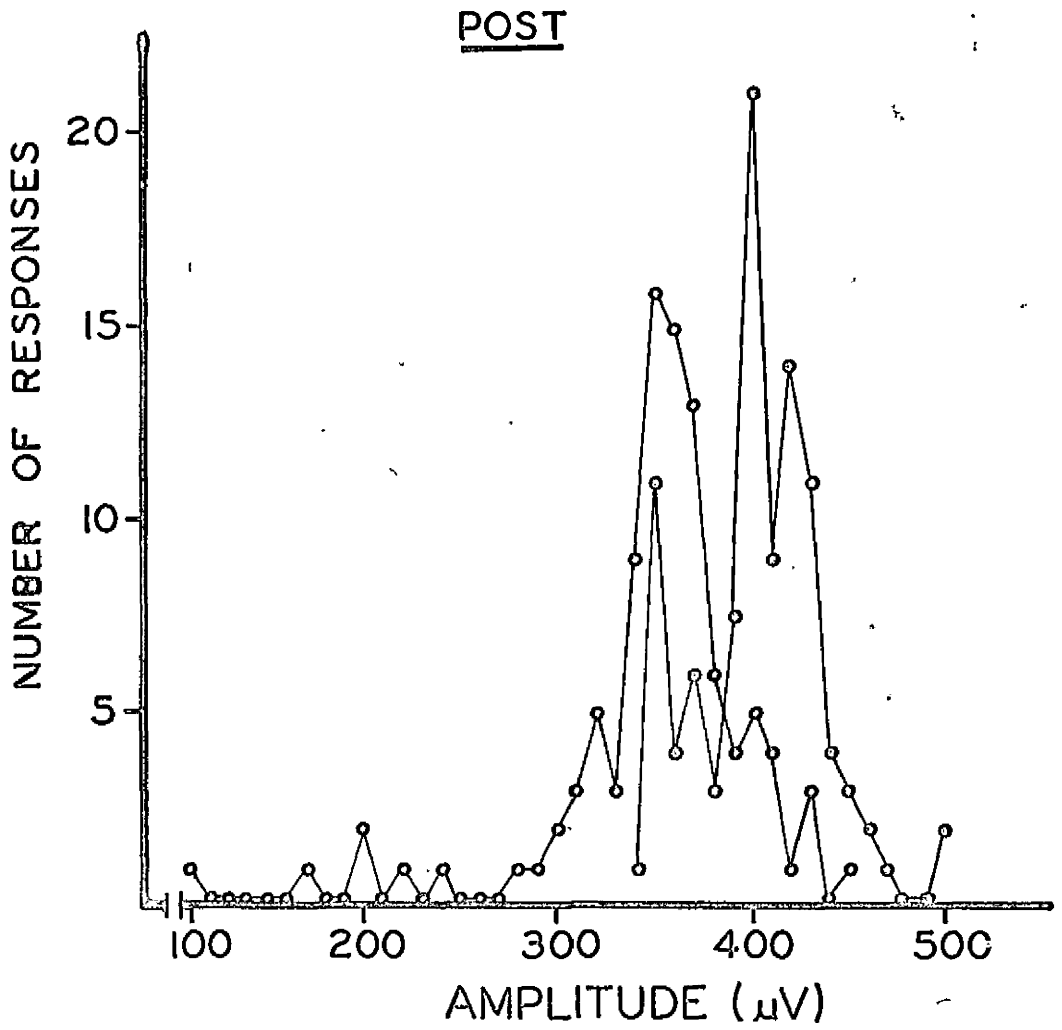
BACKGROUND

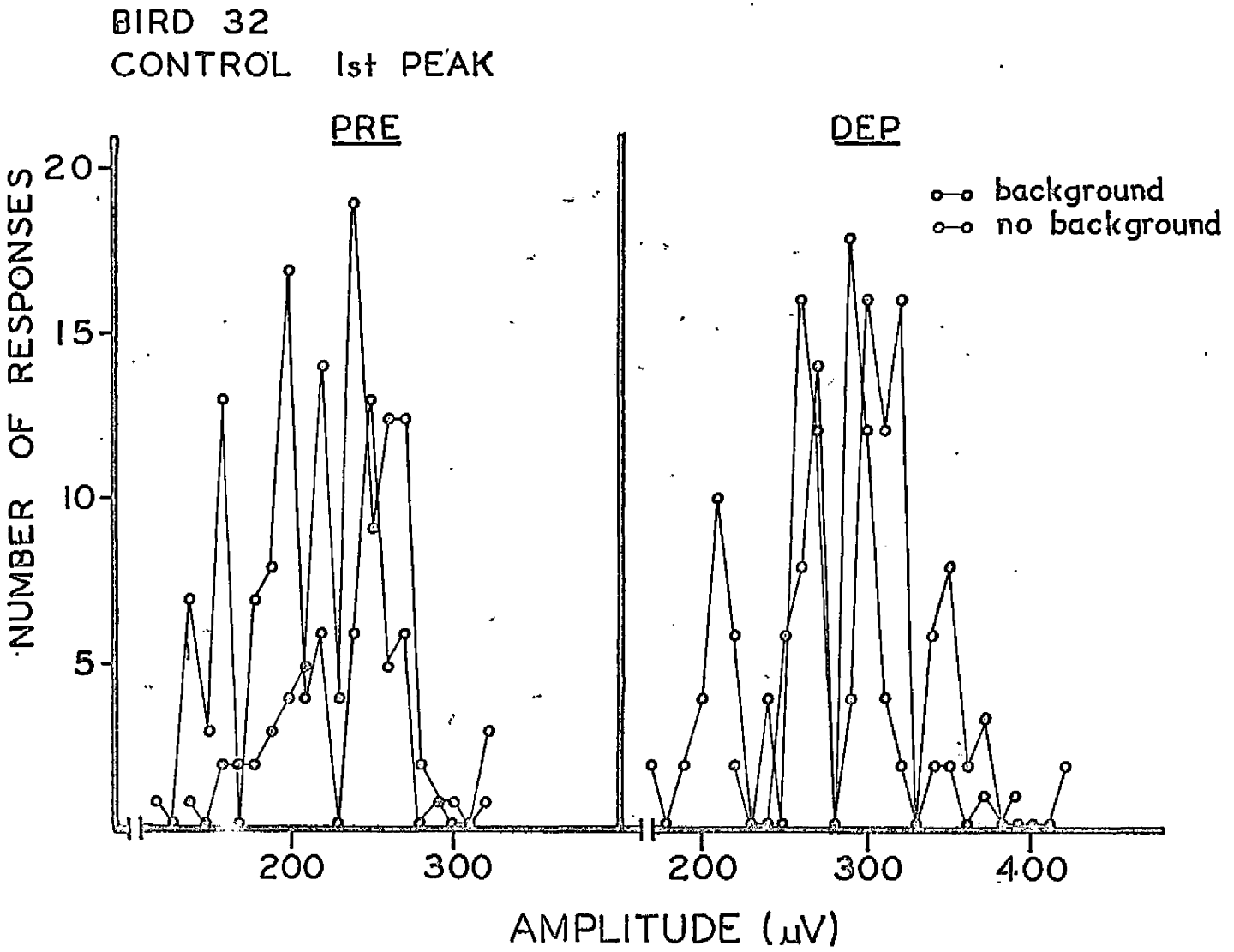


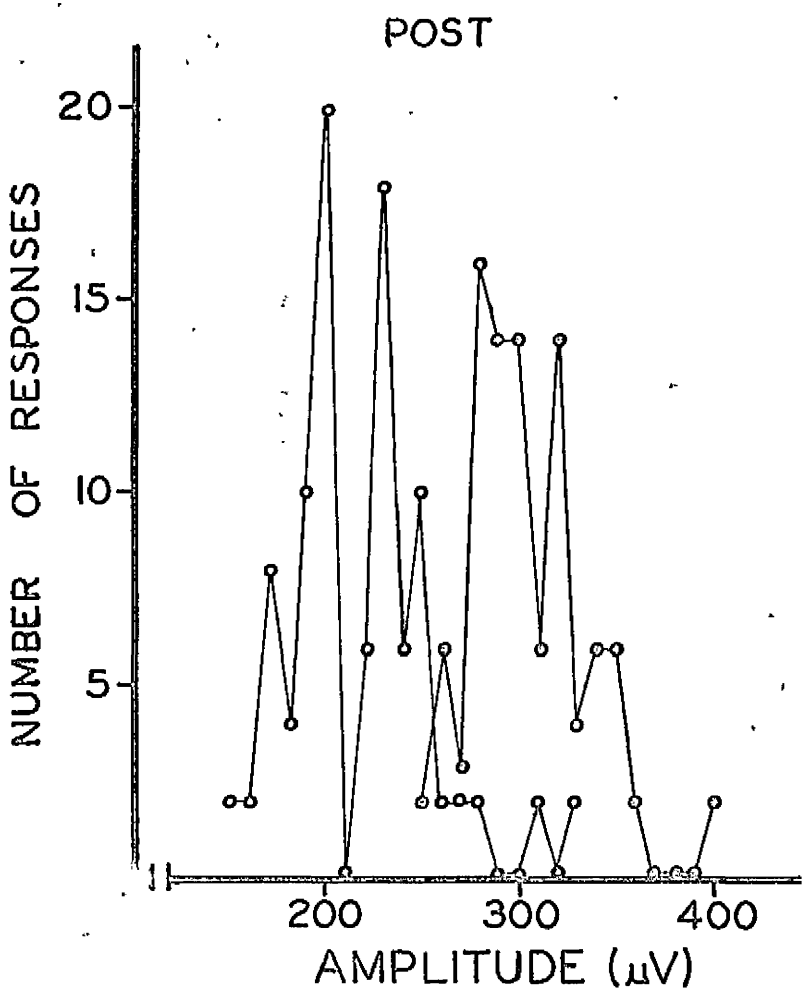
APPENDIX 2

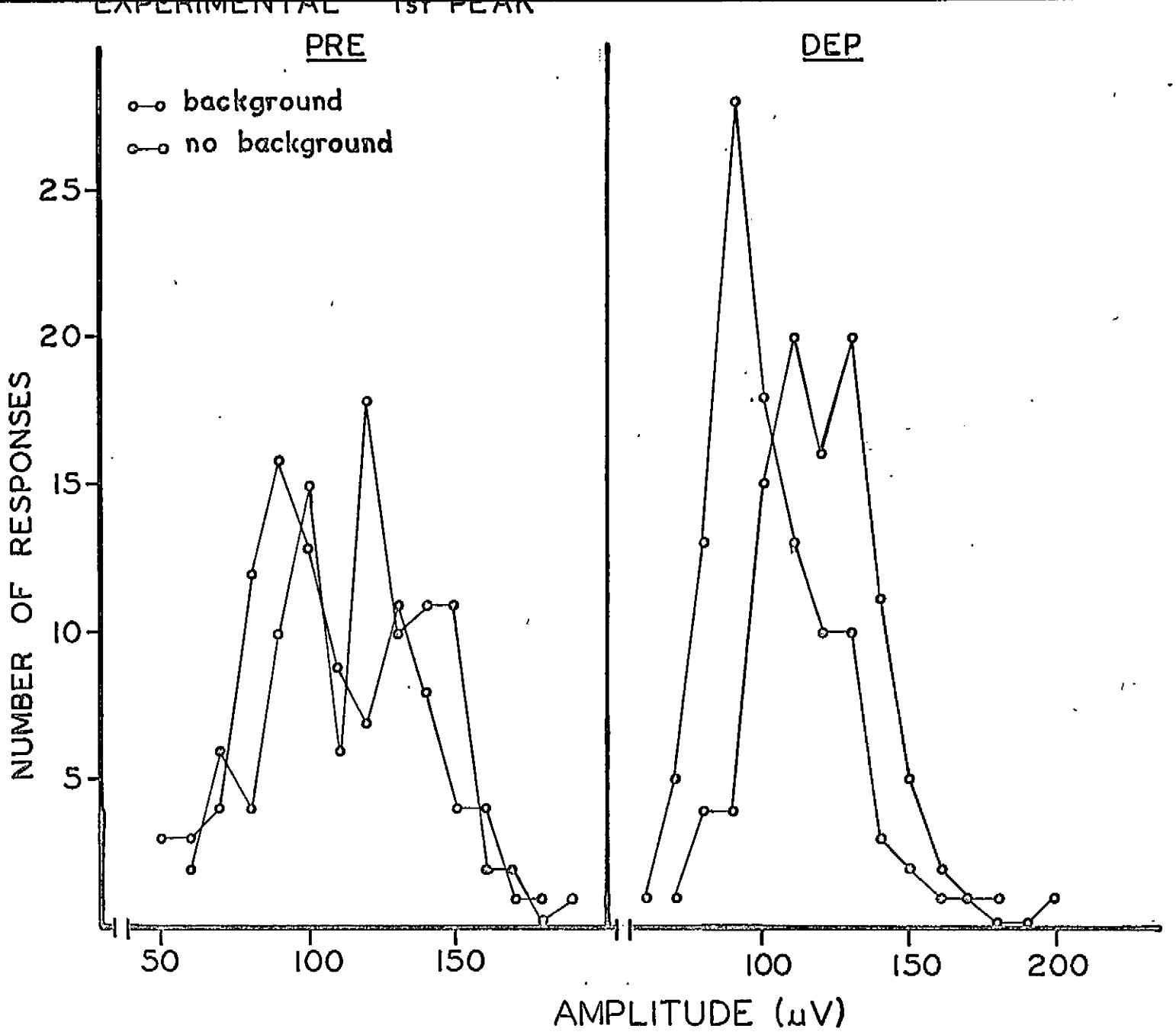
Frequency distribution for the amplitudes of the primary peak.

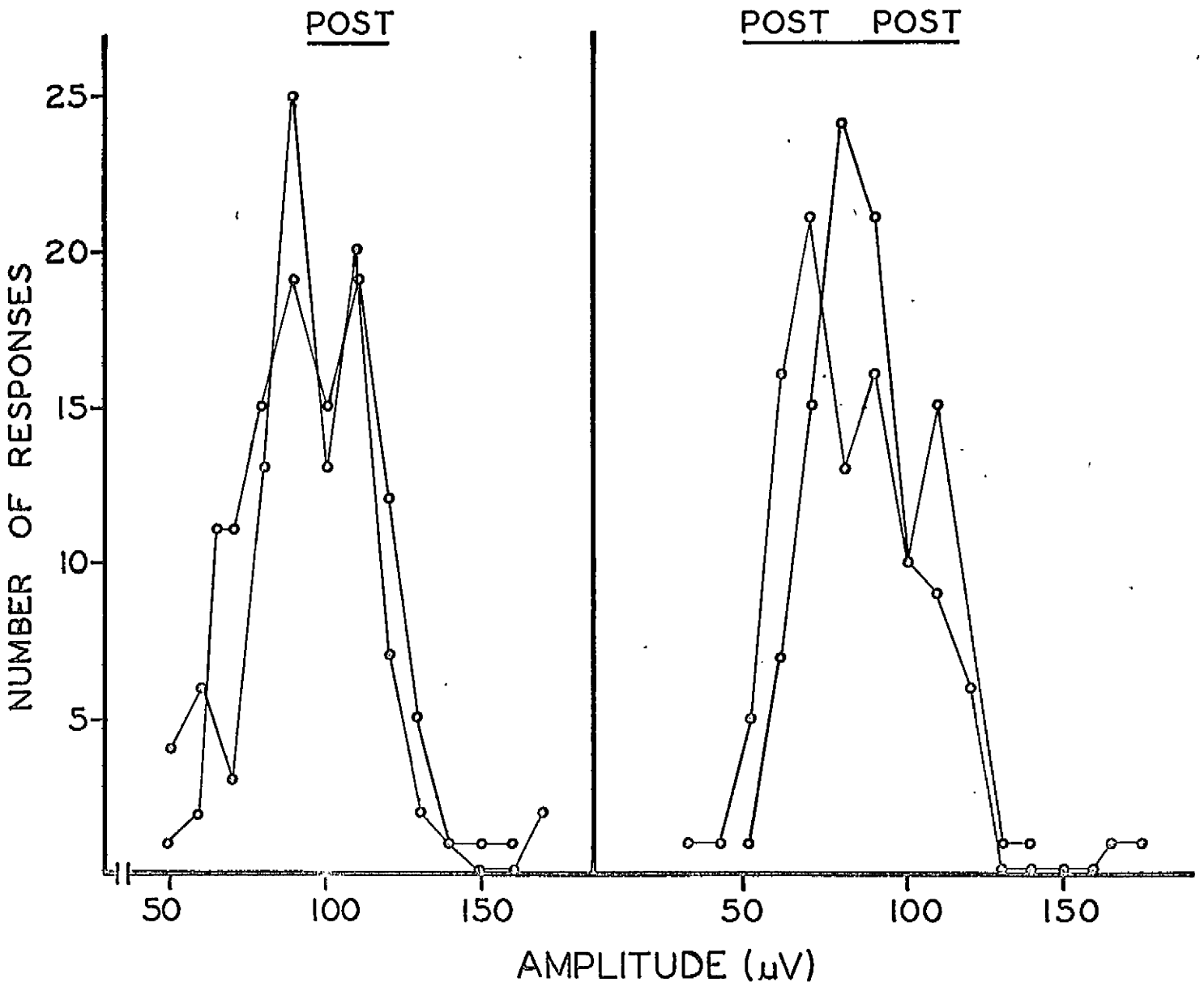

BIRD 32
EXPERIMENTAL 1st PEAK
PRE
DEP
background
no background
NUMBER OF RESPONSES
20
15
10
5
200
300
400
500
400
500
AMPLITUDE (μV)

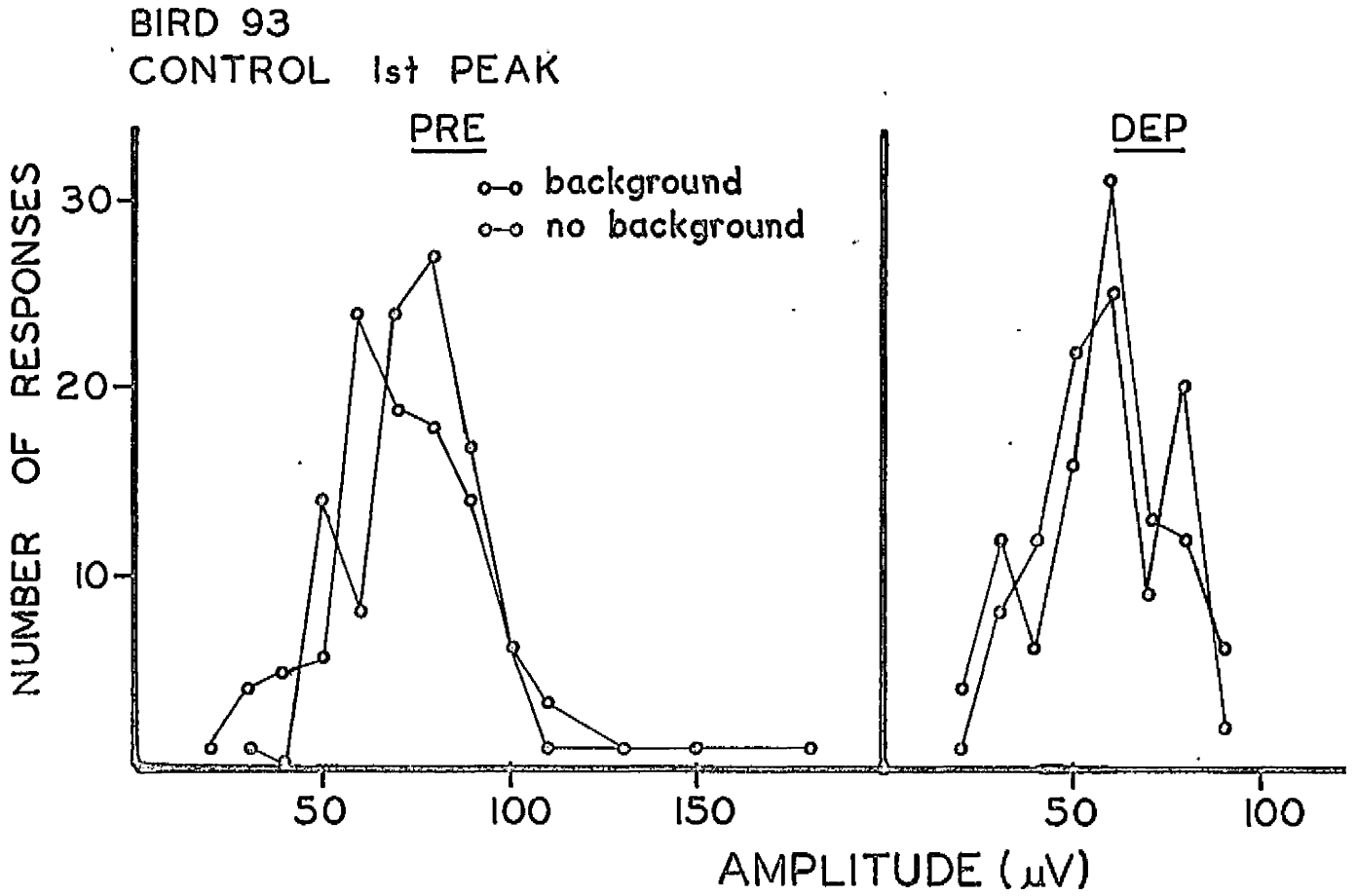

POST
NUMBER OF RESPONSES
20
15
10
5
100
200
300
400
500
AMPLITUDE (μV)

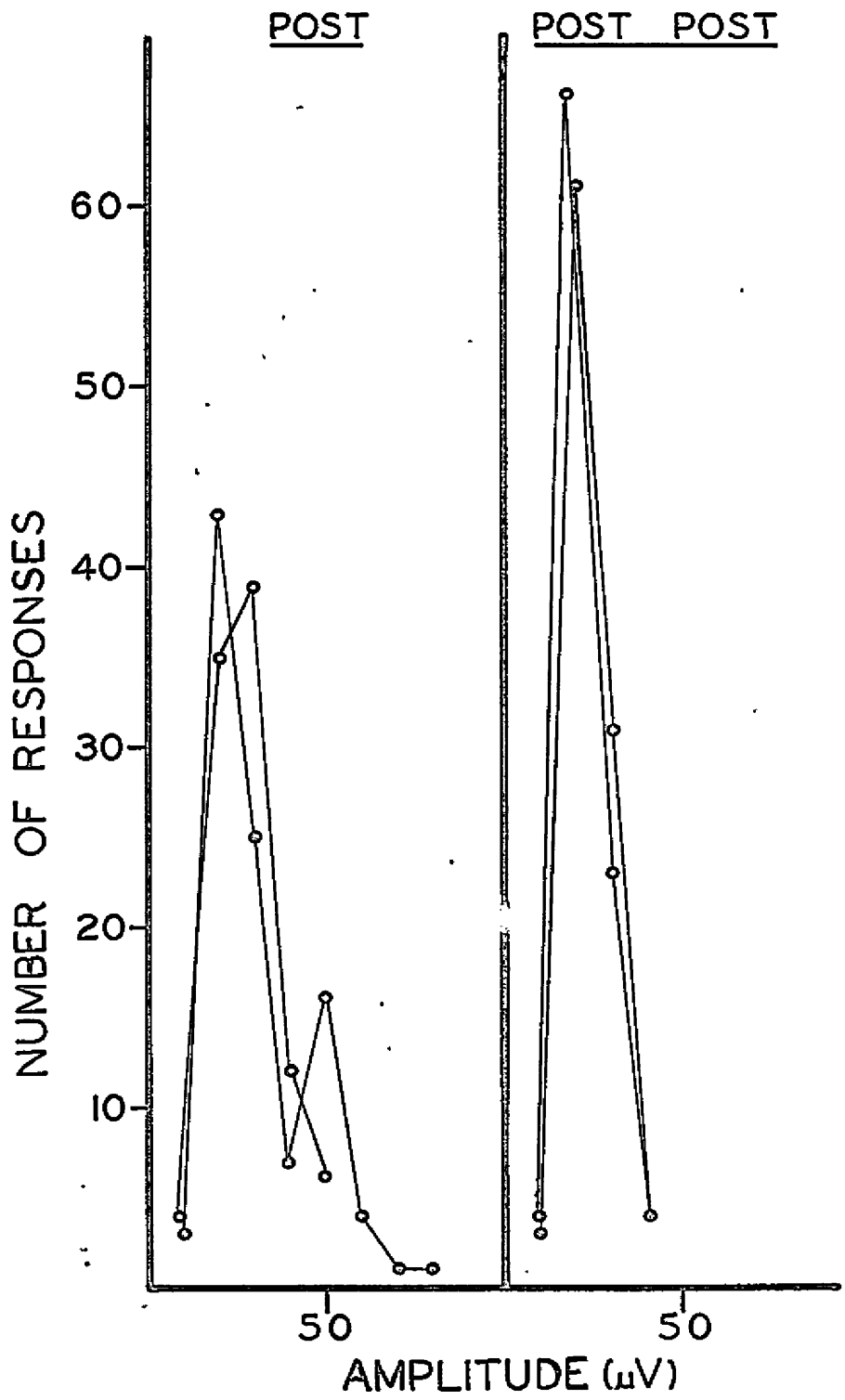

BIRD 32
CONTROL 1st PEAK
PRE
DEP
background
no background
NUMBER OF RESPONSES
20
15
10
5
200
300
200
300
400
AMPLITUDE (μV)

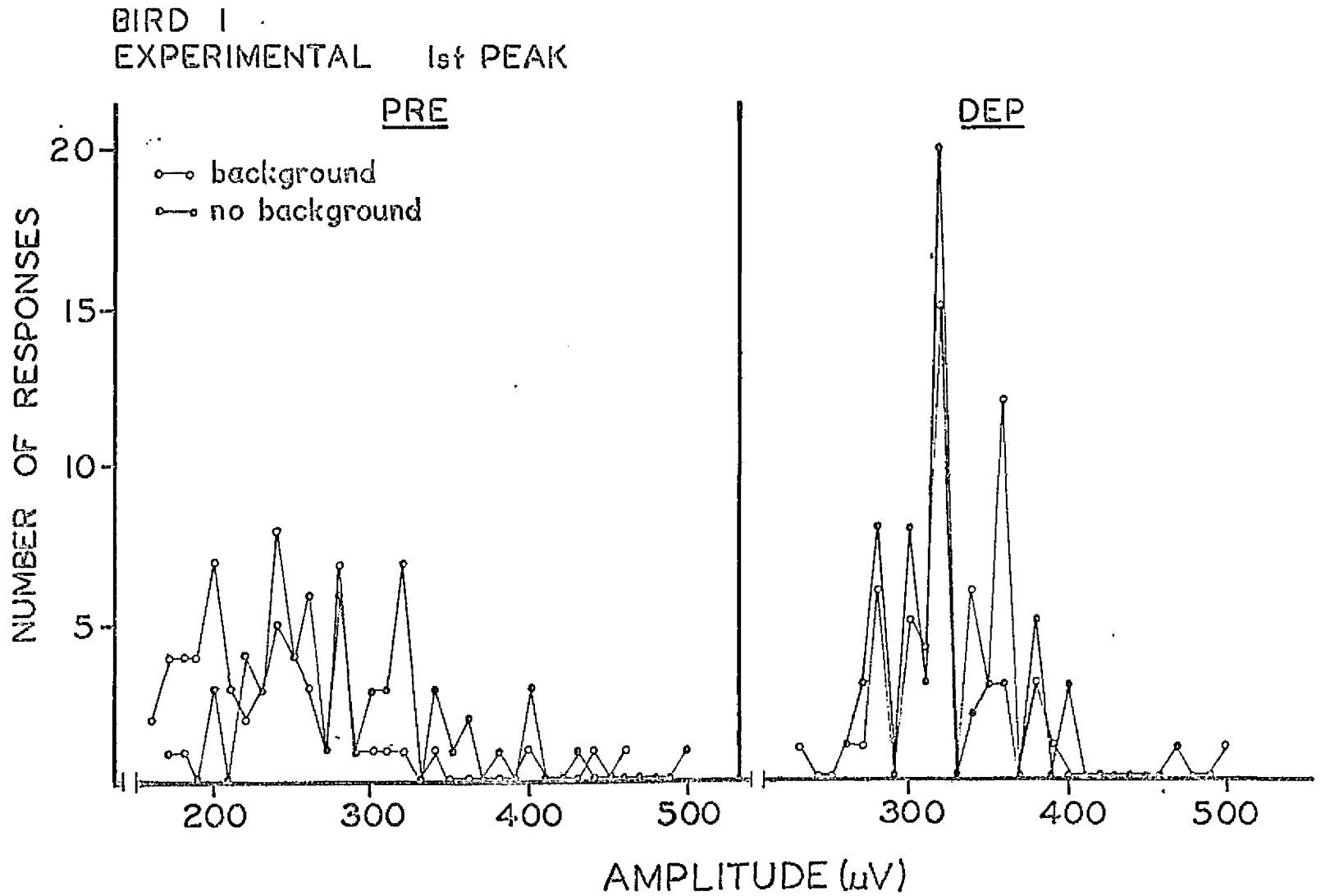

POST
NUMBER OF RESPONSES
20
15
10
5
1
200
300
400
AMPLITUDE (μV)

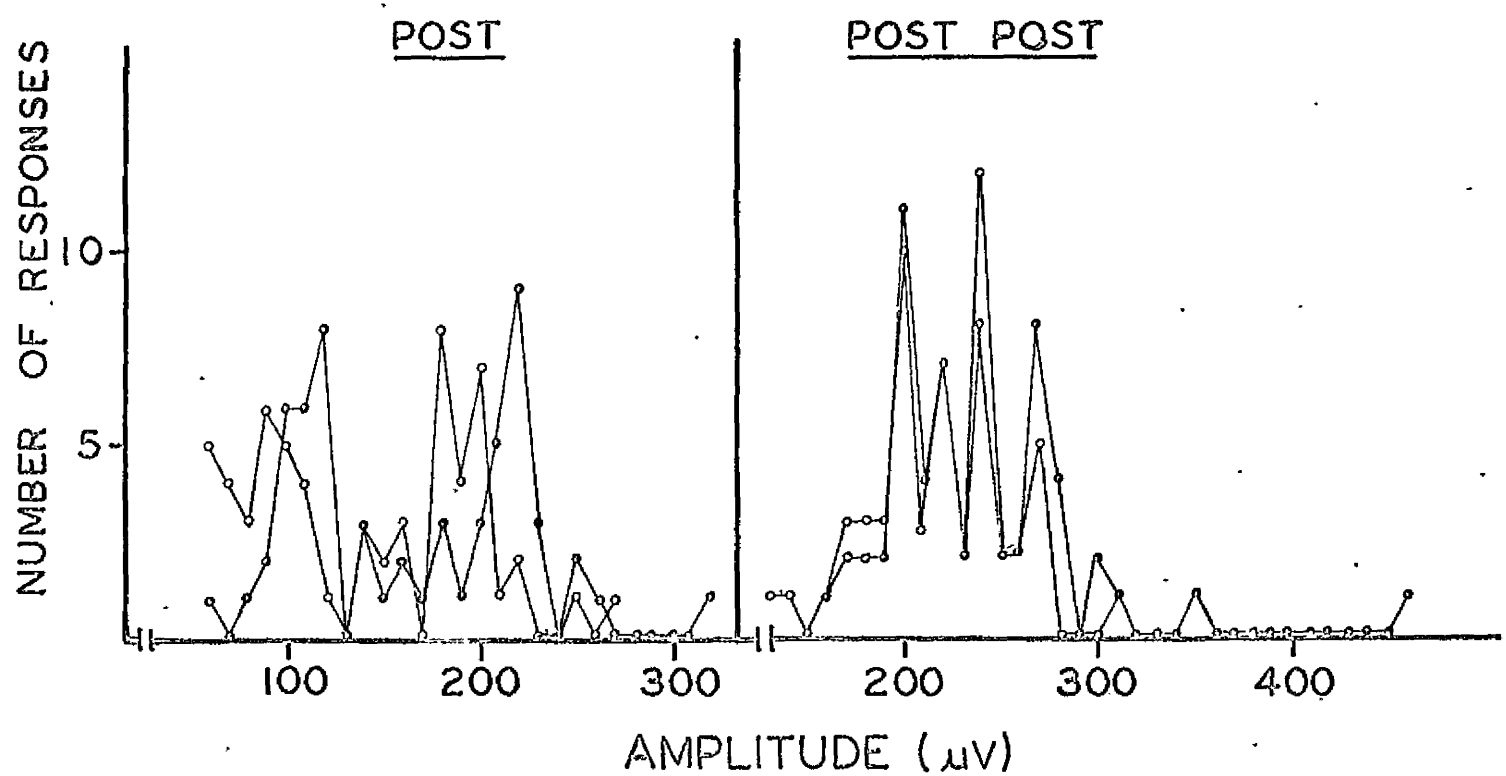

EXPERIMENTAL 1st PEAK
PRE
DEP
background
no background
NUMBER OF RESPONSES
25
20
15
10
5
50
100
150
100
150
200
AMPLITUDE (μV)

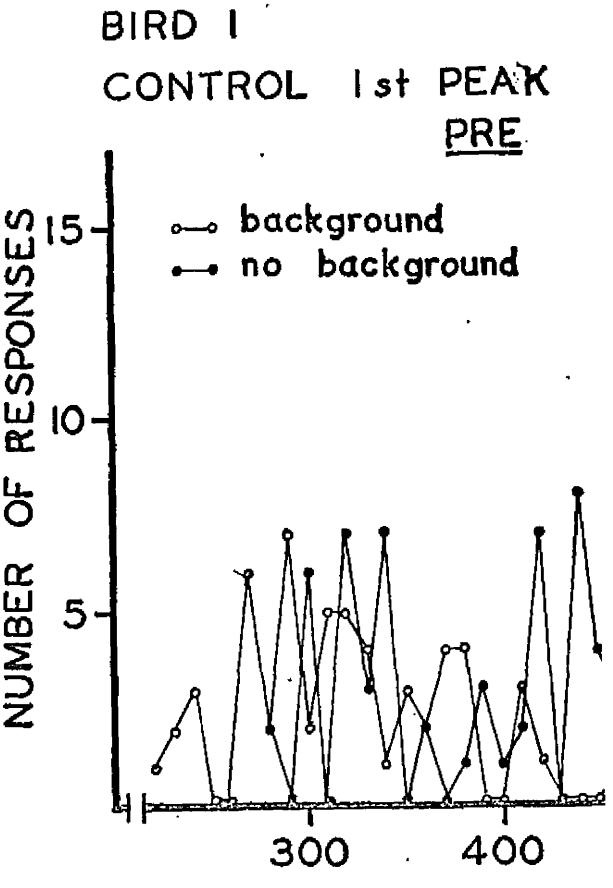

POST
POST POST
NUMBER OF RESPONSES
25
20
15
10
5
50
100
150
50
100
150
AMPLITUDE (μV)

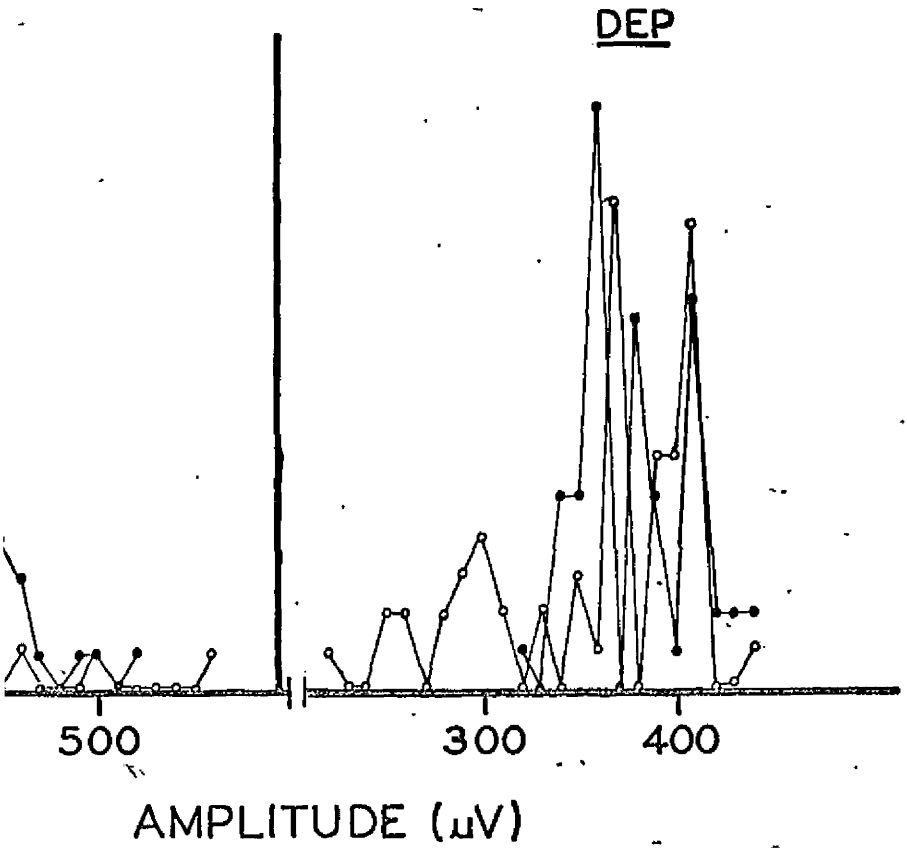

BIRD 93
CONTROL 1st PEAK
PRE
DEP
o–o background
o–o no background
NUMBER OF RESPONSES
30
20
10
50
100
150
50
100
AMPLITUDE (μV)

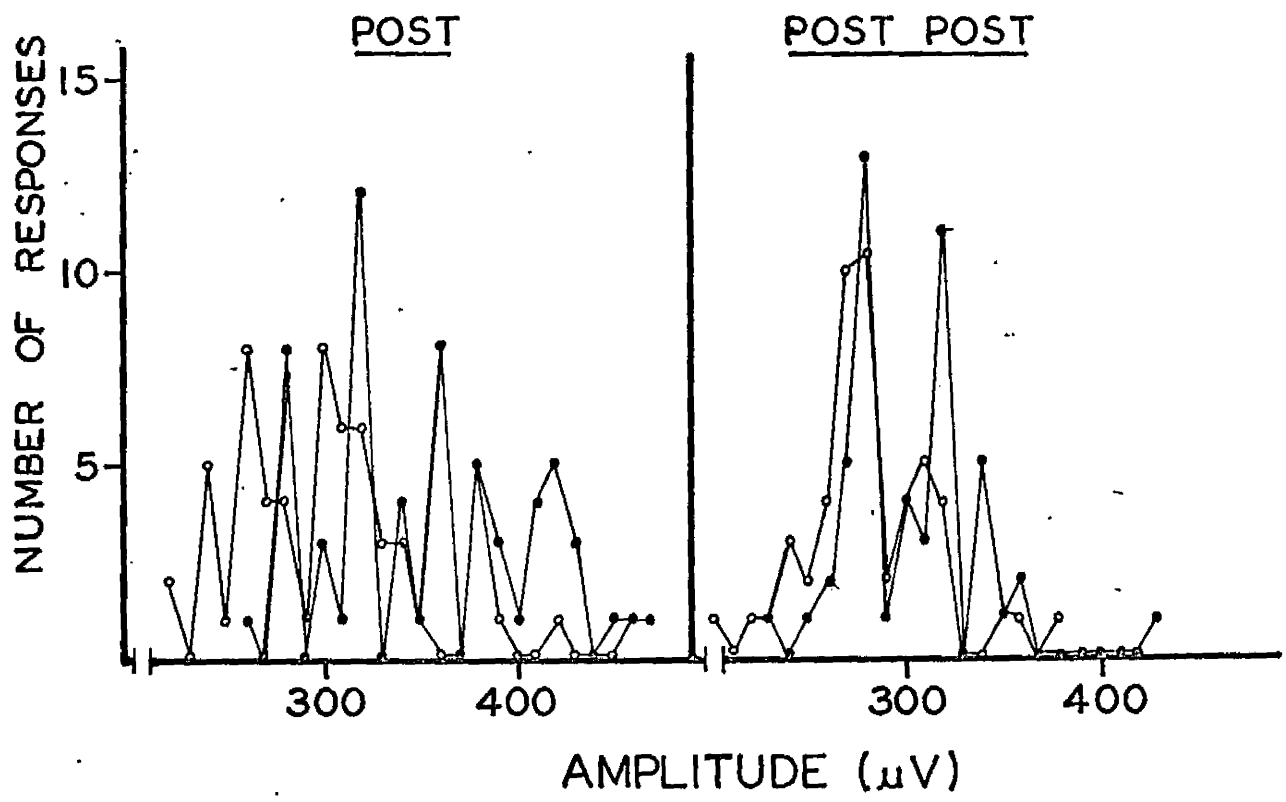

POST
POST POST
60
50
40
30
20
10
NUMBER OF RESPONSES
50
50
AMPLITUDE (μV)


BIRD I
EXPERIMENTAL 1st PEAK
PRE
DEP
background
no background
NUMBER OF RESPONSES
20
15
10
5
200
300
400
500
300
400
500
AMPLITUDE (μV)


POST
POST POST
NUMBER OF RESPONSES
10
5
100
200
300
200
300
400
AMPLITUDE (μV)

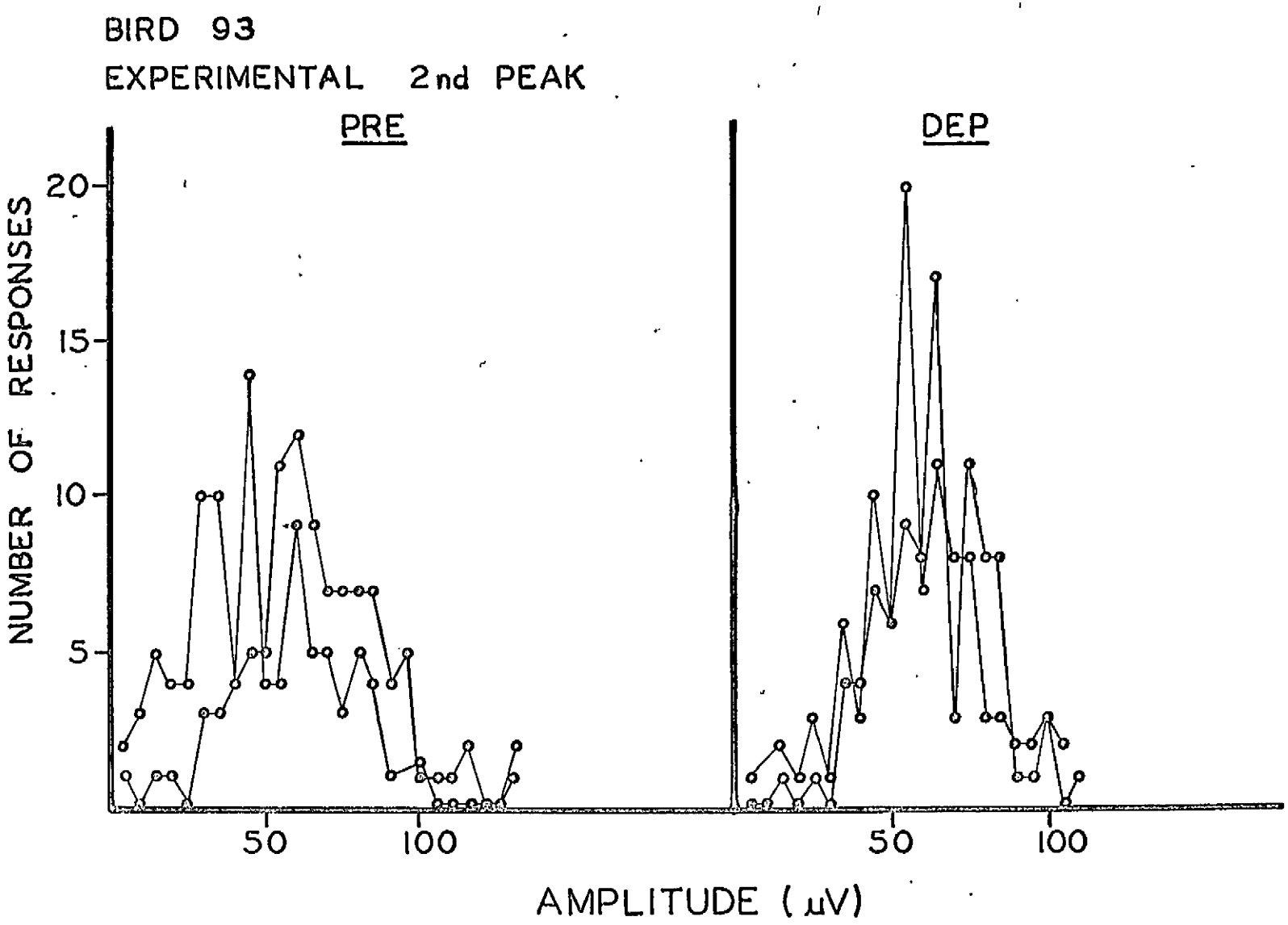

BIRD I
CONTROL 1st PEAK
PRE
background
no background
NUMBER OF RESPONSES
15
10
5
300
400

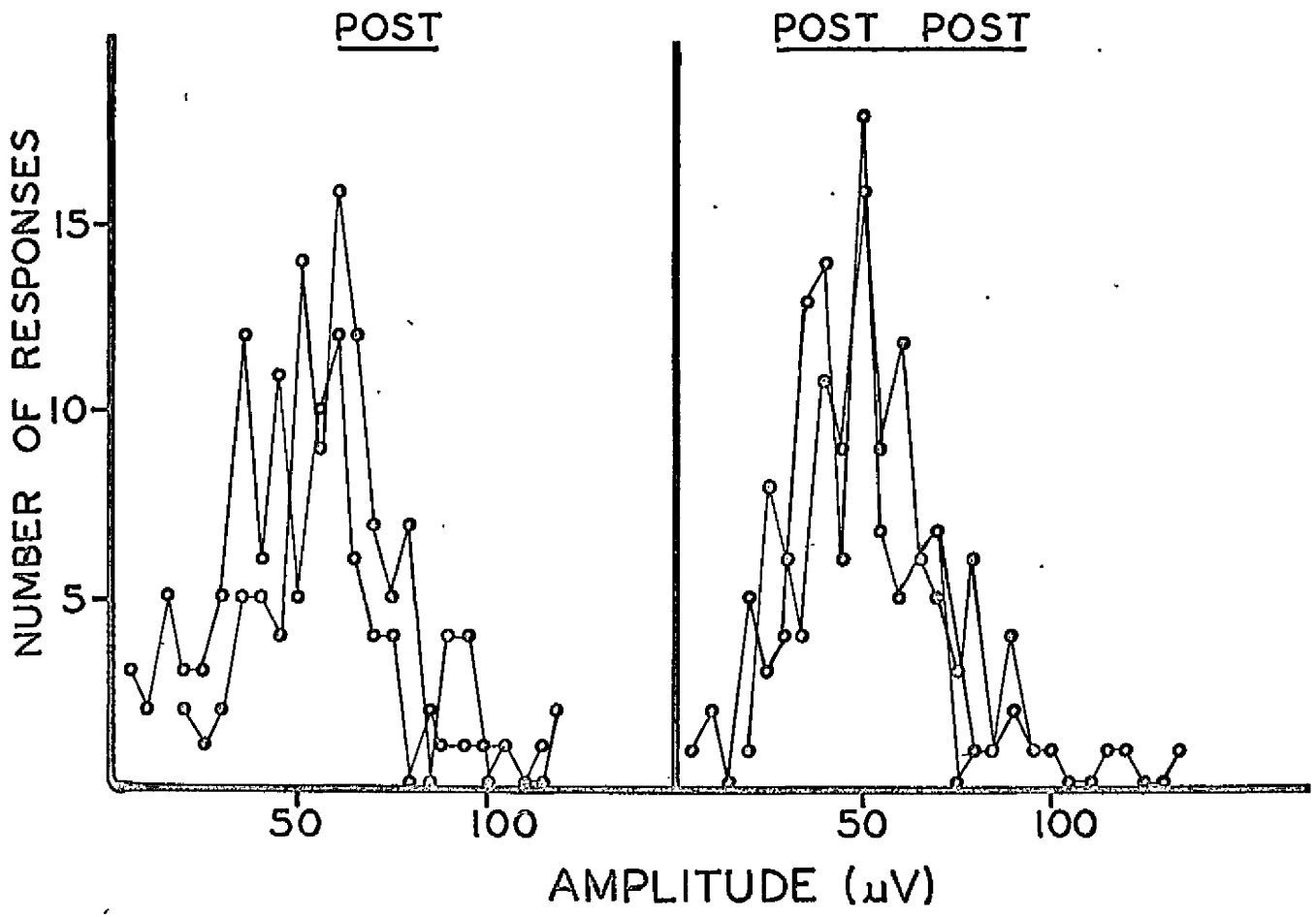

DEP
500
300
400
AMPLITUDE (μV)

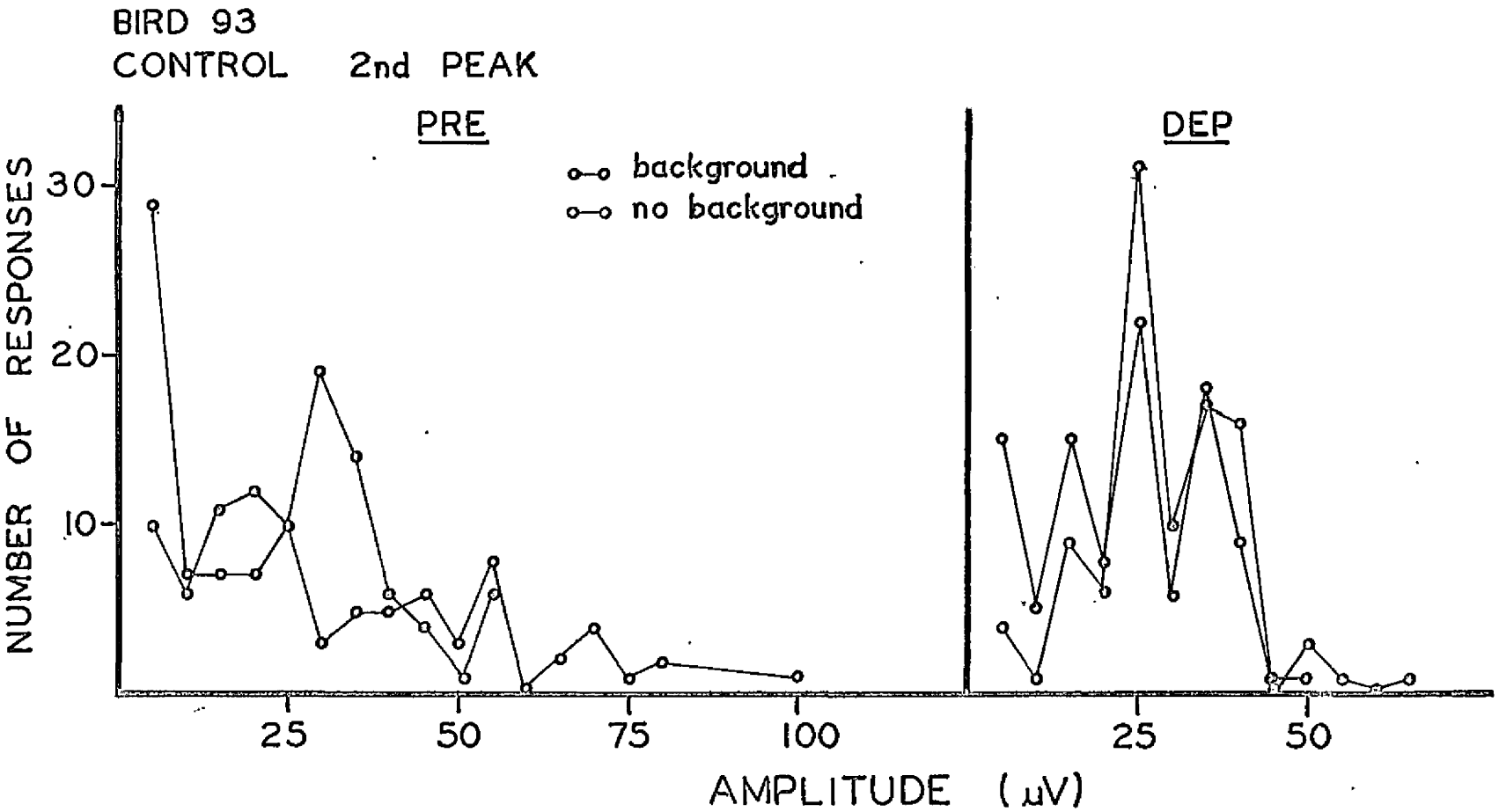

POST
POST POST
NUMBER OF RESPONSES
15
10
5
300
400
300
400
AMPLITUDE (μV)

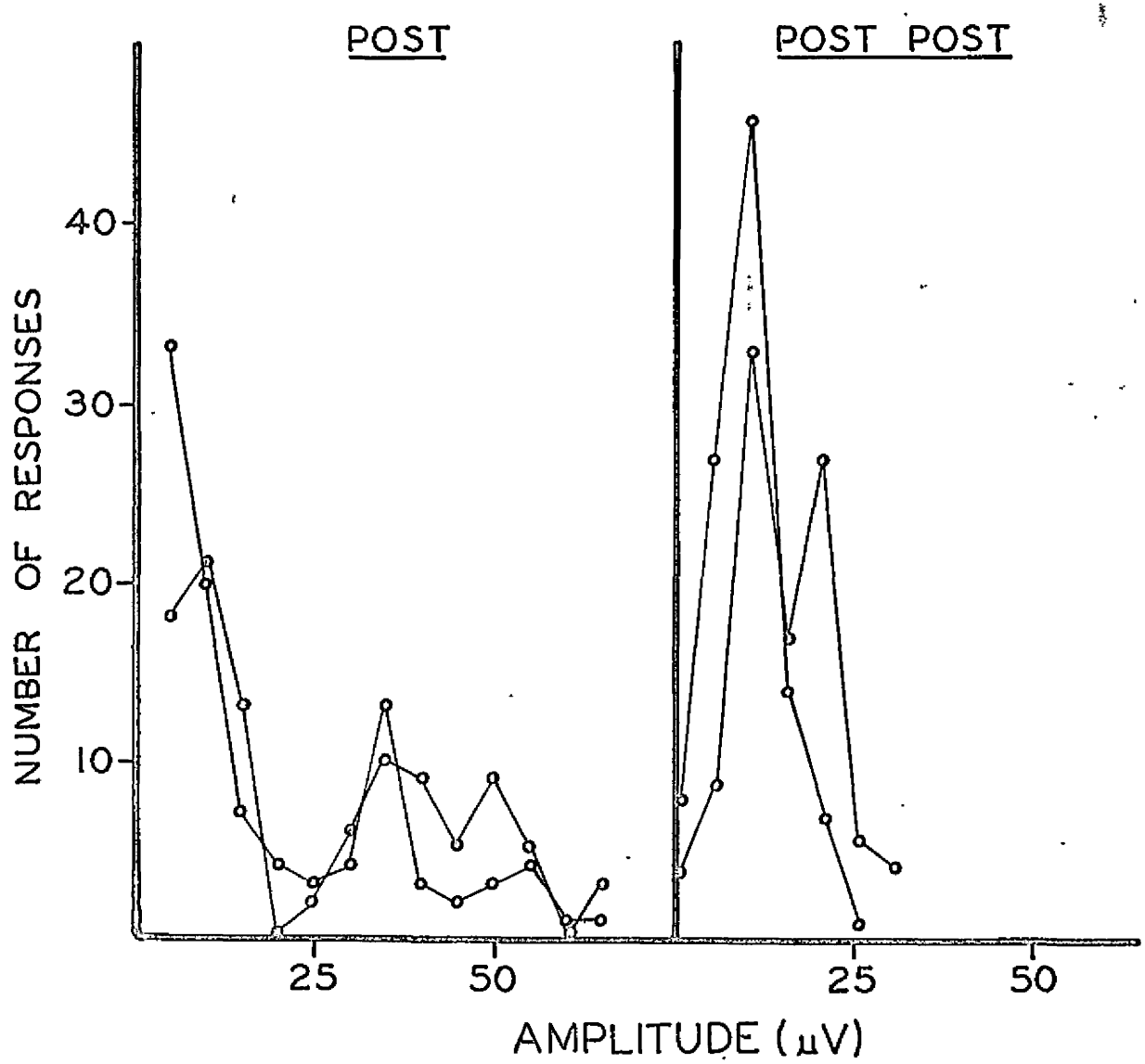
APPENDIX 3

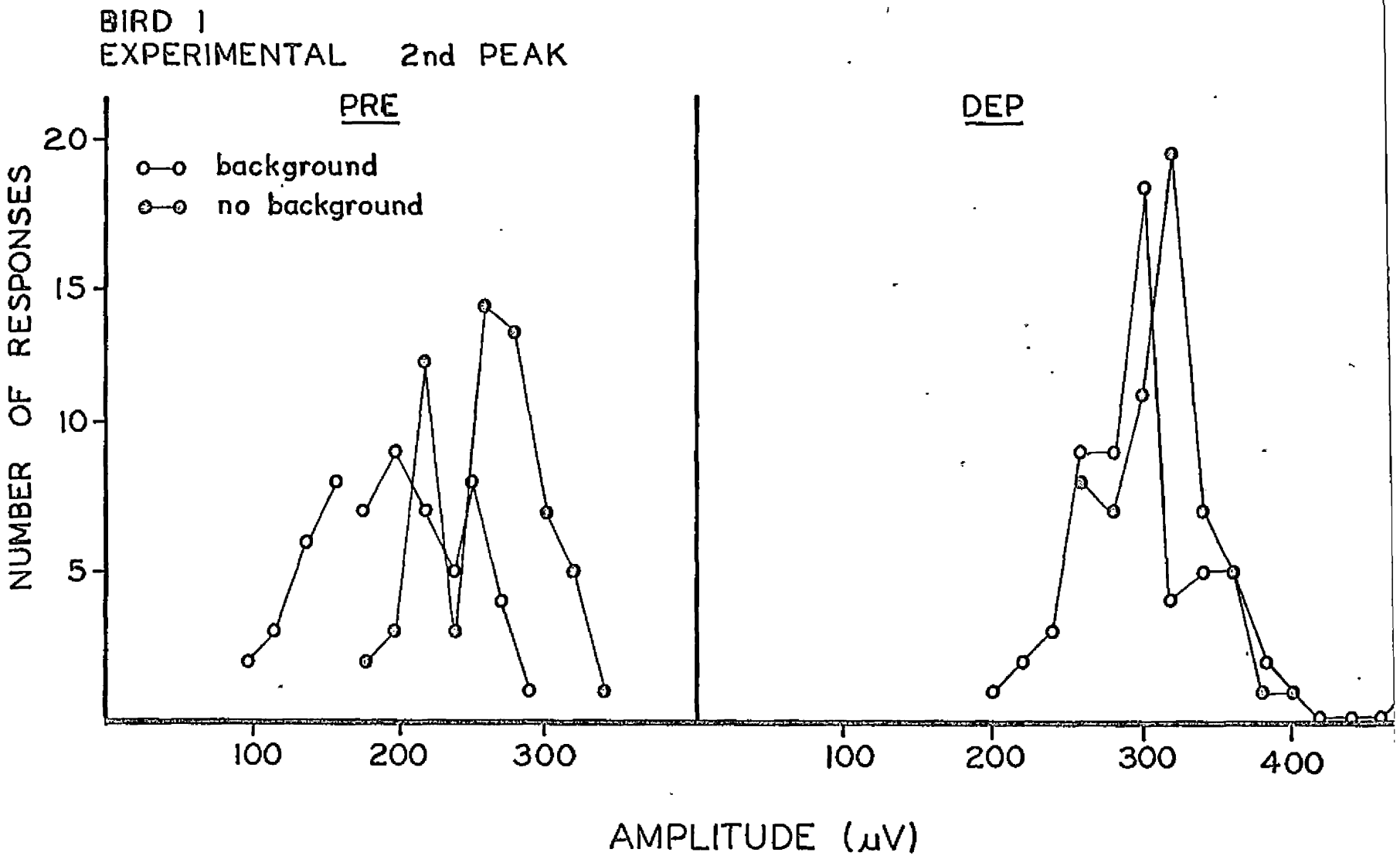
The frequency distribution for the amplitudes of the second peak.

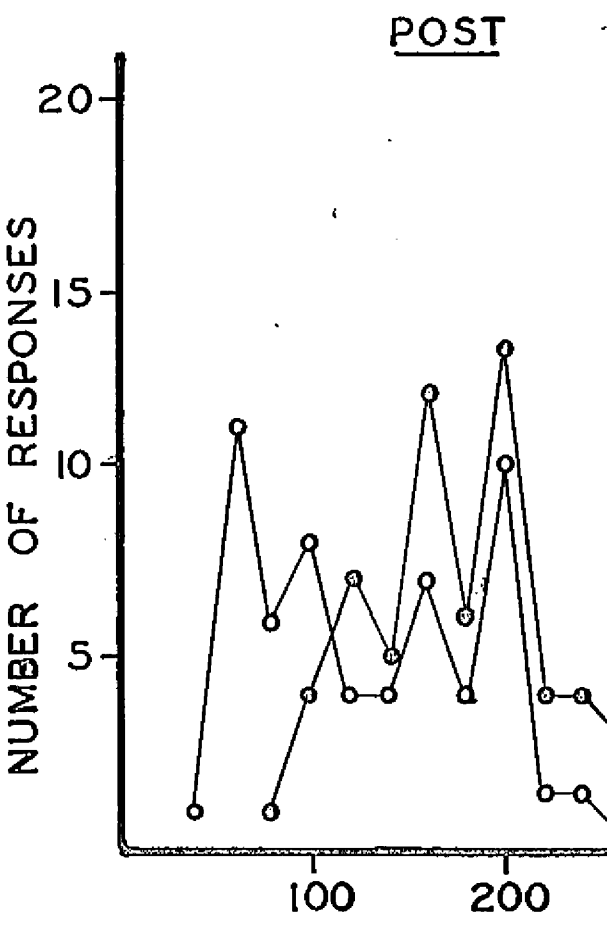

BIRD 93
EXPERIMENTAL 2nd PEAK
PRE
DEP
NUMBER OF RESPONSES
20
15
10
5
50
100
50
100
AMPLITUDE (μV)

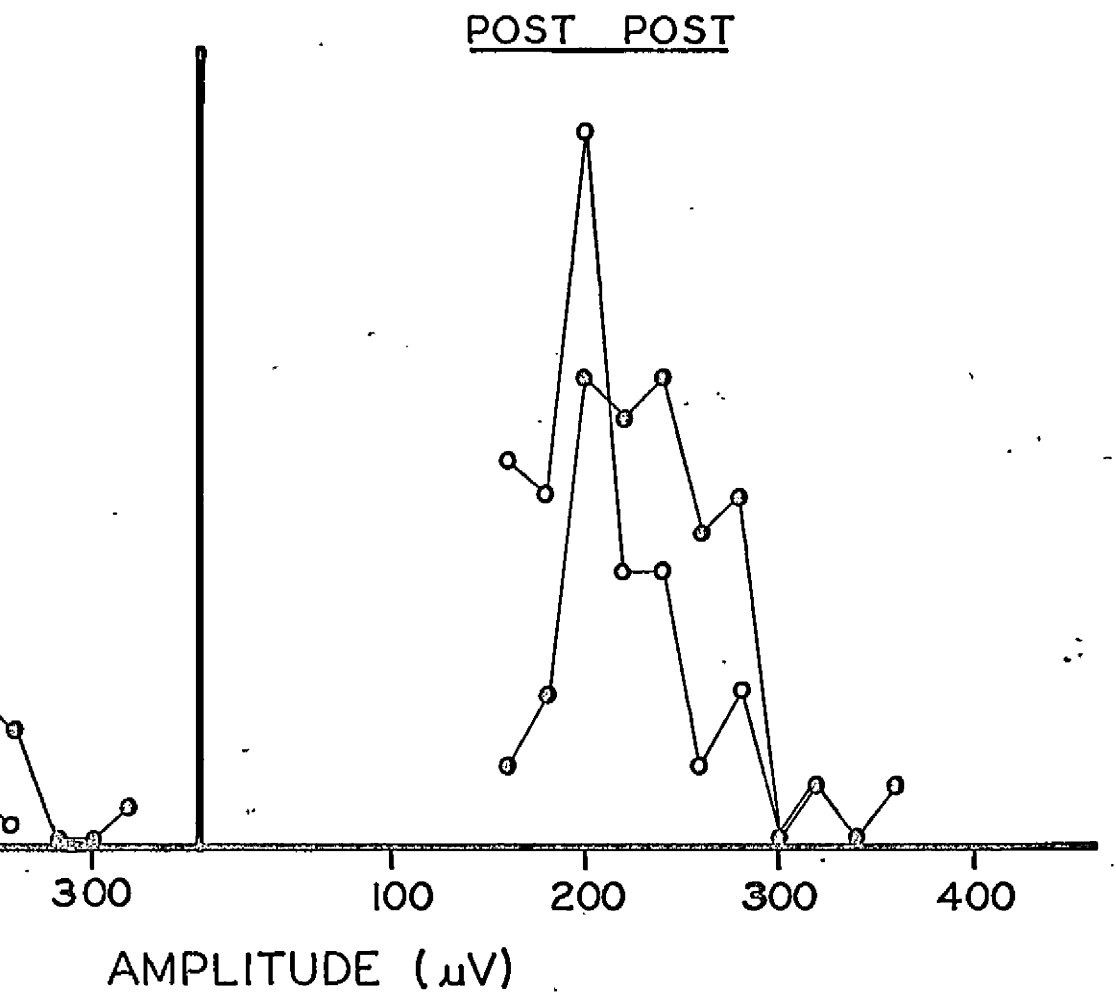

POST
POST POST
NUMBER OF RESPONSES
15
10
5
50
100
50
100
AMPLITUDE (μV)

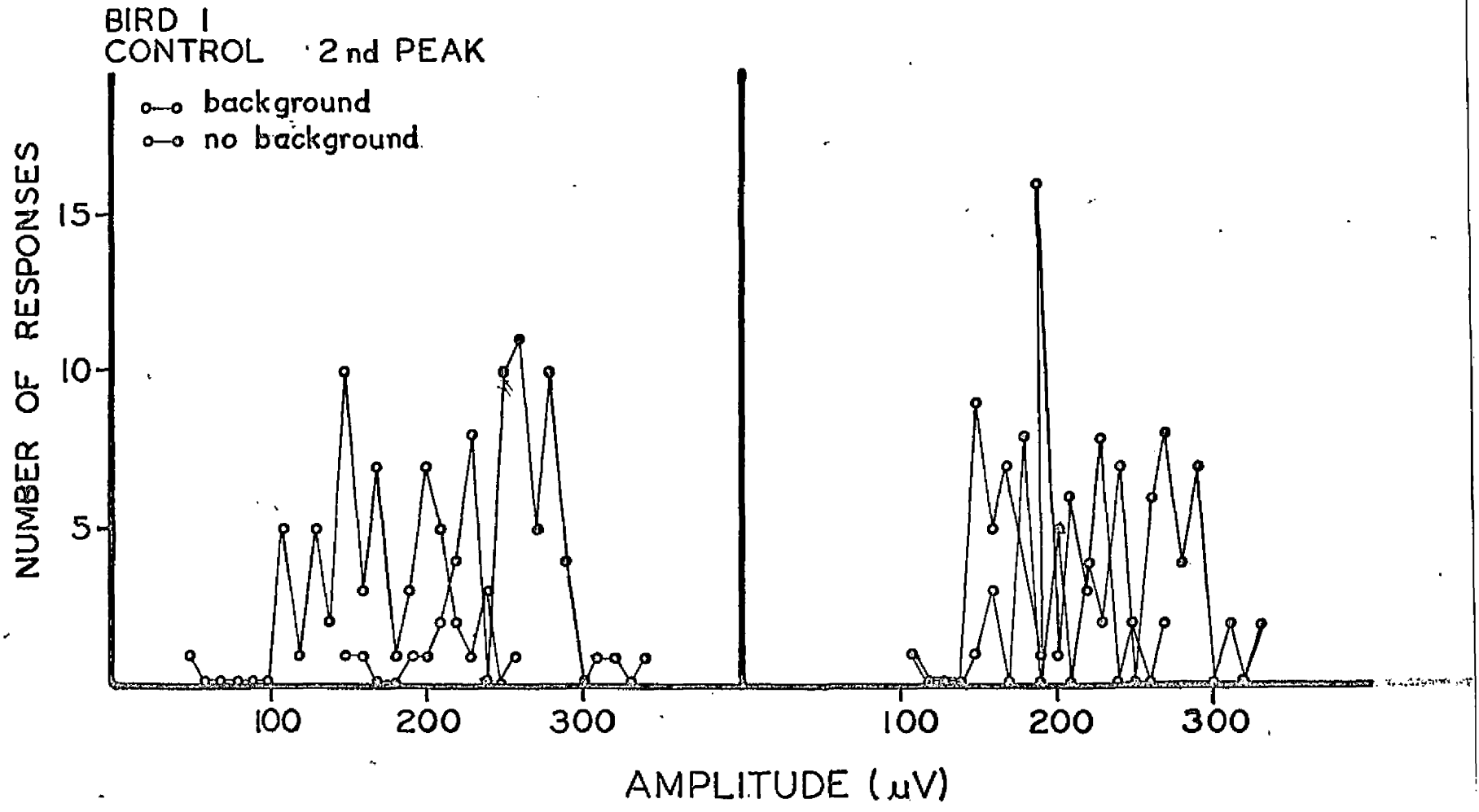

BIRD 93
CONTROL 2nd PEAK
PRE
DEP
background
no background
NUMBER OF RESPONSES
30
20
10
25
50
75
100
25
50
AMPLITUDE (μV)

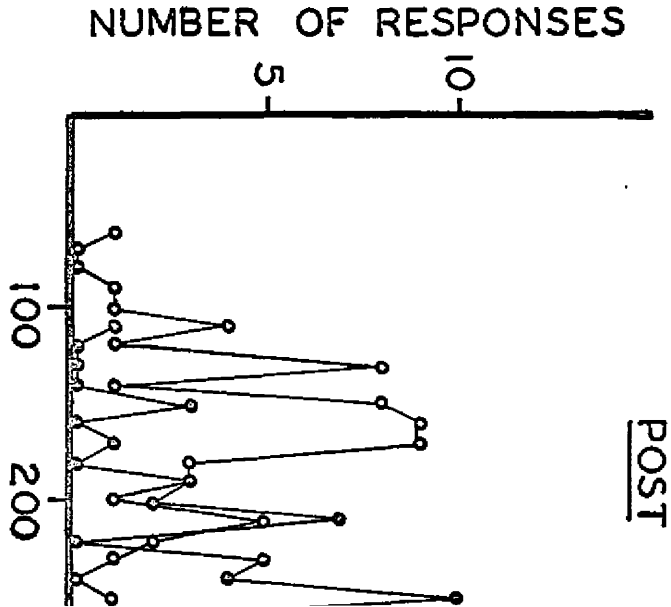

POST
POST POST
NUMBER OF RESPONSES
40
30
20
10
25
50
25
50
AMPLITUDE (μV)

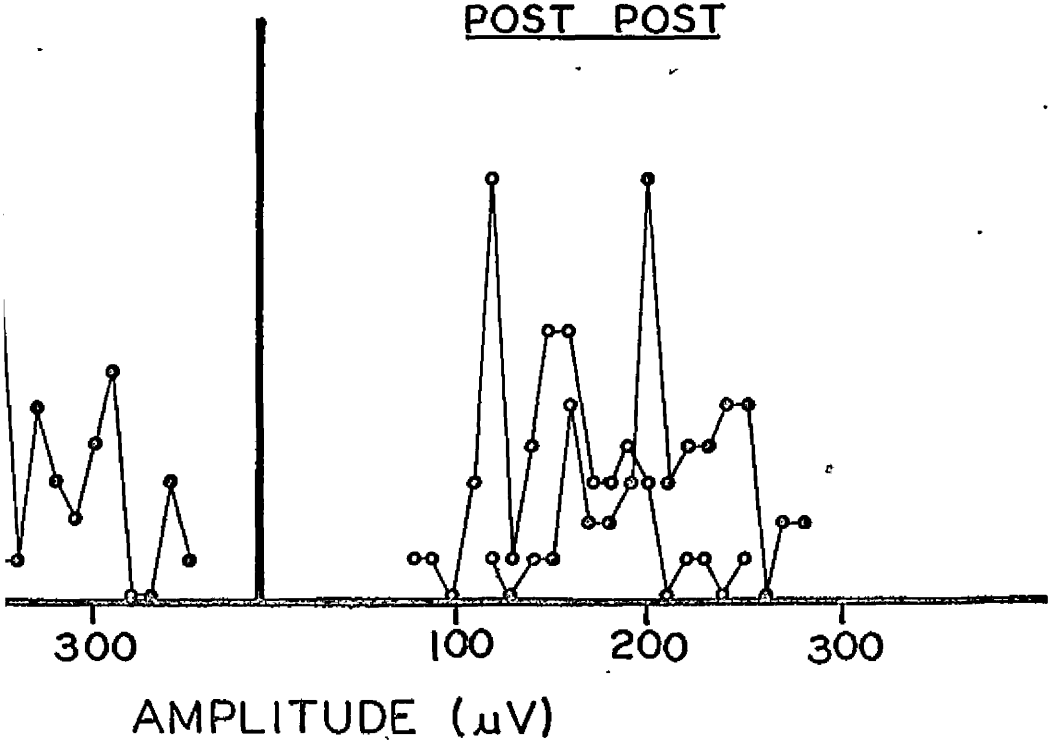

BIRD 1
EXPERIMENTAL 2nd PEAK
PRE
DEP
background
no background
NUMBER OF RESPONSES
20
15
10
5
100
200
300
100
200
300
400
AMPLITUDE (μV)

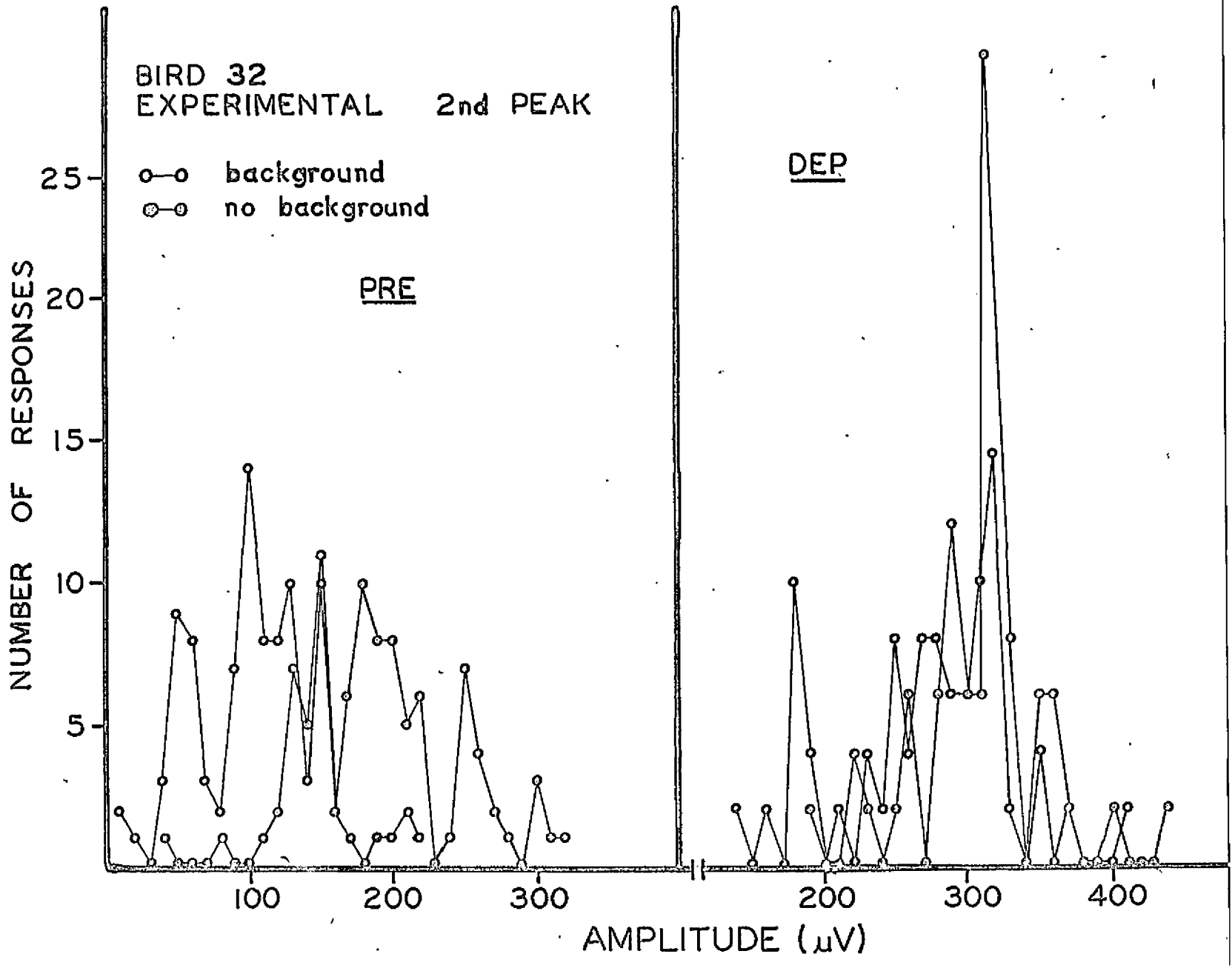

POST
NUMBER OF RESPONSES
20
15
10
5
100
200

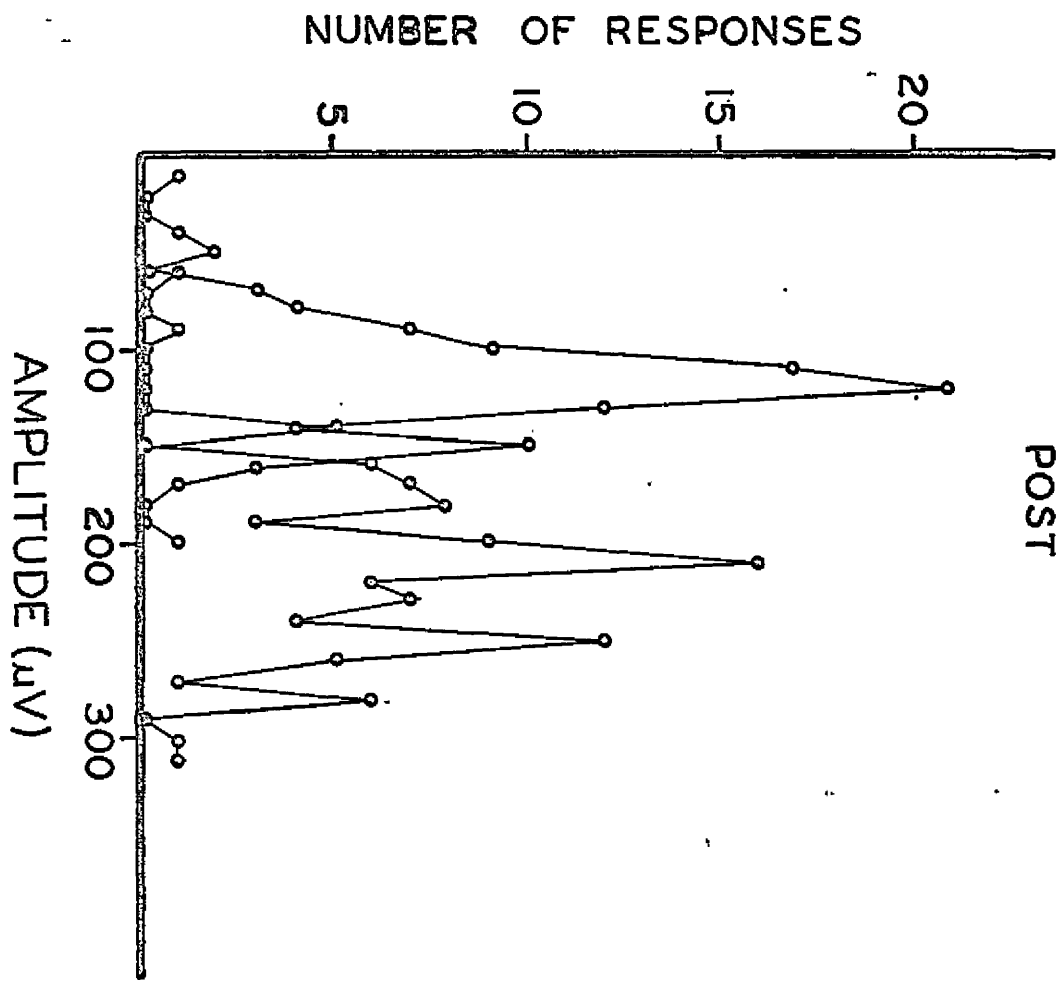

POST POST
300
100
200
300
400
AMPLITUDE (μV)

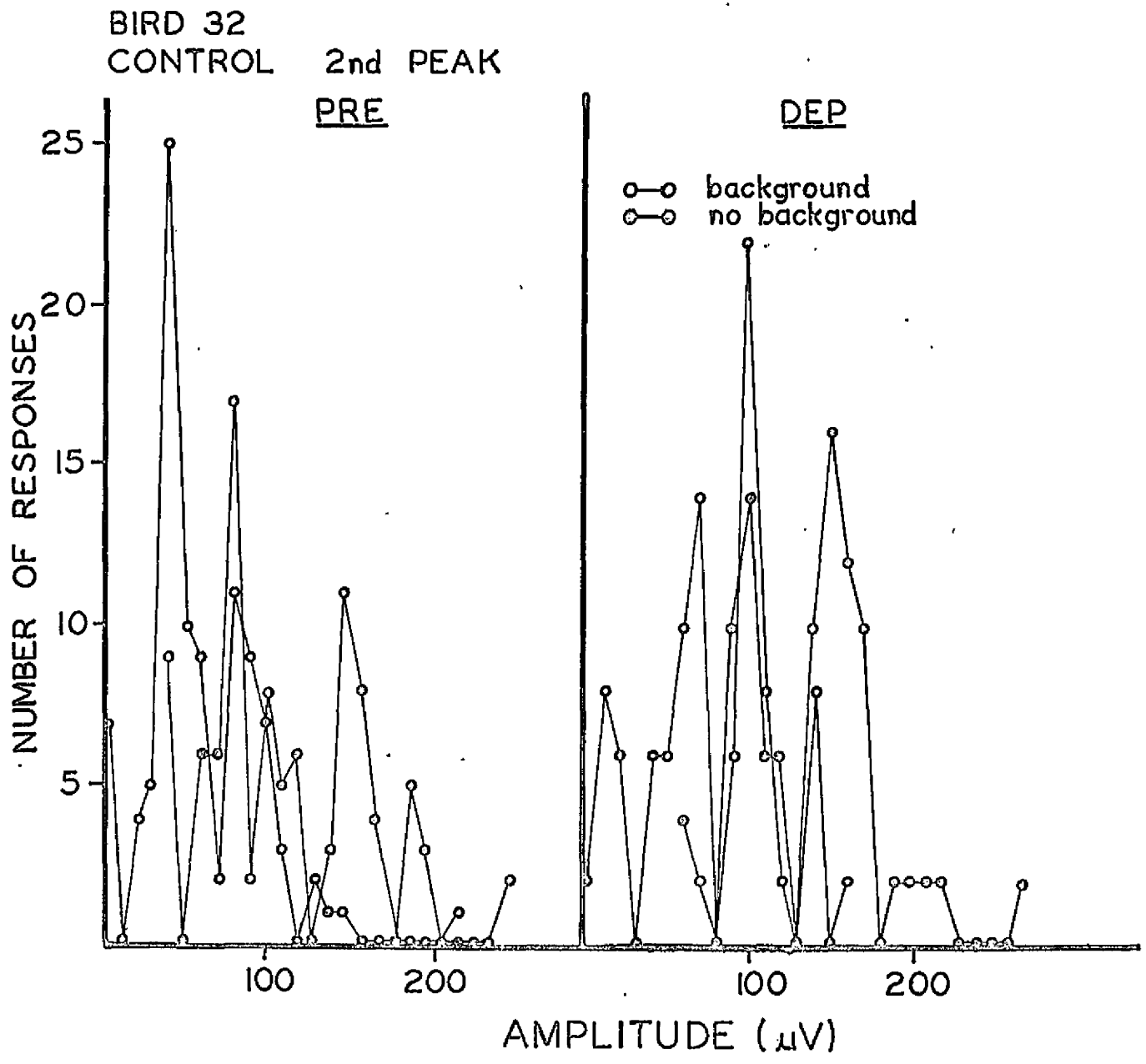

BIRD I
CONTROL 2nd PEAK
background
no background
NUMBER OF RESPONSES
15
10
5
100
200
300
100
200
300
AMPLITUDE (μV)

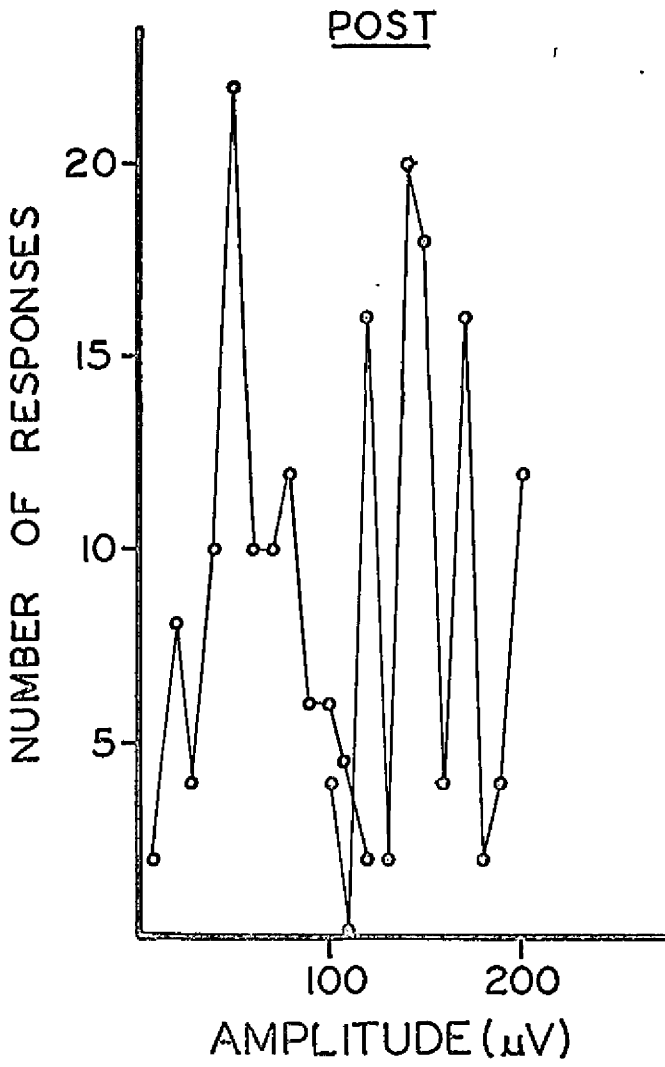

NUMBER OF RESPONSES
5
10
100
200
POST


POST POST
300
100
200
300
AMPLITUDE (μV)


BIRD 32
EXPERIMENTAL 2nd PEAK
o—o background
⊙—⊙ no background
PRE
DEP
NUMBER OF RESPONSES
25
20
15
10
5
100
200
300
200
300
400
AMPLITUDE (μV)


NUMBER OF RESPONSES
5
10
15
20
AMPLITUDE (μV)
100
200
300
POST


BIRD 32
CONTROL 2nd PEAK
PRE
DEP
background
no background
NUMBER OF RESPONSES
25
20
15
10
5
100
200
100
200
AMPLITUDE (μV)


POST
NUMBER OF RESPONSES
20
15
10
5
100
200
AMPLITUDE (μV)