MONOCULAR LIGHT DEPRIVATION OF THE PIGEON

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By

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SCOPE AND CONTENTS:

Monocular deprivation of visual input in the pigeon alters the visual evoked response of the contralateral optic lobe. When the procedure is repeated, and the previously normal eye deprived of light, the responses of both optic lobes change. The EEG did not change with deprivation. The results for animals deprived of patterned visual input are unclear in terms of both the evoked response and the EEG.

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TABLE OF CONTENTS

Introduction	Page	1
Methods		12
Results		26
Discussion		80
Summary		84
Bibliography		86

FIGURES AND TABLES

		Page
FIGURE 1	Eye Ring, Occluder, and Flash Tube Holders	15
FIGURE 2	Stimulating and Recording Equipment	18
FIGURE 3	Test Session	21
FIGURE 4	Deprivation Procedure	23
FIGURE 5	EEG in Dark and Light	28
FIGURE 6	Power Spectra of EEG	30
FIGURE 6a	Power Spectra of EEG	32
FIGURE 7	Normal Evoked Responses	35
FIGURE 8	Amplitudes of Response Components in Figure 7	38
FIGURE 9	Normal Evoked Responses	40
FIGURE 10	Control and Experimental Responses from Bird 39 in Experiment I	42
FIGURE 11	Difference Waveforms for Responses in Figure 10	49
FIGURE 12	Excursion Scores for Waveforms in Figure 11	51
FIGURE 13	Control and Experimental Responses of Bird 84 in Experiment II	53
FIGURE 14	Positive Component Amplitudes for Responses of Bird 84	56
FIGURE 15	Positive Component Amplitudes for Responses of Bird 67	58
FIGURE 16	Difference Waveforms for Responses of Bird 84	60
FIGURE 17	Excursion Scores for Difference Waveforms of Bird 84	63
FIGURE 18	Excursion Scores for Bird 67	65
FIGURE 19	Control and Experimental Evoked Responses of Bird 85	67

FIGURE 20 Positive Component Amplitudes for Responses of Bird 85 70

FIGURE 21 Difference Waveforms for Responses of Bird 85 72

FIGURE 22 Excursion Scores for Difference Waveforms of Bird 85 74

FIGURE 23 Control and Experimental EEG Recordings from Bird 84 (Light Deprivation) 77

FIGURE 24 Control and Experimental EEG Recordings from Bird 85 (Pattern Deprivation) 79

TABLE I Amplitudes of Tectal Responses for Birds 39 and 46 43

TABLE II Amplitudes of Tectal Responses for Birds 6963 and 83 44

TABLE III Amplitudes of Rotundal Responses for Birds 39 and 46 45

TABLE IV Amplitudes of Rotundal Responses for Birds 6963 and 83 46

INTRODUCTION

A large number of studies have investigated the neural and behavioral effects of depriving organisms of normal amounts of sensory input. While a great deal of this research has been concerned with the changes brought about by sensory deprivation during an animal's growth, more recently there has developed an interest in the results of deprivation of the adult nervous system in an attempt to understand to what degree sensory input contributes to the maintenance of normal neural and behavioral functioning. The role of the visual modality has been most widely investigated in this regard because of the relative simplicity with which the amount of visual input can be manipulated and the fact that for most vertebrates vision is the most important sense.

One technique used in these studies is that of unilateral visual deprivation; that is, elimination or modification of visual input to one eye while allowing the other eye normal vision. The principle advantage of this technique is that optic centers receiving input from the deprived eye can be contrasted with those in the same animal receiving input from the normal eye, thus making it possible to use each subject, in a sense, as its own control. Variables such as the arousal level and general physiological state of the animal are controlled for both deprived and undeprived structures, an otherwise difficult problem in those studies involving paired experimental and control animals.

Bondy has noted that monocular deprivation is particularly appropriate for use on the avian visual system because of two characteristics of the avian brain. First, the complete decussation of the optic tract

at the optic chiasm results in each eye sending optic fibers only to the contralateral brain hemisphere. Second, the absence of any interhemispheric commissure as prominent as the mammalian corpus callosum indicates that there is a relatively lesser degree of interhemispheric communication. As a result, monocular modification of visual input should produce differing effects in the two hemispheres, permitting each to serve as a control for the other. By contrast, if a mammal such as the cat were monocularly deprived the incomplete crossing of optic fibers at the chiasm would result in both hemispheres being partially deprived of input.

Bondy has carried out three types of unilateral modification of visual input in both the immature and mature chicken visual system -- deprivation of both patterned visual input and light, deprivation only of patterned input, and increase of light with no change in patterned input-- and has related these manipulations to the changes in the blood flow in the brain as measured by a radioactive tracer technique. Reduction of both light and patterned visual input was accomplished by suturing one eye in 2, 15, and 30 day old chicks for 15 minutes, with the result that blood flow in the optic lobe, cerebral hemisphere and thalamic regions contralateral to the closed eye significantly decreased relative to the same brain areas contralateral to the open eye. Placing an opaque film (Parafilm) over one eye prevents patterned input but not light from entering the eye. One hour of this pattern deprivation failed to result in any change in blood flow in the brains of either newly-hatched or 30 day old chicks. Topical application of a pupillary dilator (10% phenylephrine hydrochloride) in the presence of a high level of illumination increases the amount of light falling on the retina but does not change the pattern content to the eye. One hour after such an application to

one eye of a chick, there was an increase in blood flow through the contralateral optic lobe and thalamic regions (Bondy 1973; personal communication).

Bondy's findings strongly suggest that the amount of light and not the information content in the visual input an animal experiences is the more important factor in the development and maintenance of cerebral circulation. Similar differences between the effects of light versus pattern deprivation were reported by Cornwell and Sharpless (1968) in their study of the electroretinogram (ERG) of the monocularly deprived cat. These investigators unilaterally deprived adult cats of either light or patterned vision with plastic occluders for periods ranging from 36 hours to 3 weeks and then measured in both deprived and undeprived eyes the amplitude of the b-wave of the ERG evoked by single light flashes of various intensities. One week or more of monocular light deprivation reduced the amplitude of the b-wave of the deprived eye's ERG for all of the flash intensities used while seven days of pattern deprivation did not affect the ERG. When the light deprived animals were exposed to a three or four flash per second stimulus for 30 seconds, it was observed that after some initial variation during the first few flashes, the amplitude of the b-wave of the nonoccluded eye reached a stable value and then remained constant. On the other hand, the response of the light deprived eye to the flicker progressively declined and often disappeared completely after 15 to 30 seconds of stimulation. When a low level of background illumination was superimposed on the flash the responses of both eyes were reduced, that of the light deprived eye to a much greater extent that that of the control eye.

The pattern deprived cats were not tested with either the repetitive stimulation or the background illumination.

The alterations in adaptation to the flicker and constant illumination suggested to Cornwell and Sharpless that a retinal mechanism for light adaptation is changed by light deprivation. According to Dowling (1967), the bipolar-amacrine cell system may comprise the substrate for an inhibitory loop involved in adaptation, in which the sensitivity of the bipolar cell is reduced in proportion to the amount of excitation in it, and as a result of the synaptic contacts between bipolar and amacrine cells, inhibition is transmitted to adjacent bipolars. Cornwell and Sharpless hypothesize that deprivation reduces the excitation in the bipolar-amacrine system thus increasing its sensitivity, so that when input is restored the amount of inhibition produced is greater than normal, resulting in smaller flash-evoked response amplitudes.

The research reviewed thus far indicates that monocular deprivation of light can result in measurable changes in the functioning of the mature visual system. The positive effects obtained by Bondy and Cornwell and Sharpless stand in contrast to the negative findings of Hubel & Wiesel in two studies on the monocularly deprived adult cat. Wiesel and Hubel (1963) sutured the lid of the right eye in an adult cat, sacrificing the animal after 3 months of eye closure. They found that the cells in the layers of the lateral geniculate nucleus receiving input from the closed eye were no different in appearance and staining properties from cells in geniculate layers receiving input from the open eye. Hubel and Wiesel (1970) found no changes in single unit firing after 16 months of unilateral eye closure. After opening the deprived eye, they presented light flashes to each eye

separately and observed that the number of cells in the striate cortex driven by each eye did not differ, leading to the conclusion that "An adult cat seems completely resistant to monocular deprivation (1970, p. 427)".

Two methodological differences between the study of Cornwell and Sharpless and those of Hubel and Wiesel should be emphasized when comparing the former authors' positive results with the negative data of the latter workers. Cornwell and Sharpless examined an essentially retinal phenomenon while Hubel and Wiesel were concerned with anatomical and physiological changes in the geniculate and cortex, and it may be that deprivation has a greater effect on the peripheral as opposed to the more central levels of the visual system. Second, and more important, the deprivation technique used by Cornwell and Sharpless--the suturing of a black plastic disk over the eye--was more severe in that it eliminated more visual input than the lid suturing technique of Hubel and Wiesel, in which there is a possibility of some amount of light penetrating the closed eyelid and entering the eye. This residual input may have been enough to maintain a certain level of activity in the nervous system sufficient to prevent detectable deterioration.

Zubek and Bross report an unusual interocular effect on critical flicker frequency (CFF) in several studies on monocularly deprived adult humans. In the first paper in this series Zubek and Bross (1972) measured the CFF of only the nondominant eye in 30 male university students, after which half of the subjects had the dominant eye covered by a black eye patch, while the others were allowed normal vision. In this and all subsequent studies, subjects were then confined in groups of three or four in a large

furnished room containing a radio, a television set and reading material. The CFF of the nondominant eye in each subject was determined again at 3, 6, 9, 15, and 24 hours after the first determination. In subjects with one eye occluded, the CFF of the nonoccluded, nondominant eye decreased significantly after six hours of deprivation, returned to its approximate predeprivation level after 9 hours, and was greater than baseline at 24 hours. On the other hand, the CFF of the corresponding eyes in subjects allowed normal vision did not change during the 24 hour period. A second experiment which duplicated the conditions of the first except that measurements were taken only from the covered eye of monocularly deprived subjects failed to show any CFF shifts. Zubek and Bross concluded that the initial depression and later enhancement of the CFF is specific to the uncovered eye in monocularly deprived subjects.

A second paper (Bross & Zubek, 1972) investigated extending the period of monocular deprivation to one week. The CFF of the nonoccluded, nondominant eye in 16 monocularly deprived experimental subjects was measured after 0, 1/3, 1, 2, 3, 5, and 7 days of deprivation, and compared to the CFF of the nondominant eye of 16 control subjects allowed normal vision. The nonoccluded eyes of the experimental subjects all exhibited a negatively accelerated increase in the CFF with gains ranging from 0.62 to 4.14 flashes per second and a mean rise of 2.34 over the seven days of occlusion. The CFF of the undeprived subjects did not change at any of the intervals at which it was tested. Control experiments showed that the CFF of the occluded eye did not change, and that it did not matter whether the dominant or nondominant eye of the experimental subjects was occluded.

Next, Zubek and Bross (1973a) measured the CFF before and during 14 days of monocular deprivation, and during 14 days after the restoration of normal vision. The nonoccluded eye of the experimental subjects showed a negatively accelerated increase in CFF, reaching a plateau between 7 and 9 days of deprivation, then registering an additional rise at 11 days and no further change at 14 days. With the removal of the eyepatch, the CFF showed a gradual decline toward the predeprivation baseline through the 14 day postdeprivation period, and it is interesting that although significant after-effects were present only until postdeprivation day 3, the mean CFF on postdeprivation day 14 was still greater than the mean predeprivation CFF. As usual, there were no changes in the eyes of undeprived control subjects or in the occluded eyes of experimentals.

Since the eyepatch technique used in the above studies deprives an eye of patterned visual input while depriving it of light, these authors felt it necessary to establish whether deprivation of pattern vision but not light could affect the CFF. Accordingly, Zubek and Bross (1973b) placed a white translucent occluder over one eye in 14 experimental subjects for 72 hours and tested the CFF of only the uncovered eye at 0, 3, 6, 9, 15, 24, 28, and 72 hours of deprivation. No CFF change was apparent at any time Subsequent control studies also failed to reveal any change in the occluded eye of experimentals or in control subjects allowed normal vision. These negative findings indicate that deprivation of light and not of pattern vision is the variable that produced the CFF changes in the previous studies.

Bross and Zubek (1972) interpret their CFF enhancement results in terms of a disuse supersensitivity model. As set forth by authors such as Stavraky (1961) and extended by Sharpless (1964, 1969) the concept of disuse

supersensitivity refers to the increased excitability of nervous tissue following an extreme and prolonged reduction in the input to that tissue produced by administration of drugs, sensory deprivation or sectioning of neural pathways. The first paper to demonstrate hyperexcitability in the sensory cortex subsequent to its partial isolation by means of subcortical lesion of its afferent pathways was the work of Speigel and Szekely (1955). They implanted electrodes epidermally above the sigmoid gyri and other areas of the cerebral cortex of the cat. While monitoring the spontaneous activity of these cortical areas, they placed an electolytic lesion in one ventral posterior thalamic nucleus, whose efferents terminate in the ipsilateral posterior sigmoid gyrus, and noted that the spontaneous activity of that gyrus immediately decreased. After at least three days however, the spontaneous activity returned, and often its amplitude was greater than it had been preoperatively. When bilateral electric shocks were administered to the sciatic or ulnar nerves the evoked potentials of the posterior sigmoid gyrus ipsilateral to the lesioned thalamic nuclei were greater than the potentials in the gyrus contralateral to the subcortical lesion.

Bross and Zubek (1972) hypothesize that a similar change takes place in the primary sensory system in response to monocular deprivation. Specifically,

> "....it is possible that our occlusion procedure may be producing a state of temporary partial deafferentation of the visual system resulting in an enhancement of the CFF. However, this deafferentation is of a functional rather than of a surgical or drug induced nature, i.e, it is produced by visual deprivation in the normal biologically intact organism (1972, p. 51)."

Comparing their findings to those of Speigel and Szekely (1955), Bross and Zubek note that both studies showed that increased excitability of the nervous system occurred in response to decreased input and that this increase was preceded by a period of depressed activity. Viewing the phenomenon in these terms permits one to include the CFF depression-enhancement data, the increased tactile sensitivity found in studies of partial occlusion of the skin (Aftanas & Zubek, 1964), and other hyperexcitability findings all within the same category of supersensitivity phenomena resulting from disuse of neural pathways.

Although the experiments of Bross and Zubek are carefully controlled and executed, there appears to be a major difficulty associated with the inclusion of the CFF enhancement findings under the heading of disuse supersensitivity. In the Speigel and Szekely and the Aftanas and Zubek studies the structure deprived of input showed the increase in sensitivity to stimulation, in the former study it was the sensory cortex ipsilateral to the thalamic lesion, in the latter, it was the area of skin on the arm occluded with a plastic cover and the homologous, nonoccluded area on the opposite arm that showed the supersensitivity effects. But in the Bross and Zubek studies it was the eye that was not deprived of input that showed the increased sensitivity, while the eye that was deprived of input failed to exhibit any change in CFF. Had the occluded eye alone, or both the occluded and non-occluded eyes displayed a CFF increase there would perhaps exist greater justification for explanation of the results in terms of disuse supersensitivity.

Three basic conclusions emerge from a consideration of the experiments reviewed in this Introduction. First, it appears from all of the

studies except those of Hubel and Wiesel that monocular deprivation of visual input results in marked changes in the functioning of the adult visual system, though the type of effects differ with the species studied and the methodology employed. Second, the total amount of light entering the eye and not its informational content is the more important variable in determining the degree to which the nervous system is disrupted. Third, although authors such as Sharpless and Zubek have postulated underlying mechanisms like increased activity of inhibitory circuits or greater neural excitability as being responsible for their deprivation results, there is no clear evidence pointing to precisely what changes occur in the nervous system during monocular deprivation to produce the effects observed.

Although the studies reviewed indicate clearly that monocular visual deprivation of the adult animal produces changes in the visual system it is nevertheless striking that the central effects of deprivation have not been assessed. That is, except for the small amount of data reported by Hubel and Wiesel the experiments discussed above examined cerebral circulation, evoked retinal activity, or judgements of visual fusion, and not the characteristics of the brain itself in evaluating the effects of deprivation. As a result it is unclear what the effects of visual deprivation of the adult brain are though the work of Bondy and Bross and Zubek suggests that they should exist. Accordingly, the present study measured two types of brain activity, the visual evoked potential and the electroencephalogram (EEG) of the pigeon, in an attempt to uncover central effects of visual deprivation.

Two additional matters were also investigated. One was the description of the computer-calculated distribution of frequencies present

in the normal EEG of the pigeon. The second was the examination of the relationship between the rate of visual stimulation and the amplitude of the pigeon's visual evoked response. When the present research was conceived the goal was to investigate the effects of both pattern and light deprivation. The data which were gathered for the former, however, were too variable for technical reasons to rule out the possibility that pattern deprivation has no effect. They suggested that it did not have a strong effect. As there were limitations of time, it was decided to begin a second study and examine light deprivation.

METHODS

Subjects and Surgical Procedures

Seven (500-750 gm.) adult White Carneaux pigeons were used as subjects. They were fed ad lib and housed in wire mesh cages kept in a room whose fluorescent lights were on an automatic 12 hour light/dark cycle.

Size 00 insect pins were connected with wire to male Amphenol pins and coated in Epoxylite. About 0.5 mm. of insulation was scraped from the pin's tip and the assembly was tested for insulation breaks by applying a weak current while it was in a saline solution.

After 24 hours of food deprivation, the pigeon was anesthetized by injecting 30mg./kg. of Nembutal into the brachial vein and was placed in a Kopf stereotaxic instrument equipped with a Revzin head holder (Karten & Hodos, 1967, pp. 5-6). The feathers on the scalp were clipped away and a sagittal incision made. The periosteum was scraped from the bone, holes were drilled in the skull and jewelers' screws inserted to act as anchors and electrodes. One placed in the frontal bone served as a reference electrode and one over each orbit detected eyeblinks.

The depth electrodes were held in Kopf manipulators and implanted in the nuclei rotundi (AP. +6.25, D. 6.00, L.$\pm$3.00) and optic tecta (AP. +5.00, D. 5.00, L. $\pm$5.50; Karten & Hodos, 1967) while the responses to light flashes were monitored on an oscilloscope. The electrode was secured at the depth which showed the largest response and the difference between this position and the depth coordinates given above was never more

than 0.5 mm. Each electrode was secured with dental cement and after all four were implanted a 9/64" vanadium bone screw, which served to fix the head during data recording, was inserted a short depth into the frontal bone and cemented in place. The Amphenol pins wired to the electrodes were inserted into Amphenol connectors which were embedded in a cap of dental cement.

Plastic eye rings (Fig. 1A) were then placed around the pigeon's eyes and cemented to the cap. After surgery the bird was returned to its home cage and given at least two weeks to recover. Infections were controlled by penicillin injections and by application of Neo-Sporin to affected eyes or ears.

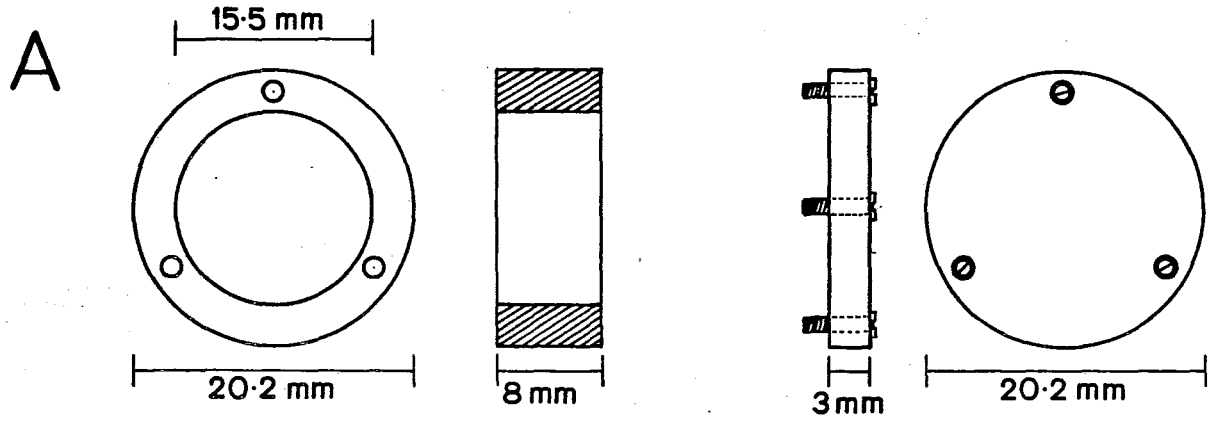
Two types of visual occluders were used, the first deprived an eye of patterned visual input while allowing diffuse light to enter the eye, the second deprived the bird of all visual input to one eye. The former consisted of a 3 mm. thick disk of Perspex made translucent by sandpapering both sides and was fastened to the eye ring by three small screws (Fig. 1A). The latter type was a hood of opaque black cloth with holes for the beak and one eye.

Stimulation and Recording Apparatus

To diminish the intensity of the light flash in Experiment I and so ensure the occurrence of a submaximal response, five layers of No. 7 filter paper were placed over the glass shield of the flash tube holder of a Grass PS 2 photostimulator. The flash unit was coupled to the eye ring on the bird by fastening the wide end of a large funnel to the flash unit and inserting its narrow end into a plastic cylinder which was then firmly fitted to the eye ring (Fig. 1B).

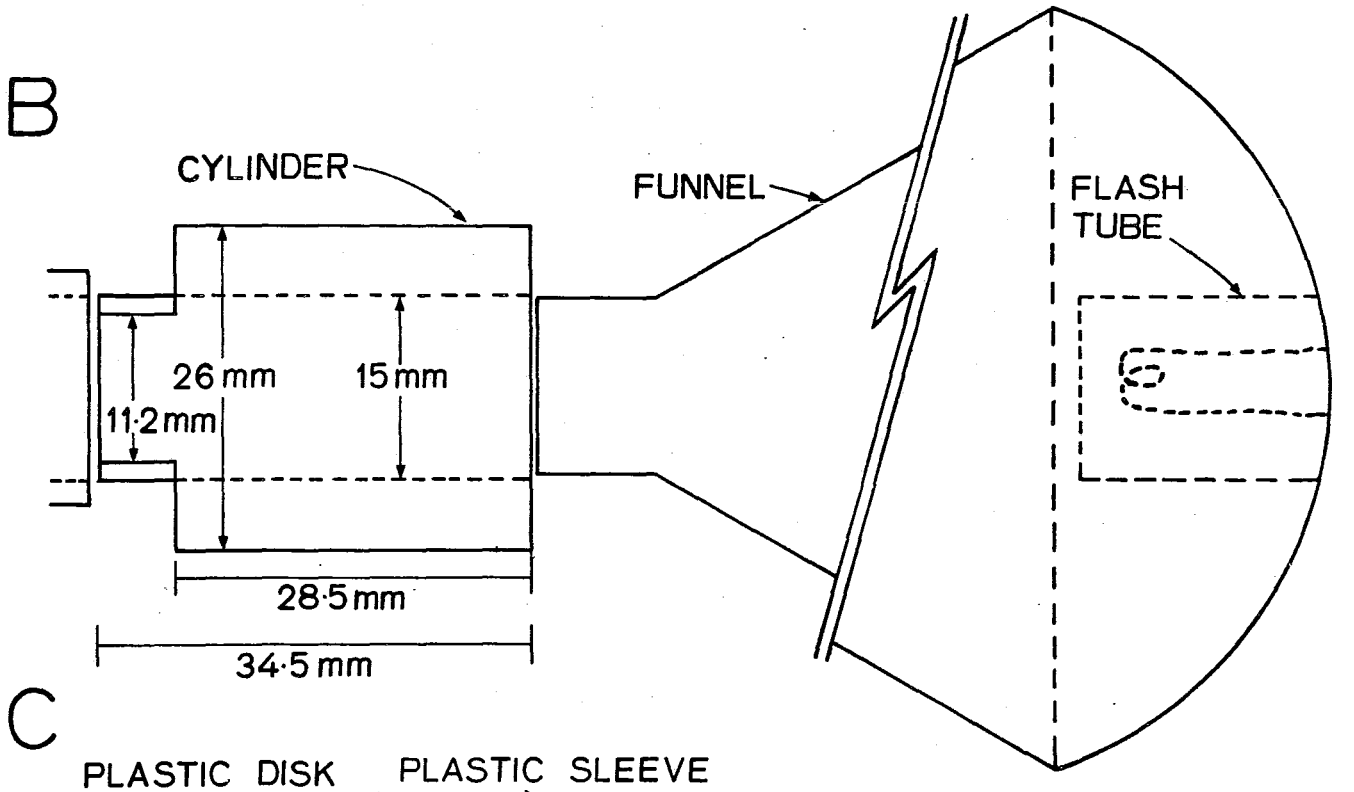
FIGURE 1.

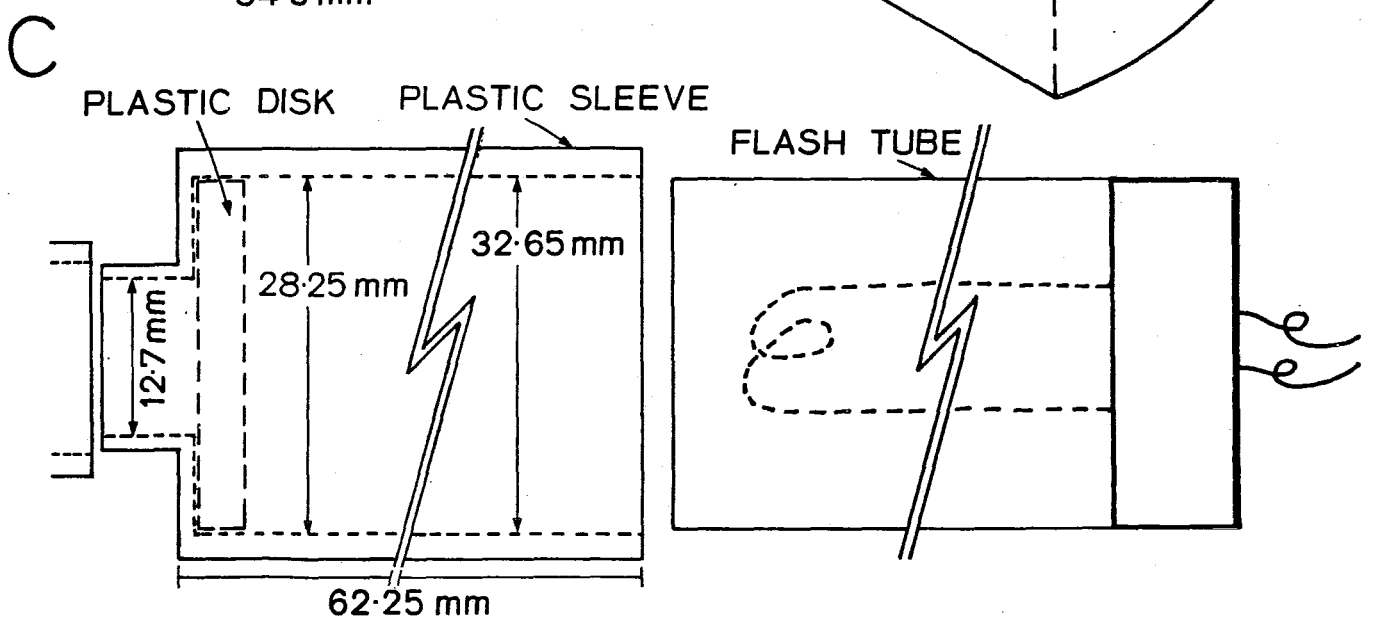
A. Diagrams of eye ring and occluder used to eliminate patterned visual input. B. Apparatus for fastening tube holder to eye ring in Experiment I. C. Same apparatus for Experiment II.



EYE RING.

OCCLUDER FOR PATTERN DEPRIVATION.





The data for Experiment I showed some variability and it was felt that this was due to instability of the apparatus. Accordingly, a different stimulating system was used in Experiment II. The flash tube and transformer were removed from the tube holder and the transformer housed in a small, grounded metal box with wires leading to the flash tube, which was inserted in a plastic sleeve with the open end blocked by a disk of light-absorbing plastic. The sleeve had a lip protuding from one end which mated with eye ring and the sleeve-flash tube assembly was held by this connection (Fig. 1C).

Because it is very difficult to measure the absolute intensity of the light flash, a relative measure of flash intensity was obtained. The lens of a photometer (Photo Research, Hollywood) was placed at the same distance from the stimulus source as was the pigeon's eye during testing. The photostimulator was triggered 40 times per second and the resulting reading on the photometer noted. The light flash used in Experiment I, where the intensity setting of the PS 2 was at 4, yielded a reading of 20 foot lamberts. For Experiment II, where the intensity setting was at 1, the reading was 26 foot lamberts.

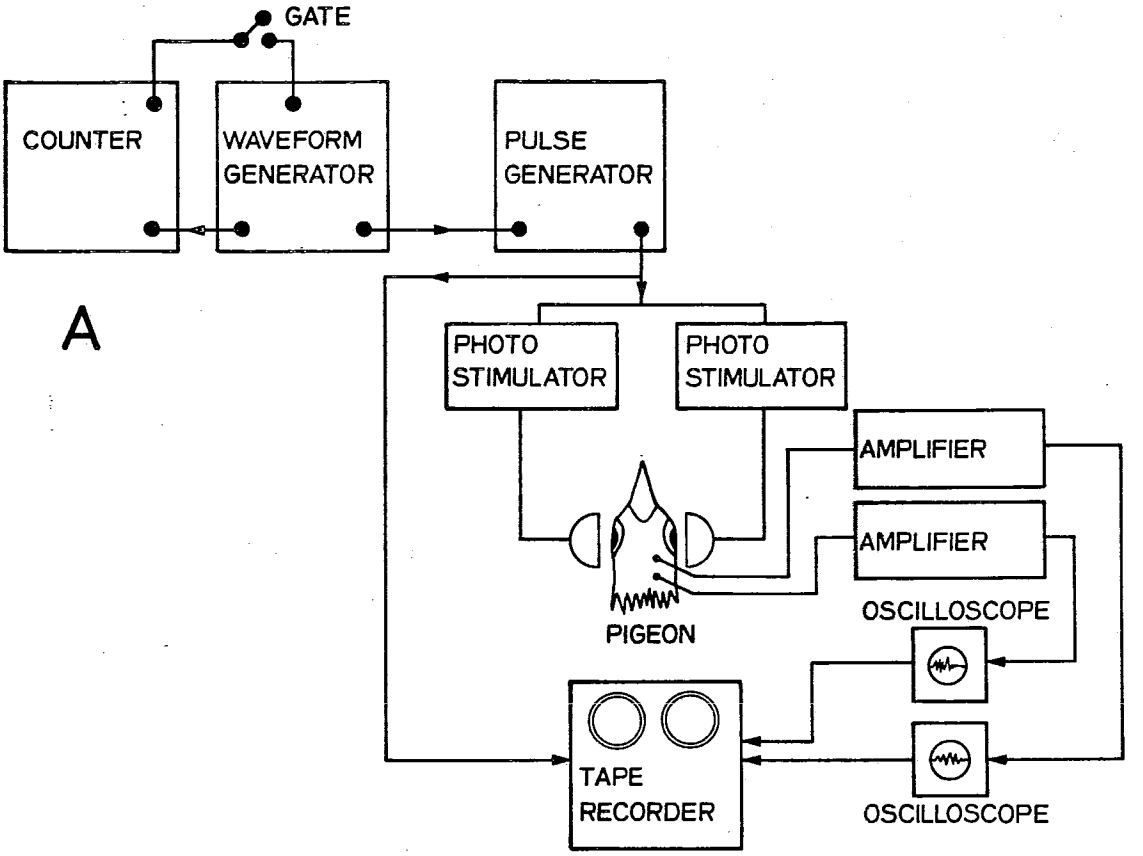
When the room lights were off the only illumination was provided by a shaded 7 watt bulb about 2 m. behind the subject. Placing the photometer this distance from the bulb and orienting its lens at right angles to it produced a reading of 2.5×10^{-3} foot lamberts on the meter.

The apparatus in Experiment I (Fig. 2A) was designed to deliver a preset number of flashes. The EEG was amplified by two Grass dual P9 amplifiers with filters set at 0.5 and 2000 Hz, then led into two Tektronix 502A oscilloscopes and from those to an Ampex SP-300 tape recorder which also recorded trigger pulses from the pulse generator.

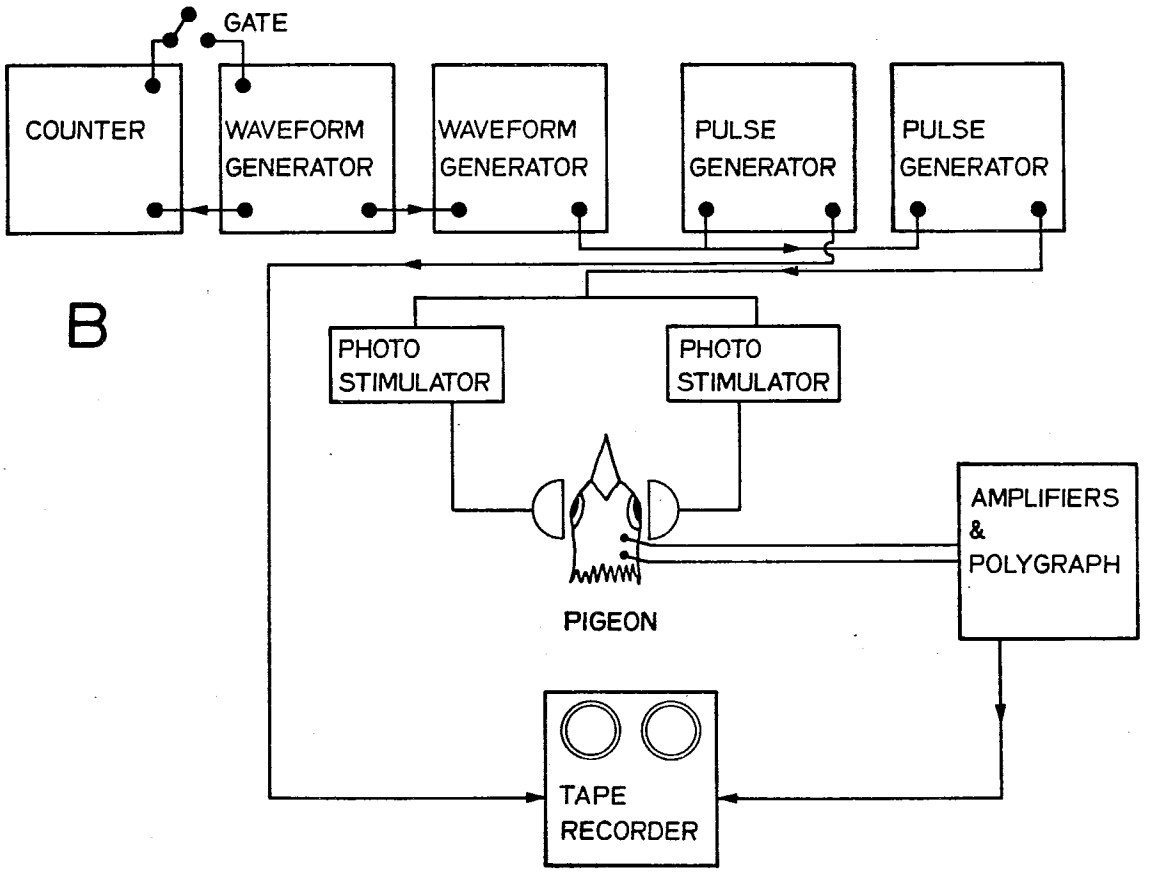
FIGURE 2.

Stimulating and recording equipment. Each delivered a preset number of light flashes before stopping. A. Apparatus used in Experiment I. B. Apparatus used in Experiment II produced a pulse 20.5 msec. prior to the light flash.

EXPT. I



EXPT. II



The apparatus in Experiment II also delivered a preset number of flashes in addition to producing a pulse 20.5 msec. prior to each flash. The amplifiers and oscilloscopes were replaced with a Grass Model IV polygraph modified to allow the amplified signals to be led into the tape recorder. When EEG was recorded and the polygraph's inkwriting unit was on the filters were set at 1 and 70 Hz, when evoked potentials were monitored and the inkwriter was off all frequencies between 1 and 300 Hz. were passed.

Testing Procedure

The pigeon was restrained by binding its legs and wrapping its body in paper or cloth, then placing it in a plastic container from which the head protruded. The head was clamped by means of the large bone screw and one PS 2 flash unit was attached to each eye ring. (Fig. 2A and B). The room lights were extinguished and the bird given 15 minutes to adapt to darkness and restraint (Fig. 3). (After this period in Experiment II the cloth covering the pigeon was removed prior to room illumination.)

In each study the pigeon was monocularly deprived of visual input twice (Fig. 4). After one week of monocular deprivation, the animal was allowed one week of unobstructed vision, then the previously unoccluded eye was covered and the previously occluded eye allowed normal vision. Following this second week of deprivation, a second week of unobstructed vision ensued. Data were recorded several times prior to the first occlusion to provide baseline data, then twice during each succeding week of deprivation and recovery.

Data Analysis

Evoked potentials were averaged by a DEC PDP 8/e computer using

FIGURE 3.

Diagram of testing procedure. The stimulation rates and number of stimuli presented during the first half of Experiment I differed from those in the second half.

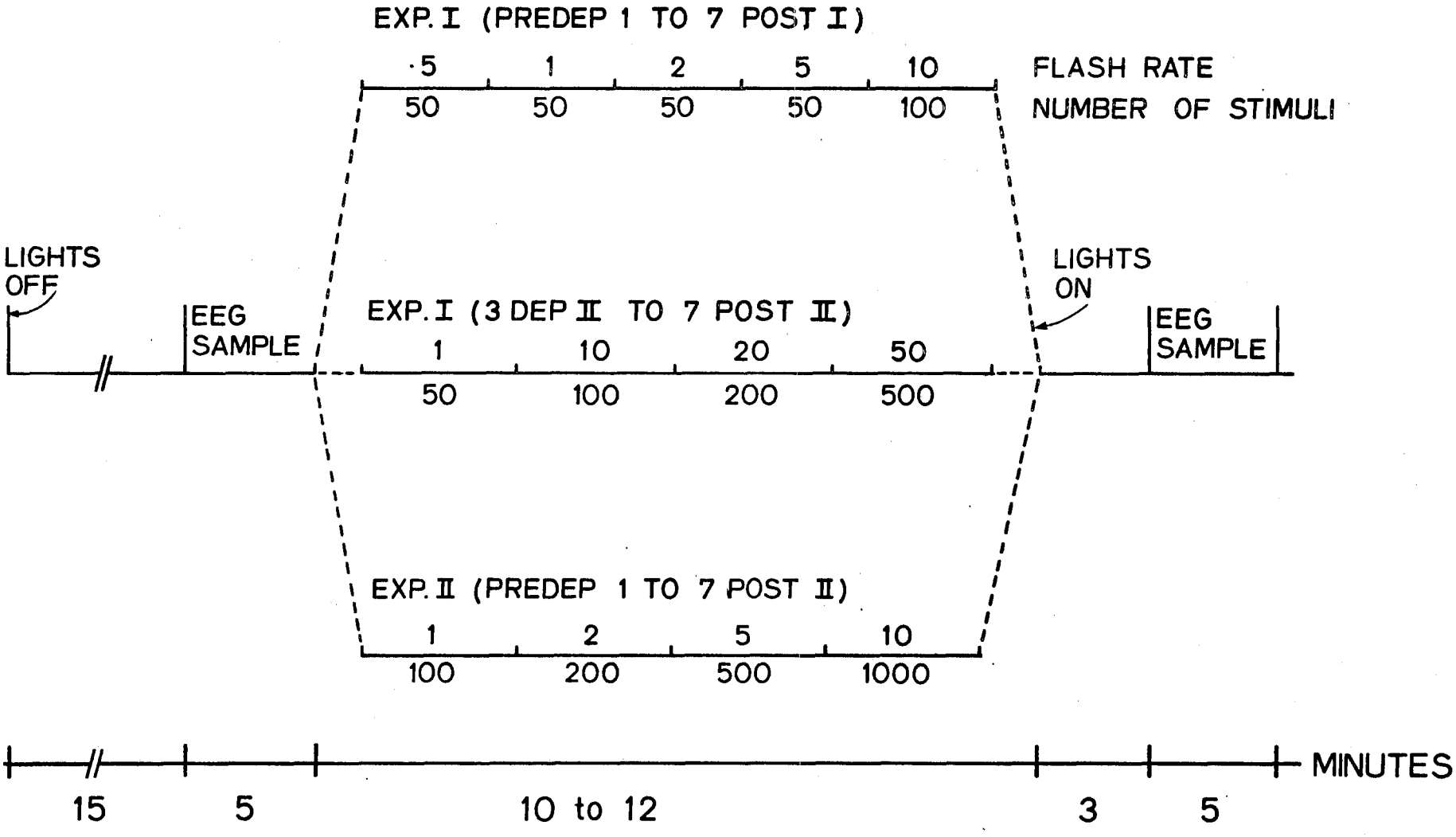
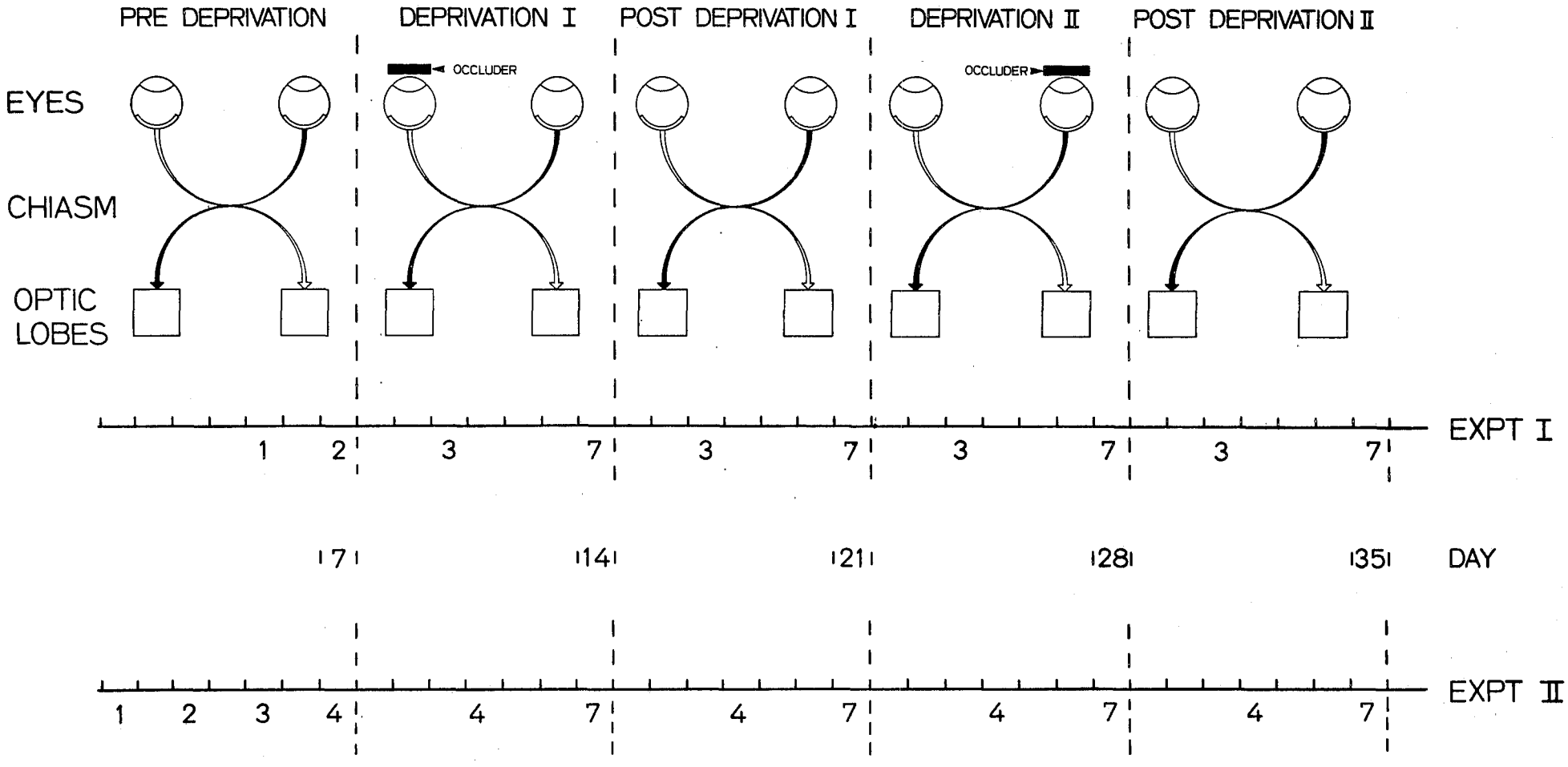

EXP. I (PREDEP 1 TO 7 POST I)
.5
1
2
5
10
50
50
50
50
100
FLASH RATE
NUMBER OF STIMULI
LIGHTS OFF
EEG SAMPLE
EXP. I (3 DEP II TO 7 POST II)
1
10
20
50
50
100
200
500
LIGHTS ON
EEG SAMPLE
EXP. II (PREDEP 1 TO 7 POST II)
1
2
5
10
100
200
500
1000
MINUTES
15
5
10 to 12
3
5

FIGURE 4.

Deprivation procedure in Experiments I and II. Dates on which the birds were tested are numbered on each scale. The left eye was not necessarily deprived first. Four animals were used in Experiment I, three in Experiment II.


PRE DEPRIVATION
DEPRIVATION I
POST DEPRIVATION I
DEPRIVATION II
POST DEPRIVATION II
OCCLUDER
OCCLUDER
EYES
CHIASM
OPTIC LOBES
1 2 3 7 3 7 3 7 3 7
EXPT I
7 14 21 28 35
DAY
1 2 3 4 4 7 4 7 4 7 4 7
EXPT II

the Daquan signal averaging program supplied by the manufacturer. For Experiment II the pulse that preceded each light flash by 20.5 msec. triggered the computer, so that the averaged evoked response included a brief period of brain activity that occurred before the stimulus. All averaged responses were punched on paper tape and analyzed with programs written in the FOCAL and BASIC programming languages which read the tapes then calculated the mean of the first several points in the response. The remaining points were searched for the positive and negative peaks in the response and the amplitude of each calculated by finding the difference between it and the mean. The latency of each peak was also found.

The evoked potentials were also analyzed using a new method ("Excursion Score") which expressed in a single number the difference between two responses. Responses obtained on a test day were subtracted from the comparable responses on day 7 POST I; for example, the response of the left optic tectum to a flash rate of 10/sec. on day PREDEP 1 was subtracted point-by-corresponding point from the response of the same structure to the same flash rate on 7 POST I. The set of difference points between pairs of responses was output on paper tape. A BASIC program read the tape and yielded a number which expressed the difference between two responses by measuring the degree of excursion in the set of difference points. This was done by calculating the mean of the difference points in the period preceding the stimulus artifact, then subtracting this baseline mean from succeeding points in the set, summing the resulting absolute values and computing the mean of this sum. The more dissimilar two responses, the more excursion in the difference points and the larger the resulting score.

EEG power spectra were computed by inputting one minute long

artifact-free EEG samples into the PDP 8/e which yielded a histogram of the power between 0 to 60 Hz with an interval width of 1 Hz. (Several spectra of the power between 0 to 100 Hz were also computed.) All averaged responses, differences between evoked responses and power spectra were plotted with a Moseley X-Y plotter.

The recording system in Experiment I was calibrated by inputting a 4.7 volt square wave to the external calibrate jacks of the P9 amplifiers, setting the voltage divider in each to 500 μV and tape recording the output of the oscilloscopes. In Experiment II a relay connected across the terminals of the internal calibrator switch of the polygraph was closed and opened so that a square wave of 100 μV was input to the amplifiers whose output was tape recorded. In both studies the recorded square wave was averaged and used to measure evoked response amplitude.

RESULTS

General Observations.

Samples of pigeon EEG recorded in the dark and in the light are presented in Figure 5. In our recording situation, the EEG ranged in amplitude from 40 to 180 μV and appeared to consist of fast activity of up to 80 Hz, intermixed with slower potentials under 5 Hz with no apparent regular rhythmic pattern. These characteristics are similar to those found by other investigators (Schaub, 1963; Sugihara & Gotoh, 1973; Tradardi, 1966; Van Twyver & Allison, 1972; Walker & Berger, 1972). The EOG channels show the eyeblinks of the pigeon. They indicate a train of rapid (about 40 Hz) oscillations which is shorter in the light (for this bird 0.5 sec.) than in the dark (for this bird 0.8 sec.). There is a small negative deflection in the EEG traces, both in dark and light, at the onset of the blinks. In the light however, this is followed by a large positive deflection most prominent in the tecta which resembles the evoked potential produced by light flash.

One striking aspect of the EEG is the apparent lack of bilateral synchrony. While the activity of ipsilateral structures shows a certain amount of synchrony, there is much less synchrony between the contralateral structures. The power spectra for EEG frequencies between 0 to 60 Hz are presented in Figure 6 and show that the greatest amount of power occurs between 3 to 4 Hz. Power decreases quickly with increased frequency and above approximately 20 Hz it is fairly evenly distributed. There are no major differences between the spectra of light and dark recordings. Figure 6a shows four power spectra of EEG frequencies between 0 to 100 Hz for the

FIGURE 5.

EEG recordings in the dark and in the light. The six channels in each sample from top to bottom are: Left Tectum, Left Rotundus, Right Tectum, Right Rotundus, Left EOG, Right EOG. Note that the gain on the EOG channels is greater.

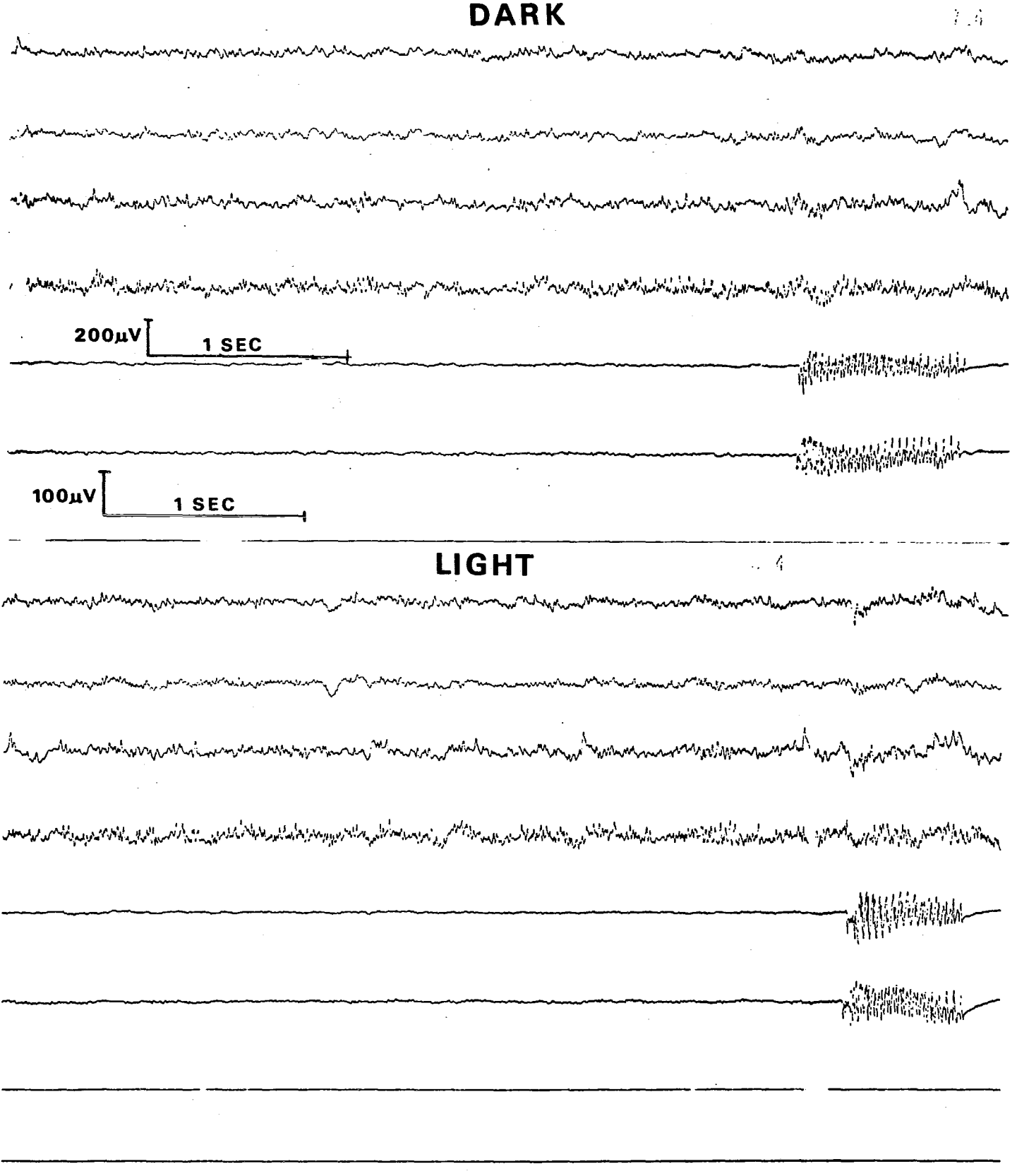

DARK
200µV
1 SEC
100µV
1 SEC
LIGHT

FIGURE 6.

Power spectra of EEG in Fig. 5. A. and E. Left Tectum. B. and F. Right Tectum. C. and G. Left Rotundus. D. and H. Right Rotundus. Vertical scales are calibrated in the percentage of total power in each spectrum, horizontal scales are in Hz.

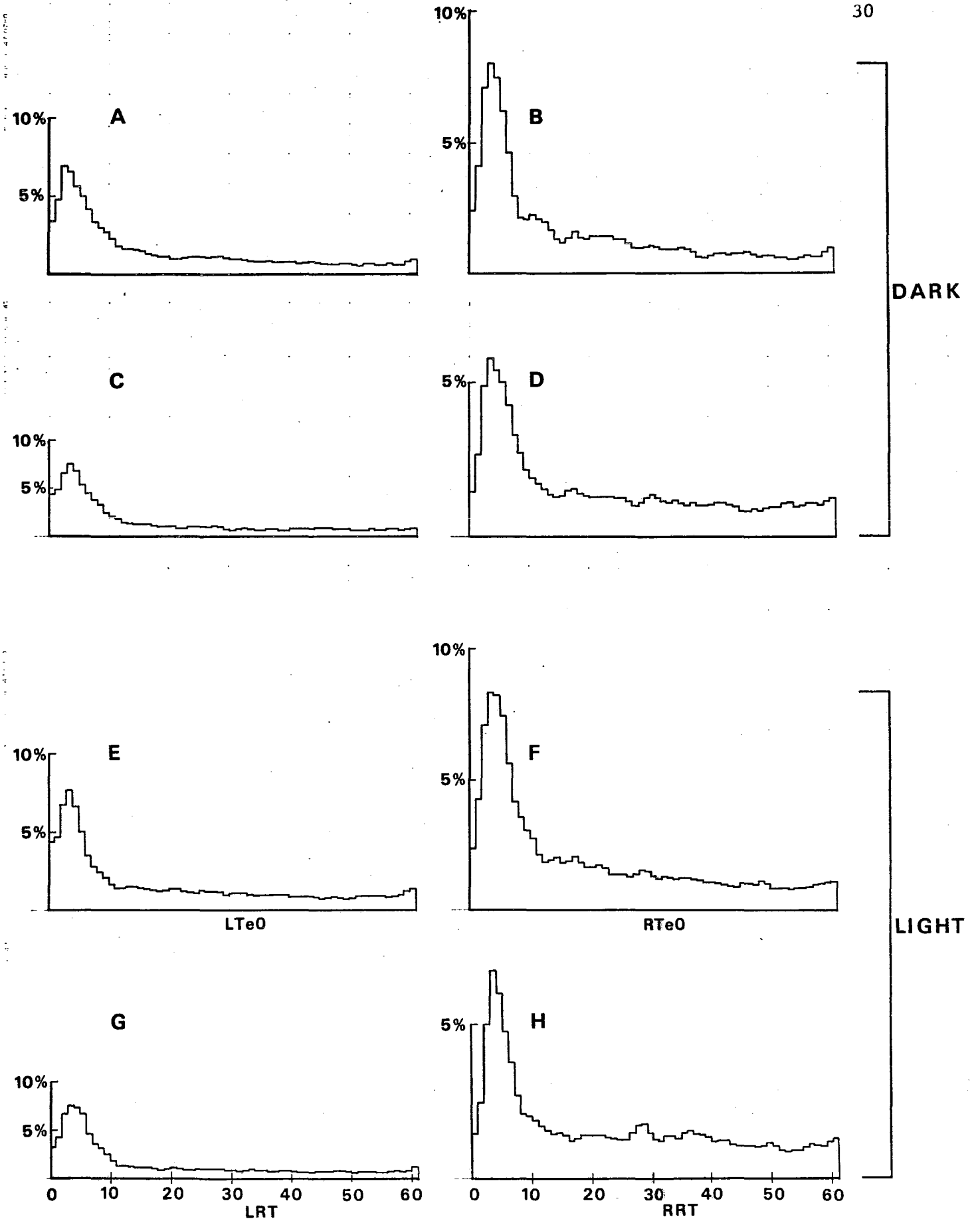
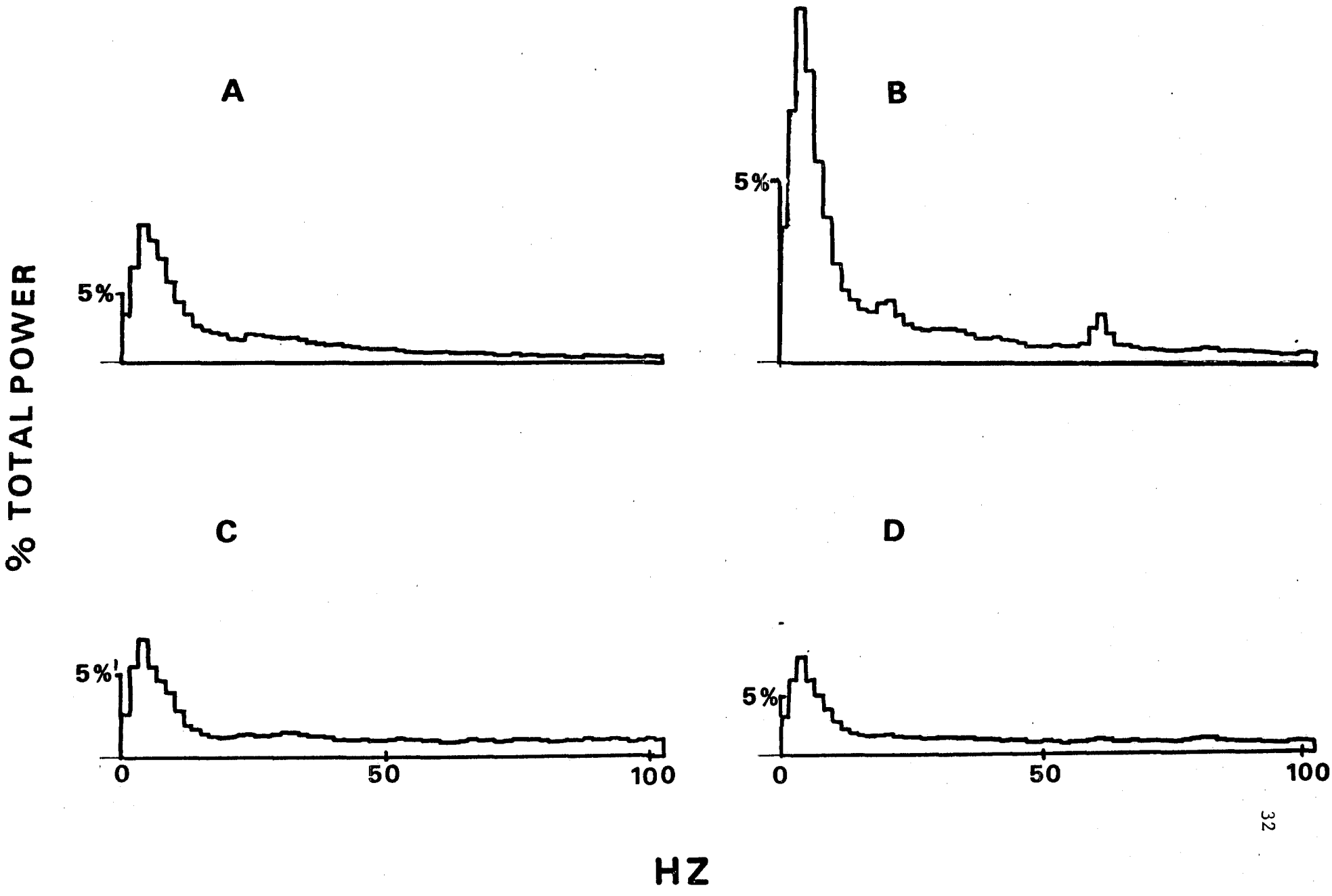

10%
A
5%
10%
B
5%
DARK
10%
C
5%
5%
D
10%
E
5%
LTeO
10%
F
5%
RTeO
LIGHT
10%
G
5%
0 10 20 30 40 50 60
LRT
5%
H
0 10 20 30 40 50 60
RRT
HZ

FIGURE 6a. Power spectra of frequencies between 0 and 100 Hz for dark recorded EEG of Figure 5. A, B, C, and D as in Figure 6. The small peak at 60 Hz in spectrum B is due to an artifact.


A
B
C
D
5%
5%
5%
5%
% TOTAL POWER
0
50
100
0
50
100
HZ

dark recorded data of Figure 5. Note that while activity is present between 60 to 100 Hz it is evenly distributed.

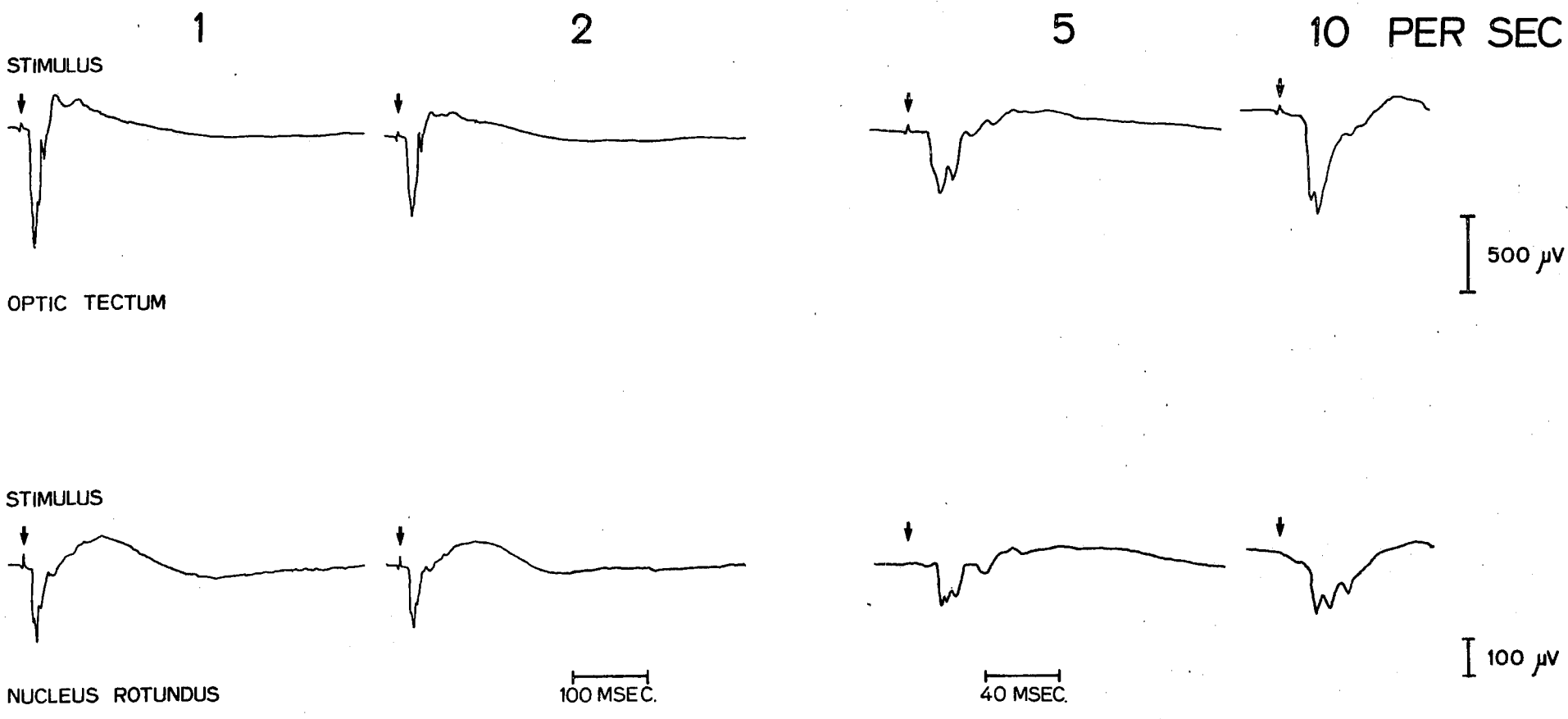
Certain well-defined changes are observed in the responses to photic stimulation during the implantation of the electrode into the optic tectum. The most marked is a reversal in polarity which occurs when the electrode enters the third layer of the tectum, the major deflection in the response changing from negative to positive. Our electrodes, on the basis of stereotaxic coordinates and the more precise criterion of the pattern of the evoked response (see Galifret, 1966, p.80a), were located in the stratum album centrale, the layer which consists of axons leaving the tectum. The response recorded at this point (see Fig. 7A, 1/sec.) is comprised of an immediate positive deflection followed by a negative potential which then returns to its baseline level.

The passage of the electrode through the nucleus rotundus does not reveal evoked response changes as dramatic as those seen in the optic tectum. A small positive potential appears as the electrode enters the optic lobe and it gradually grows in amplitude as the electrode is progressively lowered. Electrodes were fixed when the largest response was obtained. This response corresponded in shape to that reported by other investigators (e.g. Samson, 1968; Yazulla & Granda, 1972) and was observed at stereotaxic coordinates which indicated that the electrode tip was situated in the central area of the nucleus rotundus. The rotundal response (see Fig. 7B, 1/sec.) is similar to that of the tectum but is smaller in total amplitude. A large positive component occurs first, followed by a negative deflection then a small, slow positive potential.

The rate of photic stimulation has a marked effect on the waveform and amplitude of both the tectal and rotundal responses (see Fig. 7). The

FIGURE 7.

Typical tectal and rotundal evoked responses. Stimulation rates are at top. Note that the 100 msec. time mark refers to the 1 and 2 per sec. responses, the 40 msec. to the 5 and 10 per sec.


1
2
5
10 PER SEC
STIMULUS
500 µV
OPTIC TECTUM
STIMULUS
100 µV
NUCLEUS ROTUNDUS
100 MSEC.
40 MSEC.

amplitudes of the responses decrease as rate increases to 5/sec. but at 10/sec. there is an increase in the amplitudes of the positive potentials though the amplitudes of the negative components continue to decline. This effect (see Fig. 8) is similar to photic driving in humans (Cobb, 1963; Speckreijse, 1966), where the amplitude of the evoked response increases at 10/sec., and to the response potentiation seen in the cortex of the cat at 10 and 20/sec. (Sturr & Shansky, 1971). Figure 9 shows responses from Experiment I to a wide range of stimulation rates but no potentiation effect. Since the intensity of the stimulus used in Experiment II was greater than that in Experiment I, the effect's appearance in the former but not the latter study suggests that it is dependent on stimulus intensity.

Experiment I

Evoked Responses

In the first experiment each eye of the bird was deprived of patterned visual input for one week (see Fig. 4 for procedure). The evoked potentials recorded from pigeon 39 with stimulation of 1/sec. (see Fig. 10) are the most reliable obtained in Experiment I. Inspection reveals, however, that the responses from the control sessions fluctuate in total amplitude and waveform. This variability becomes even clearer when the response amplitudes shown in Table I through IV are examined. Analysis of the means of the amplitudes showed no sign of an effect.

The data were further analyzed by means of the excursion score. The difference waveforms for the data of Figure 10 are plotted in Figure

FIGURE 8.

Component amplitudes for responses in Figure 7. A. Optic Tectum. B. Nucleus Rotundus.

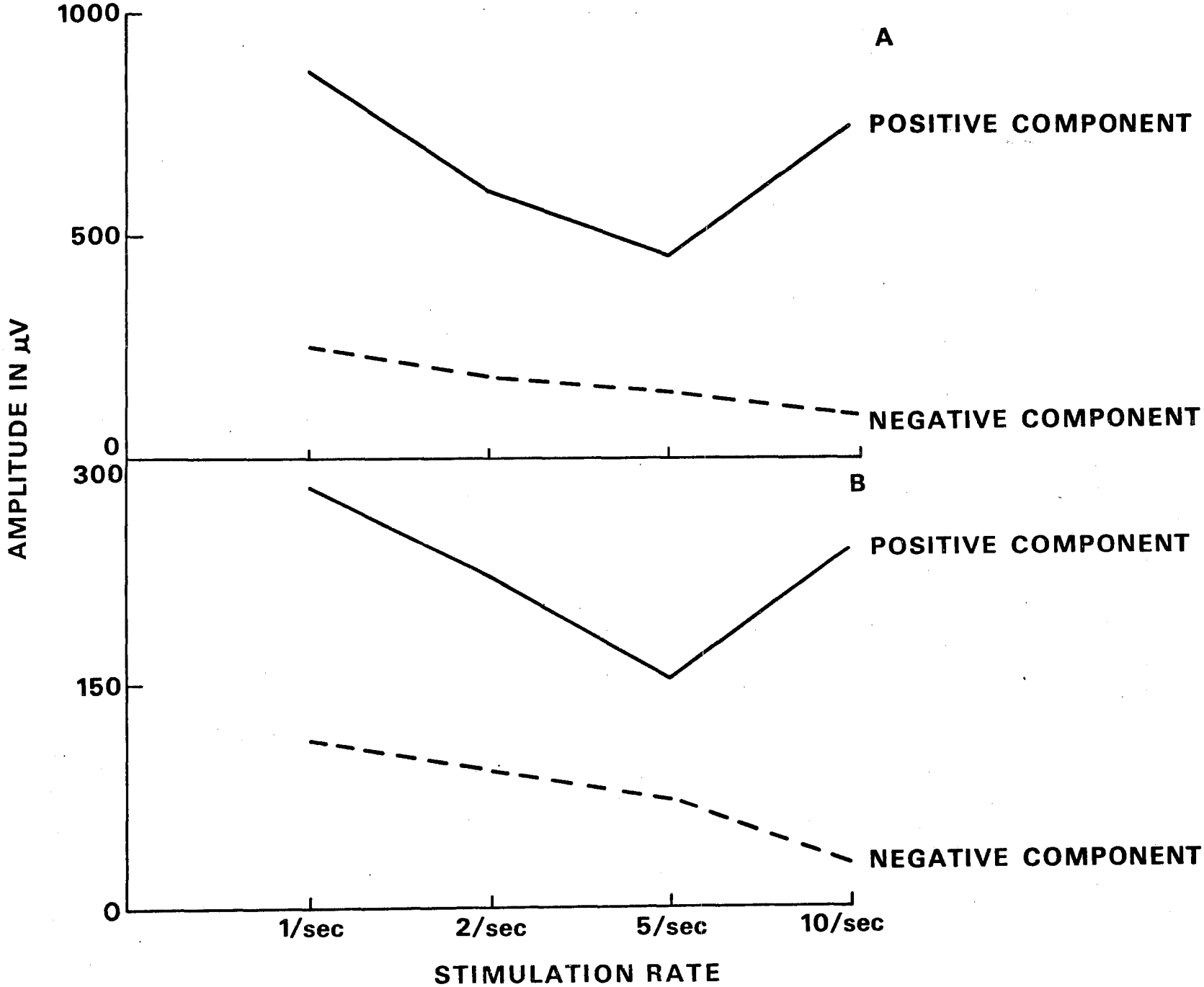

1000
500
0
300
150
0
AMPLITUDE IN μV
A
POSITIVE COMPONENT
NEGATIVE COMPONENT
B
POSITIVE COMPONENT
NEGATIVE COMPONENT
1/sec
2/sec
5/sec
10/sec
STIMULATION RATE

FIGURE 9.

Typical evoked responses from Experiment I. A. Optic Tectum. B. Nucleus Rotundus. For rates 0.5 to 5 per sec., responses to 50 stimuli were averaged. For 10, 20 and 50 per sec., 100, 200 and 500 responses were averaged respectively.

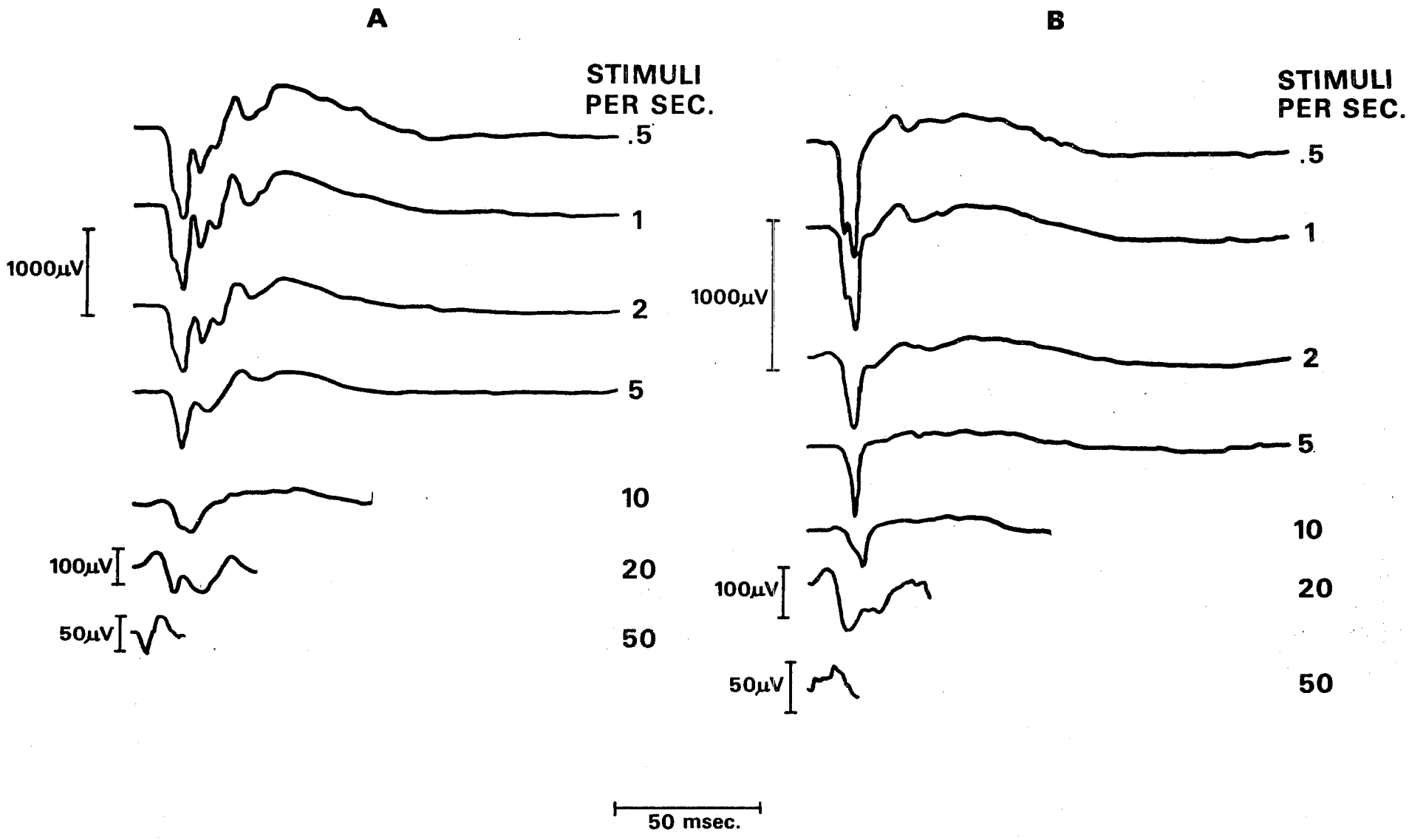
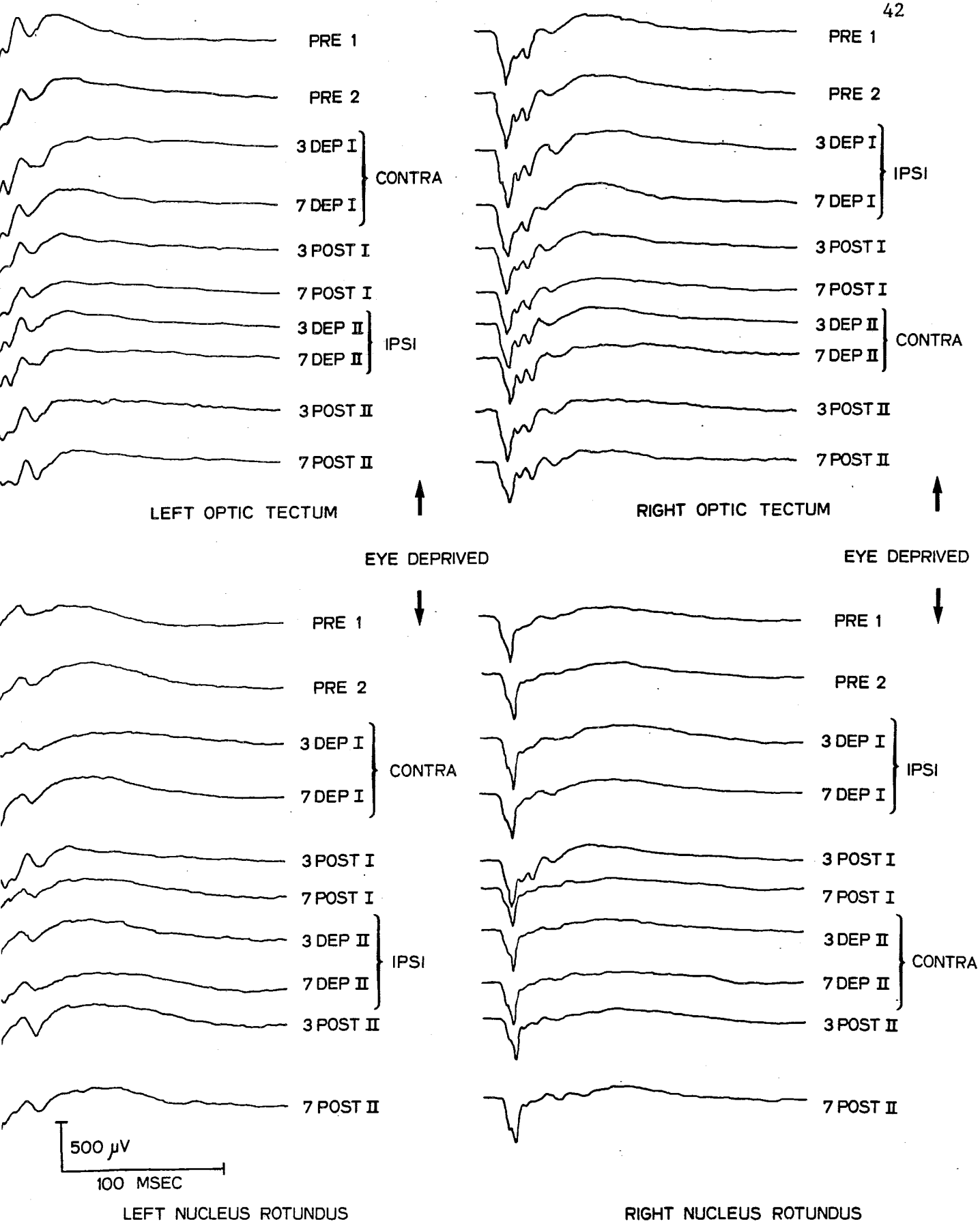

A
B
STIMULI
PER SEC.
.5
1
2
5
10
20
50
1000μV
100μV
50μV
50 msec.

FIGURE 10.

Evoked responses of bird 39 in Experiment I. The bird was deprived of pattern vision during both deprivation episodes. The labels "CONTRA" and "IPSI" refer to which eye was deprived, the contralateral or the ipsilateral. Stimulation rate: 1/sec.



EVOKED RESPONSES

TABLE I

Amplitudes of Tectal Responses for Birds 39 and 46 in μV.

BIRDS	39						46					
EYE DEPRIVED	CONTRA			IPSI			CONTRA			IPSI		
COMPONENT	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.
PRE 1	1333	939	394	1153	878	275	973	711	262	1133	865	268
PRE 2	1301	973	328	1213	925	288	962	721	241	1054	790	264
3 DEP I	1338	1042	296	1255	951	304	898	680	218	1074	804	270
7 DEP I	1317	979	338	1196	836	360	812	622	190	1117	794	323
3 POST I	1070	773	297	1027	762	265	725	546	179	904	658	246
7 POST I	1021	787	234	905	679	226	763	582	181	862	625	237
EYE DEPRIVED	IPSI			CONTRA			IPSI			CONTRA		
3 DEP II	1097	816	281	999	733	266	796	592	204	936	692	244
7 DEP II	1005	763	242	967	740	227	840	621	219	1139	911	228
3 POST II	1247	993	254	1149	908	241	723	525	198	1038	810	228
7 POST II	1260	980	280	1352	1045	307	802	615	187	1139	866	273

TABLE II

Amplitudes of Tectal Responses for Birds 6963 and 83 in μV.

BIRDS	6963						83					
EYE DEPRIVED	CONTRA			IPSI			CONTRA			IPSI		
COMPONENT	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.
PRE 1	1828	1279	549	1773	1289	484	1366	945	421	920	637	283
PRE 2	1643	1165	478	1607	1191	416	1303	867	436	887	548	339
3 DEP I	2000	1547	453	1798	1381	417	1848	1391	457	1000	670	330
7 DEP I	1871	1429	442	1619	1237	382	1282	900	382	946	607	339
3 POST I	1471	1071	400	1405	1084	321	1180	848	332	871	616	255
7 POST I	1557	1195	362	1465	1124	341	1519	1066	453	909	620	289
EYE DEPRIVED	IPSI			CONTRA			IPSI			CONTRA		
3 DEP II	1457	1090	367	1357	986	371	1747	1284	463	1086	810	276
7 DEP II	1414	979	435	1381	1073	308	1519	1126	393	1107	835	272
3 POST II	1600	1199	401	1310	986	324	1836	1460	376	974	726	248
7 POST II	1893	1402	491	1742	1441	301	1955	1515	440	969	770	199

TABLE III

Amplitudes of Rotundal Responses for Birds 39 and 46 in μV.

BIRDS	39						46					
EYE DEPRIVED	CONTRA			IPSI			CONTRA			IPSI		
COMPONENT	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.
PRE 1	874	709	165	918	760	158	-	-	-	586	432	154
PRE 2	940	726	214	893	721	172	557	388	169	548	404	144
3 DEP I	636	532	104	1041	840	201	-	-	-	589	395	194
7 DEP I	1000	758	242	944	719	225	682	442	240	549	362	187
3 POST I	775	596	179	761	611	150	573	365	208	484	329	155
7 POST I	765	603	162	740	584	156	569	363	206	505	345	160
EYE DEPRIVED	IPSI			CONTRA			IPSI			CONTRA		
3 DEP II	818	630	188	780	617	163	609	413	196	578	417	161
7 DEP II	738	575	163	821	653	168	541	376	165	568	406	162
3 POST II	821	628	193	821	650	171	609	413	196	486	329	157
7 POST II	894	694	200	882	688	194	617	435	182	508	382	126

TABLE IV

Amplitudes of Rotundal Responses for Birds 6963 and 83 in μV.

BIRDS	6963						83					
EYE DEPRIVED	CONTRA			IPSI			CONTRA			IPSI		
COMPONENT	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.	TOTAL	POS.	NEG.
PRE 1	555	351	204	584	391	193	546	402	144	433	291	142
PRE 2	527	359	168	550	390	160	597	437	160	427	280	147
3 DEP I	540	438	102	641	498	143	597	457	140	437	308	129
7 DEP I	540	395	145	625	459	166	587	403	184	450	278	172
3 POST I	405	256	149	533	381	152	530	374	156	427	294	133
7 POST I	460	333	127	550	424	126	505	356	149	398	278	120
EYE DEPRIVED	IPSI			CONTRA			IPSI			CONTRA		
3 DEP II	438	316	122	543	407	136	531	384	147	408	299	109
7 DEP II	388	254	134	516	389	127	566	417	149	500	365	135
3 POST II	474	366	108	583	474	109	581	392	189	500	375	125
7 POST II	546	440	106	655	496	159	582	433	149	491	361	130

11 and highlight the shifts in response characteristics. When the degree of excursion in each difference waveform is calculated and plotted the graphs presented in Figure 12 result. Here again, variability during control sessions makes interpretation difficult in the cases of the left tectum and left rotundus. The structures of the right optic lobe show decreases in the excursion score between sessions 7 DEP I and 3 POST I, but no corresponding rises prior to deprivation.

Experiment II

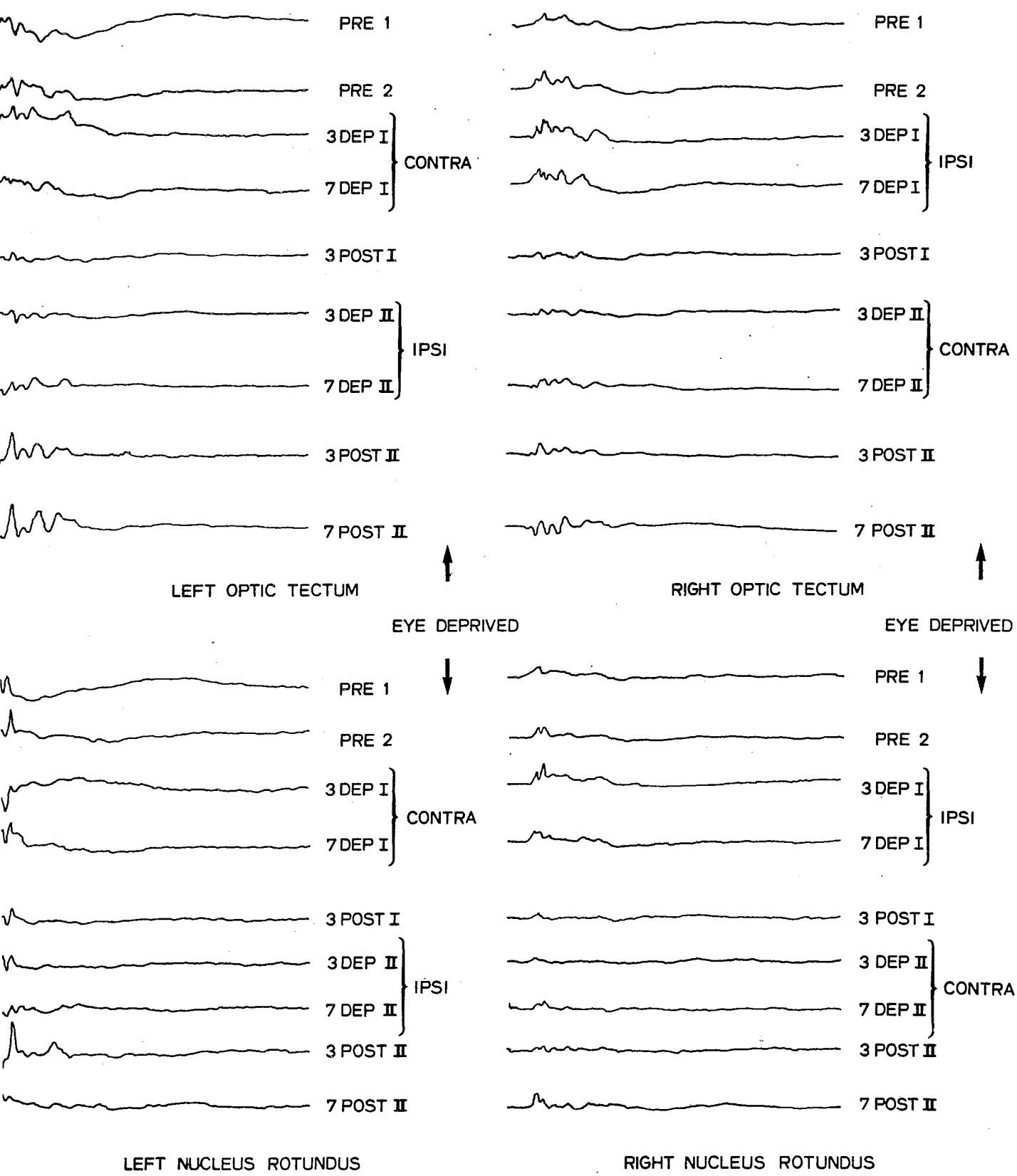
Evoked Responses

Deprivation of total visual input to one eye for one week produced marked changes in the evoked potentials of the contralateral optic tectum and nucleus rotundus. One effect was on the shape of the evoked potential; generally it became "smoother" with deprivation and smaller oscillations, particularly later ones, disappeared. There was also an overall tendency for the amplitude of the response to increase. During the first deprivation procedure, the only effect seen in the lobe ipsilateral to the covered eye was an increase in the amplitude of the response of the nucleus rotundus. Curiously enough, when the monocular deprivation procedure was repeated, with the previously uncovered eye deprived of light, responses from both optic lobes changed in shape, but only the lobe contralateral to the covered eye showed a clear increase in response amplitude.

For bird 84 the evoked responses to the 10/sec. stimulation rate (see Fig. 13) best illustrate these effects. Rotundal and tectal responses recorded during periods when the contralateral eye was covered all show a

FIGURE 11.

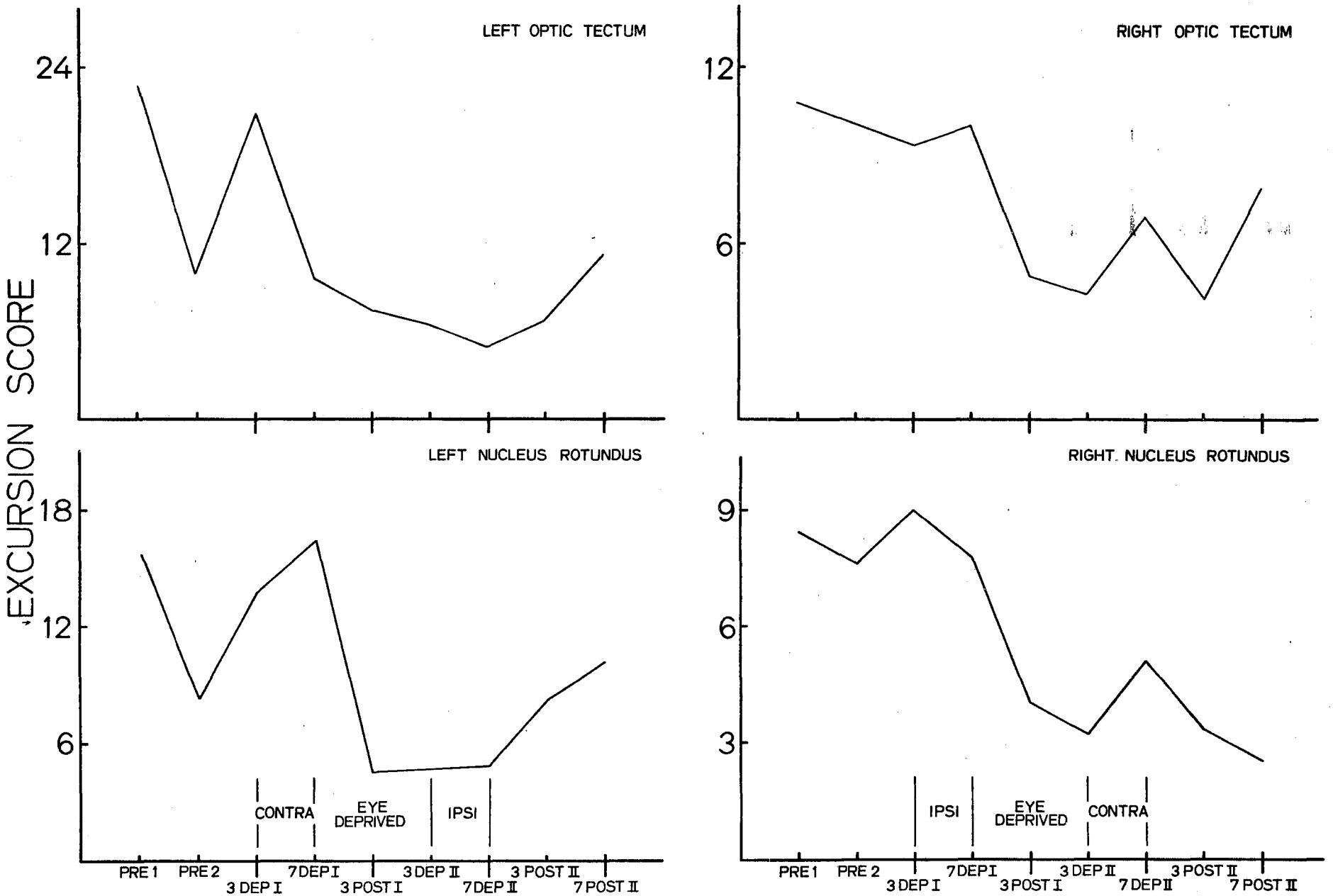
Difference waveforms for responses in Fig. 10. Each waveform is the result of subtracting a given response from the corresponding one on 7 POST I.



DIFFERENCE WAVEFORMS

FIGURE 12.

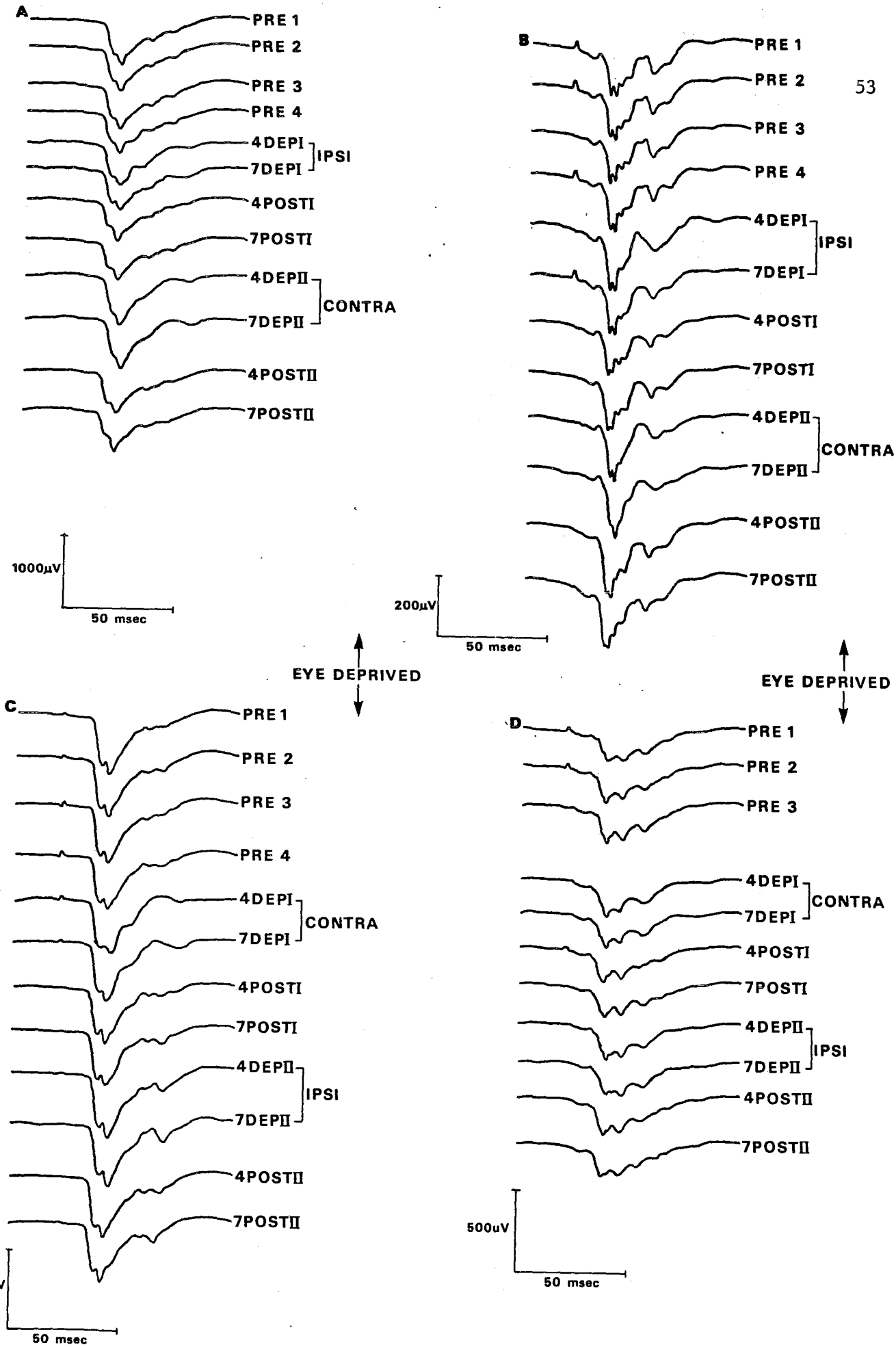
Excursion scores for difference waveforms in Fig. 11.



DIFFERENCE WAVEFORM

FIGURE 13.

Evoked responses of bird 84 to 10/sec. stimulation. A. Left Tectum. B. Left Rotundus. C. Right Tectum. D. Right Rotundus. (Response for PRE 4 not recorded due to an equipment failure.)


A
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
IPSI
4POSTI
7POSTI
4DEPII
7DEPII
CONTRA
4POSTII
7POSTII
1000µV
50 msec
B
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
IPSI
4POSTI
7POSTI
4DEPII
7DEPII
CONTRA
4POSTII
7POSTII
200µV
50 msec
EYE DEPRIVED
EYE DEPRIVED
C
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
CONTRA
4POSTI
7POSTI
4DEPII
7DEPII
IPSI
4POSTII
7POSTII
50 msec
D
PRE 1
PRE 2
PRE 3
4DEPI
7DEPI
CONTRA
4POSTI
7POSTI
4DEPII
7DEPII
IPSI
4POSTII
7POSTII
500uV
50 msec

decline in the number of faster components or their replacement by slower waves. The positive peak amplitudes of the responses of the left tectum and left rotundus increase during contralateral deprivation but the effect on the amplitudes of the right tectal and rotundal responses is not as clear. The waveform of the left rotundal response ipsilateral to the covered eye changes little during the first occlusion, but its amplitude increases. With the second occlusion ipsilateral rotundal and tectal responses alter in shape but no clear effect on amplitude emerges.

The amplitudes of the positive peaks for all responses recorded from pigeons 84 and 67 are shown in Figures 14 and 15, respectively. Note that where the order of monocular deprivation of an optic lobe is ipsilateral eye covered--contralateral eye covered, the amplitude of the positive peak of the rotundal response increases during the first occlusion at session 4 DEP I while no changes take place for the tectal response. With contralateral deprivation, however, both tectal and rotundal responses increase in amplitude, though the increase for the rotundal response of bird 84 is not as strong as it is for that of 67. The effect of occlusion for visual structures that were first contralaterally then ipsilaterally deprived is not as clear. For bird 84 no clear effect emerges except perhaps at 7 DEP II where rotundal response amplitude generally decreases. Bird 67, on the other hand, shows an increase in rotundal response amplitude during both episodes and similar but less powerful effects on the tectal response.

In order to examine the changes in the shape of the potentials more closely the procedure for calculating the amount of change in a response (excursion score) was used. The difference waveforms for the responses of Figure 13 are plotted in Figure 16, where the degree of

FIGURE 14.

Positive component amplitudes for all responses of bird 84. Deprivation of light during both deprivation episodes.

A. Left Tectum. B. Right Tectum. C. Left Rotundus. D. Right Rotundus.

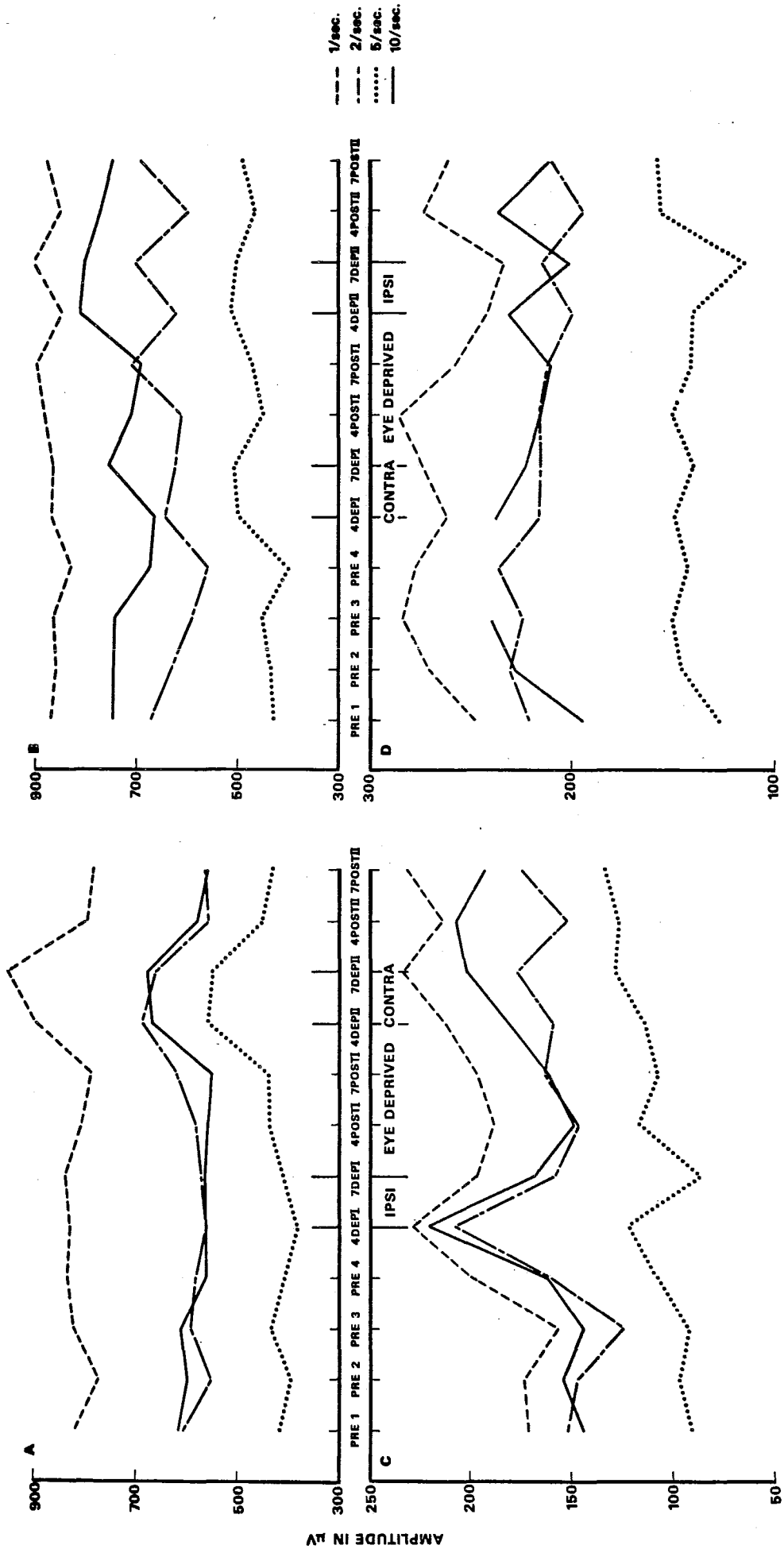

1/sec.
2/sec.
5/sec.
10/sec.
A
B
C
D
AMPLITUDE IN μV
900
700
500
300
250
200
150
100
50
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
4POSTI
7POSTI
4DEPII
7DEPII
4POSTII
7POSTII
IPSI
EYE DEPRIVED
CONTRA

FIGURE 15.

Positive component amplitudes for all responses of bird 67. Deprivation of light during both deprivation procedures. A. Left Tectum. B. Right Tectum. C. Left Rotundus. D. Right Rotundus.

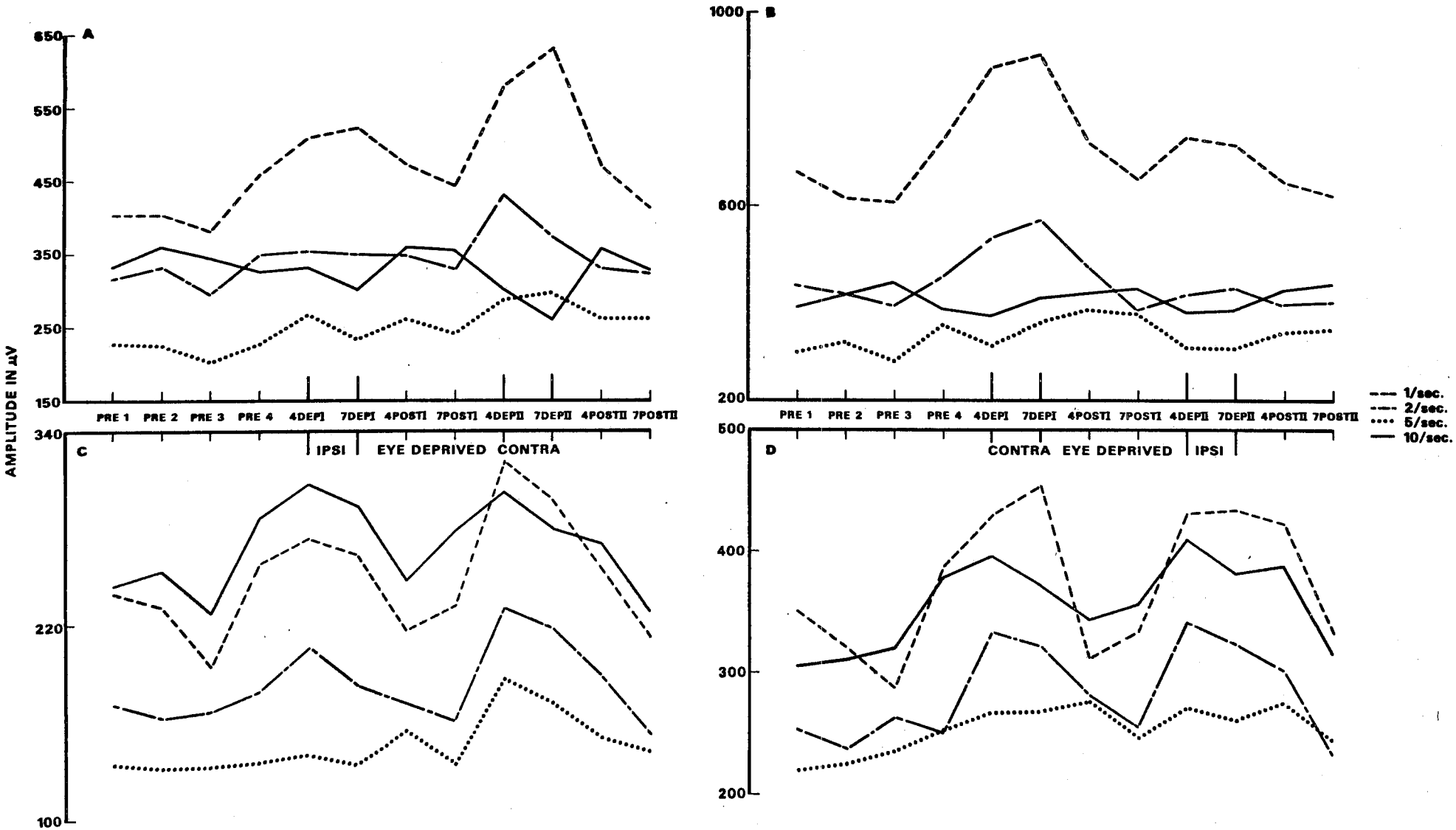
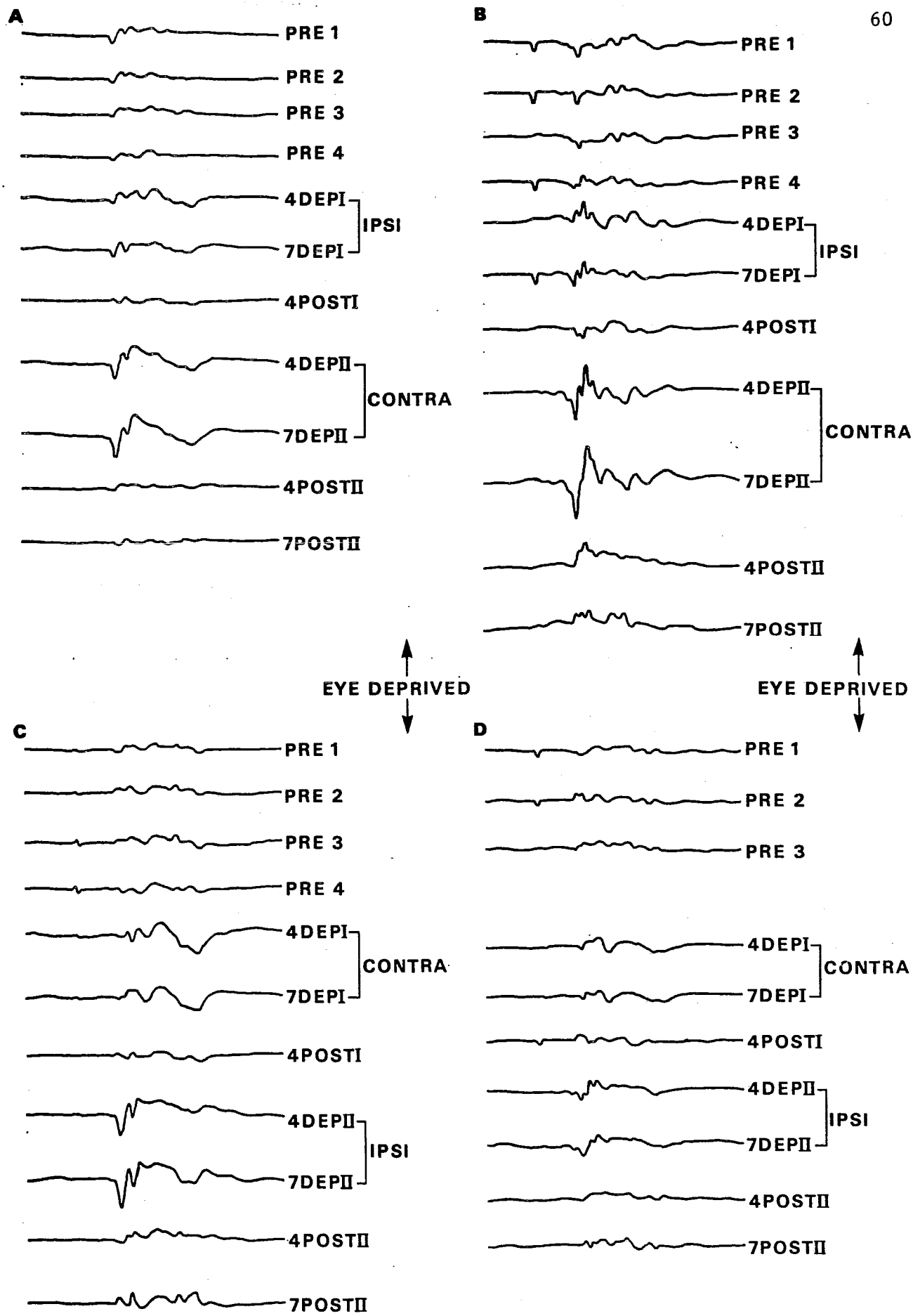

A
650
550
450
350
250
150
AMPLITUDE IN μV
PRE 1 PRE 2 PRE 3 PRE 4 4DEPI 7DEPI 4POSTI 7POSTI 4DEPII 7DEPII 4POSTII 7POSTII
B
1000
600
200
PRE 1 PRE 2 PRE 3 PRE 4 4DEPI 7DEPI 4POSTI 7POSTI 4DEPII 7DEPII 4POSTII 7POSTII
1/sec.
2/sec.
5/sec.
10/sec.
C
340
220
100
IPSI EYE DEPRIVED CONTRA
D
500
400
300
200
CONTRA EYE DEPRIVED IPSI

FIGURE 16.

Difference waveforms for data in Fig. 13. A. Left Tectum. B. Left Rotundus. C. Right Tectum. D. Right Rotundus.

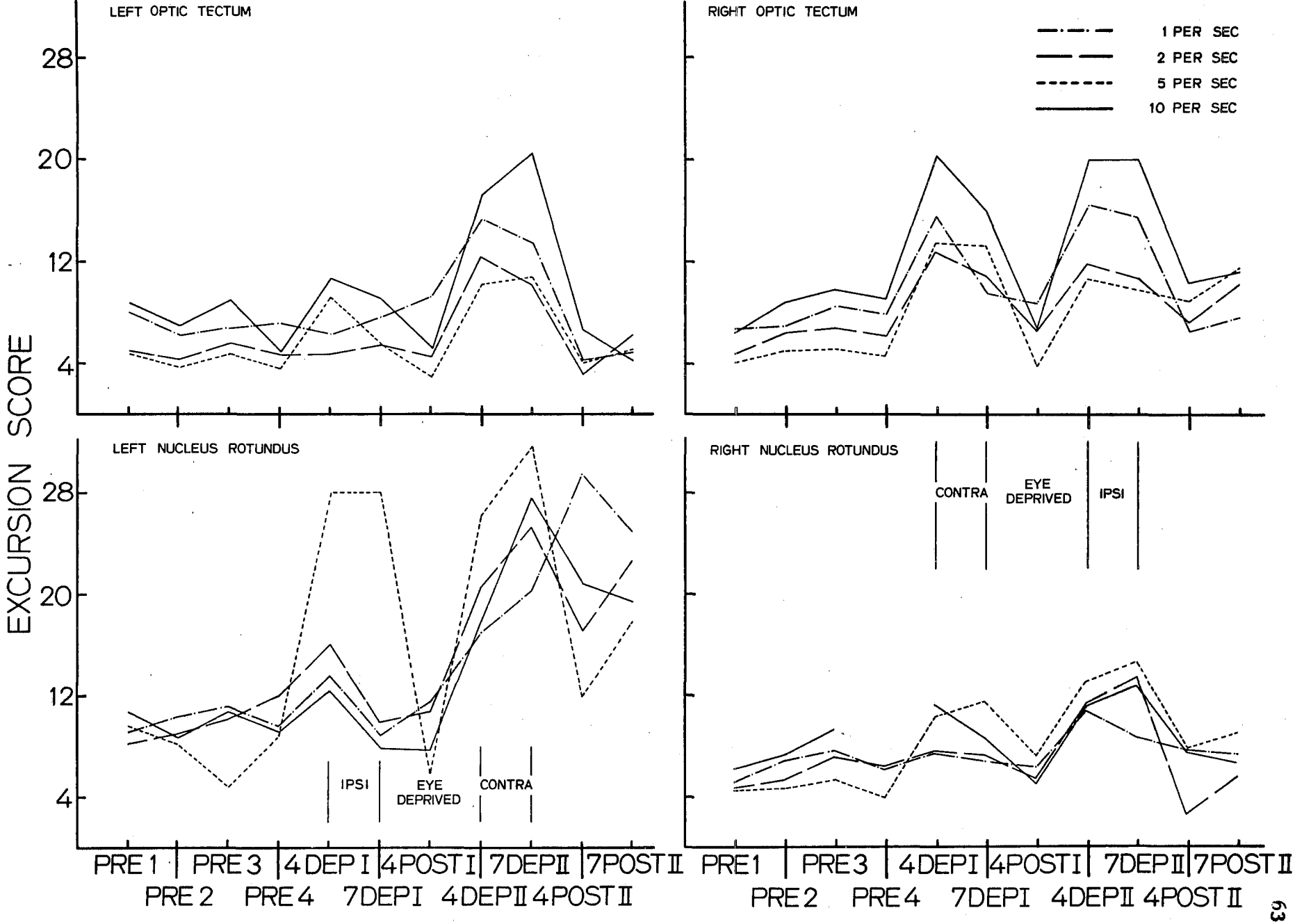

A
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
IPSI
4POSTI
4DEPII
7DEPII
CONTRA
4POSTII
7POSTII
B
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
IPSI
4POSTI
4DEPII
7DEPII
CONTRA
4POSTII
7POSTII
EYE DEPRIVED
EYE DEPRIVED
C
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
CONTRA
4POSTI
4DEPII
7DEPII
IPSI
4POSTII
7POSTII
D
PRE 1
PRE 2
PRE 3
4DEPI
7DEPI
CONTRA
4POSTI
4DEPII
7DEPII
IPSI
4POSTII
7POSTII

excursion in a waveform reflects the amount of difference between the response on a given session and the same response on 7 POST I. For the optic tecta, during the first occlusion, only the difference waveform for the structure contralateral to the covered eye shows large oscillations, but during the second they appear in both. The rotundal waveforms show a similar pattern of effects except for the oscillations in the left rotundal waveform on 4 DEP I. The amount of excursion in each difference waveform for birds 84 and 67 is graphed in Figures 17 and 18, respectively. When the order of deprivation for the tectum and rotundus is ipsilateral eye covered first, contralateral covered second, only the second procedure produces marked changes in the shape of the evoked potentials, though bird 84 does show a small change with ipsilateral deprivation. When the order of deprivation is contralateral eye first, ipsilateral second, both events produce changes in the shapes of the tectal and rotundal potentials, with the possible exception of the right rotundus of bird 84 during contralateral deprivation.

One animal in Experiment II was deprived of patterned input to one eye during the first occlusion and then of all input to the other during the second. The results of the first procedure are unclear but those of the second are certain. Figure 19 shows the changes in the responses of bird 85 to a 10/sec. flash rate. With contralateral deprivation of pattern vision the only change is in the smaller amplitude of the third positive peak in the left tectal response. But with contralateral deprivation of light the same peak in the right tectal response disappears completely and the later components in the right rotundal response become smoother. In addition, the positive amplitudes of all the responses, except that of

FIGURE 17.

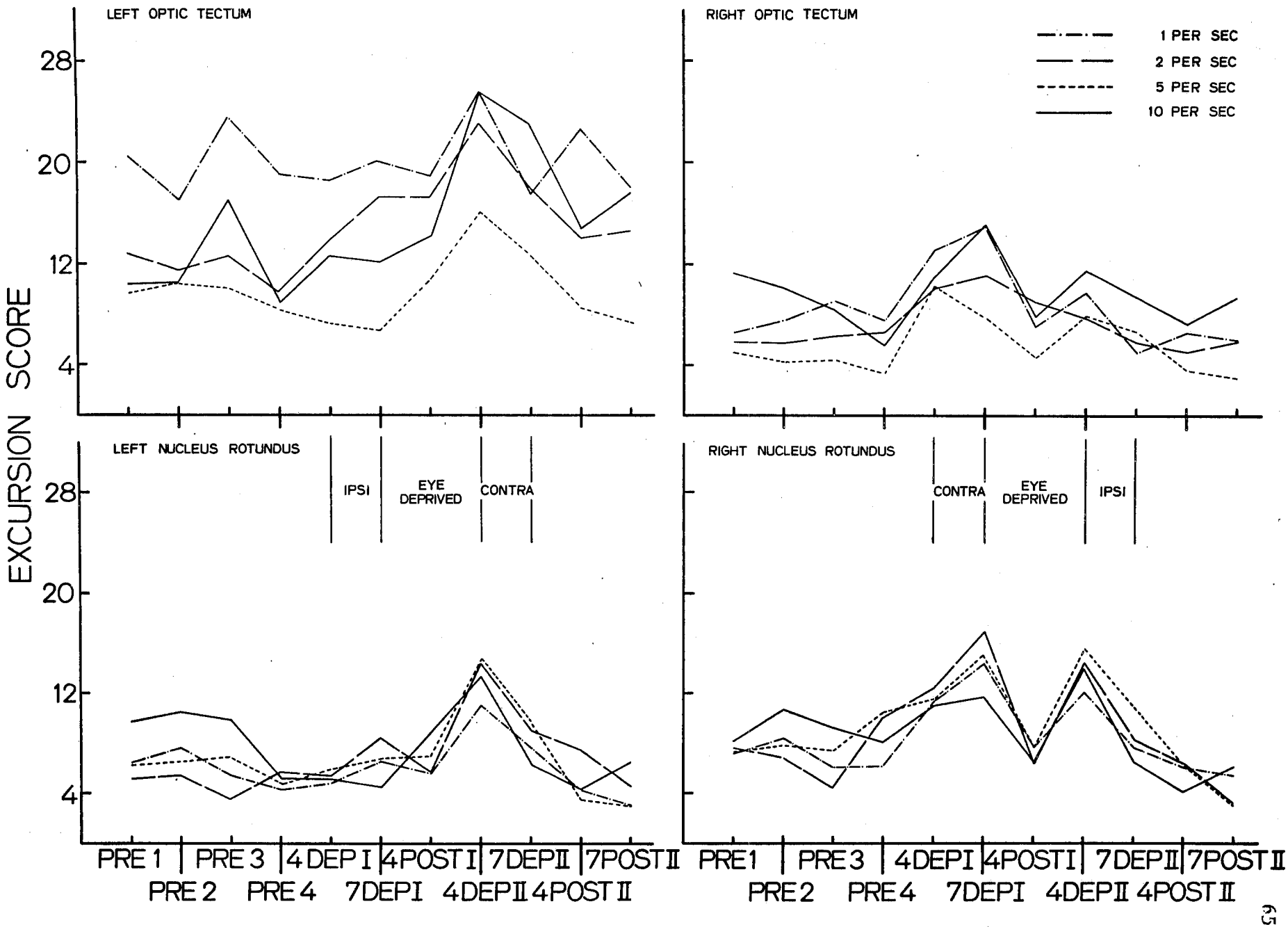
Excursion scores for bird 84. The high scores on 4DEPI and 7DEPI for the left rotundus at rate 5/sec. are due to large after-potentials in the responses.



DIFFERENCE WAVEFORM

FIGURE 18.

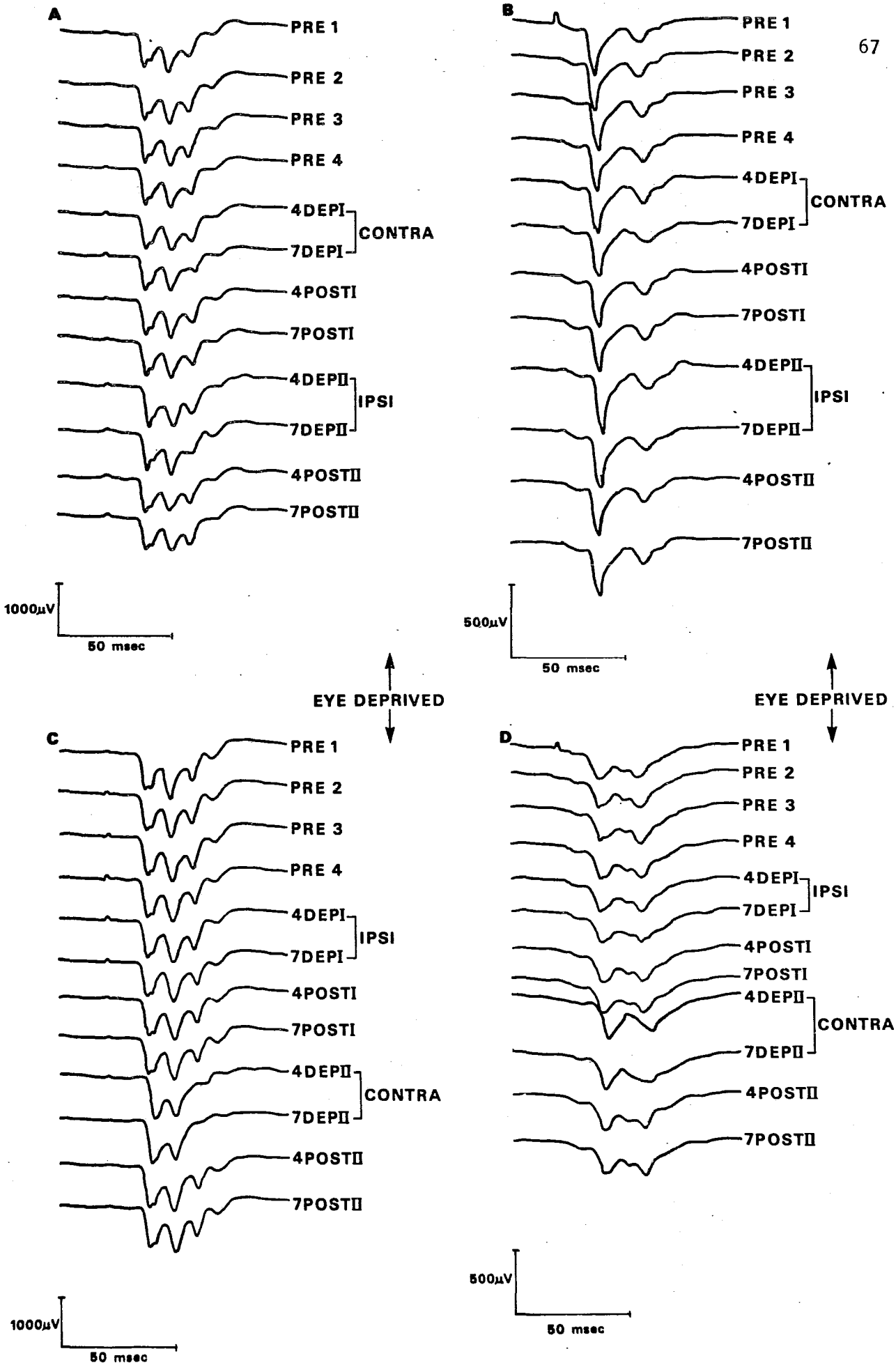
Excursion scores for bird 67.



DIFFERENCE WAVEFORM

FIGURE 19.

Evoked responses of bird 85. A. Left Tectum. B. Left Rotundus. C. Right Tectum. D. Right Rotundus. Stimulation rate: 10/sec. This bird was deprived first of patterned input, then of light.


A
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
CONTRA
4POSTI
7POSTI
4DEPII
7DEPII
IPSI
4POSTII
7POSTII
1000µV
50 msec
B
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
CONTRA
4POSTI
7POSTI
4DEPII
7DEPII
IPSI
4POSTII
7POSTII
500µV
50 msec
EYE DEPRIVED
EYE DEPRIVED
C
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
IPSI
4POSTI
7POSTI
4DEPII
7DEPII
CONTRA
4POSTII
7POSTII
1000µV
50 msec
D
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
IPSI
4POSTI
7POSTI
4DEPII
7DEPII
CONTRA
4POSTII
7POSTII
500µV
50 msec

the right tectum, increase with deprivation of light.

Figure 20 presents the positive peak amplitude of each response recorded from pigeon 85. Although both the left tectal and left rotundal responses show increases at three of the four stimulation rates on session 4 DEP I the increases appear to be too small to view as an effect of contralateral pattern deprivation. The tectum ipsilateral to the pattern deprived eye reveals no change in response amplitude and while the rotundal response decreases in amplitude at three flash rates the changes are small. Deprivation of light increases the amplitude of the contralateral rotundal response but not that of the contralateral tectum. The structures ipsilateral to the light-deprived eye both display response amplitude increases which are more pronounced for the rotundus than for the tectum.

Inspection of the difference waveforms (see Fig. 21) indicates a pattern of changes in the responses generally consistent with the pattern of amplitude increases. No large excursions are evident during pattern deprivation but monocular deprivation of all visual input produces large changes in the responses of both contralateral and ipsilateral structures. The excursion scores (see Fig. 22) also reveal no change with pattern deprivation but do show effects with deprivation of light that are stronger for the contralateral structures than the ipsilateral ones. Note, however, that the ipsilateral effects in reaction to light deprivation resemble the effects during the second and not the first episode of light deprivation in the two animals discussed above.

Visual Deprivation and the EEG

Neither deprivation of total input nor of patterned input had any

FIGURE 20.

Positive component amplitudes for responses of bird 85. A. Left Tectum. B. Right Tectum. C. Left Rotundus. D. Right Rotundus. Deprived first of patterned input, then of light.

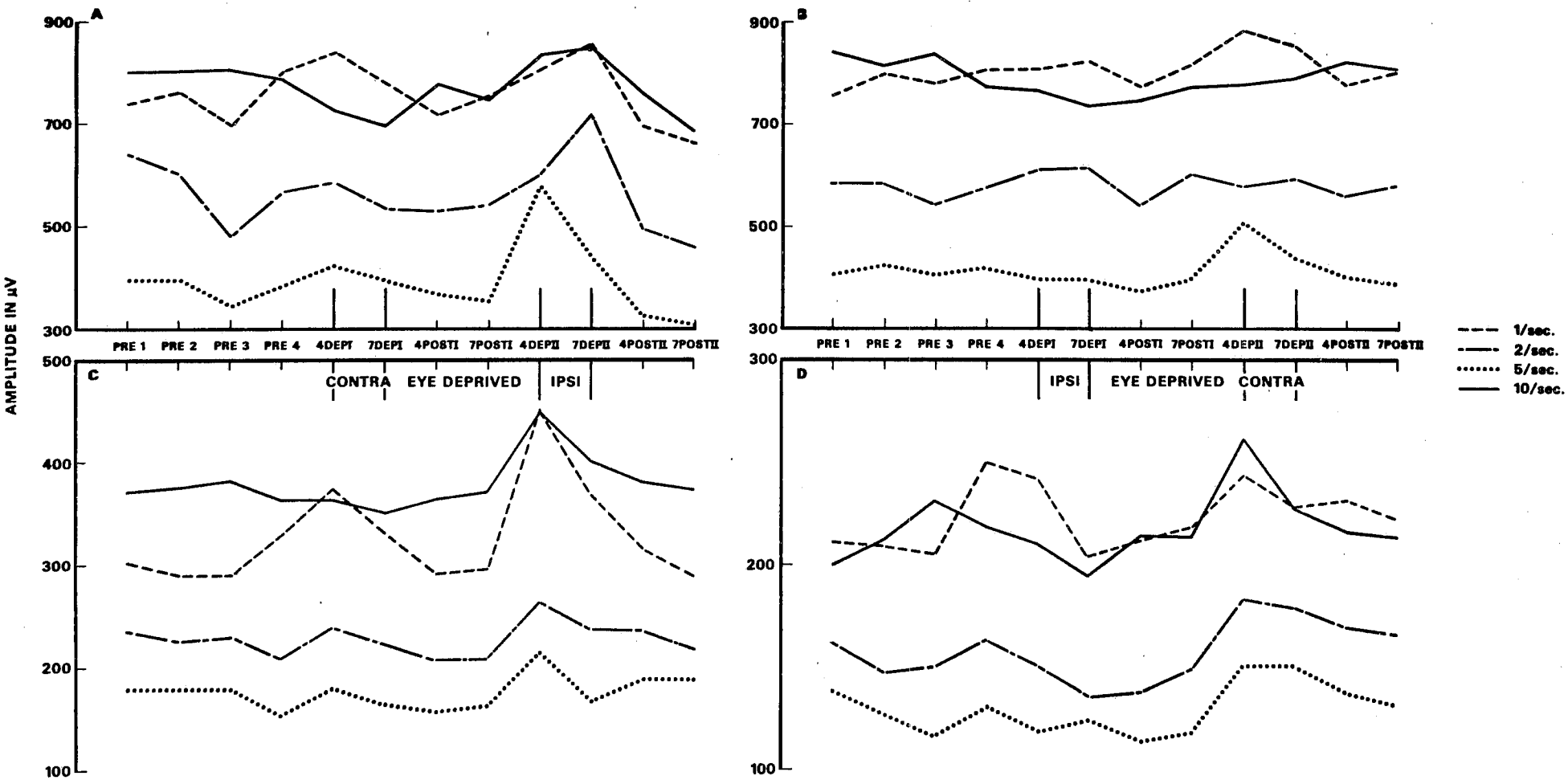

A
B
C
D
AMPLITUDE IN μV
900
700
500
300
400
200
100
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
4POSTI
7POSTI
4DEPII
7DEPII
4POSTII
7POSTII
CONTRA
EYE DEPRIVED
IPSI
IPSI
EYE DEPRIVED
CONTRA
1/sec.
2/sec.
5/sec.
10/sec.

FIGURE 21.

Difference waveforms for bird 85.

A. Left Tectum. B. Left Rotundus. C. Right Tectum. D. Right Rotundus. Pattern deprivation first, light deprivation second.

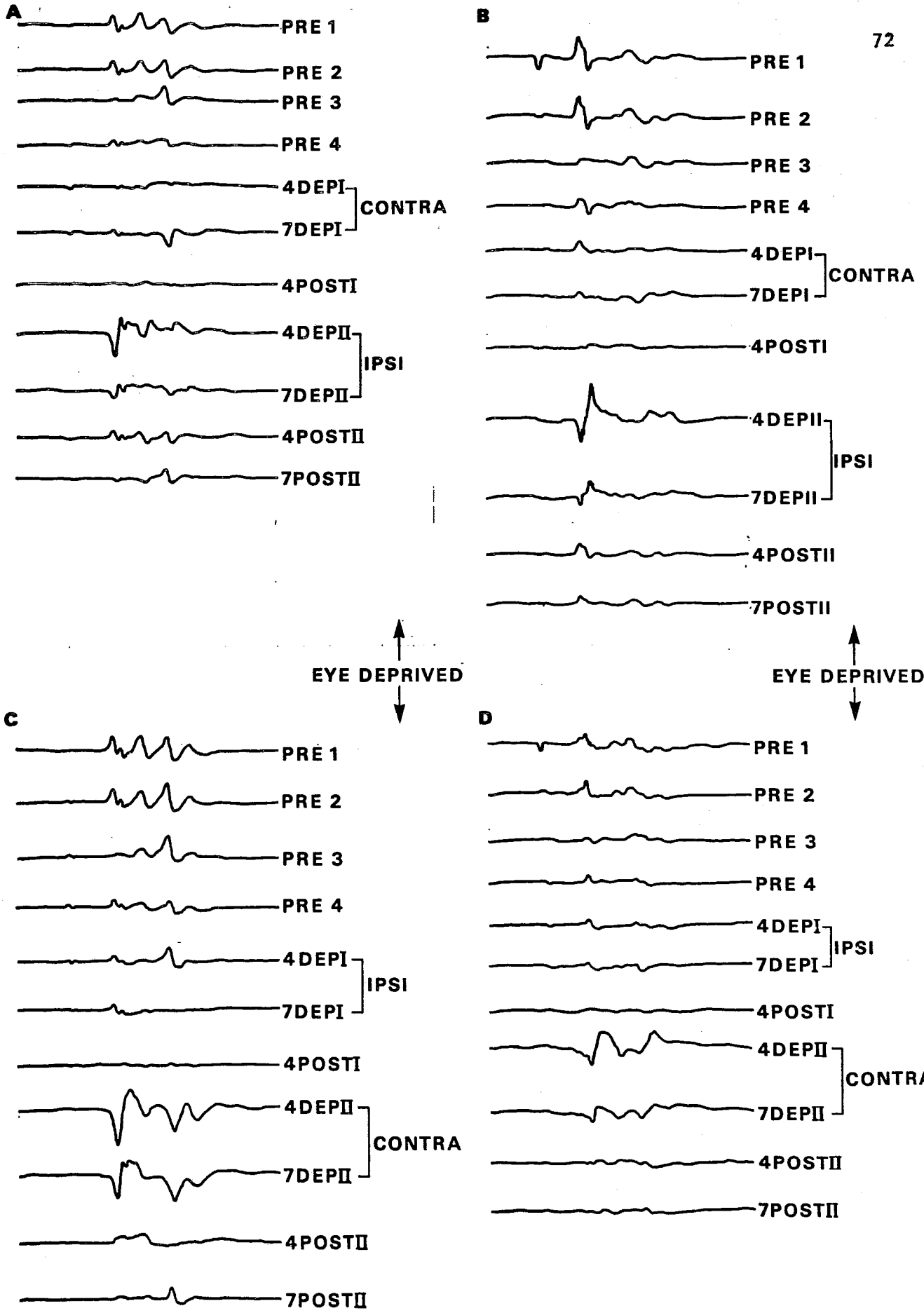
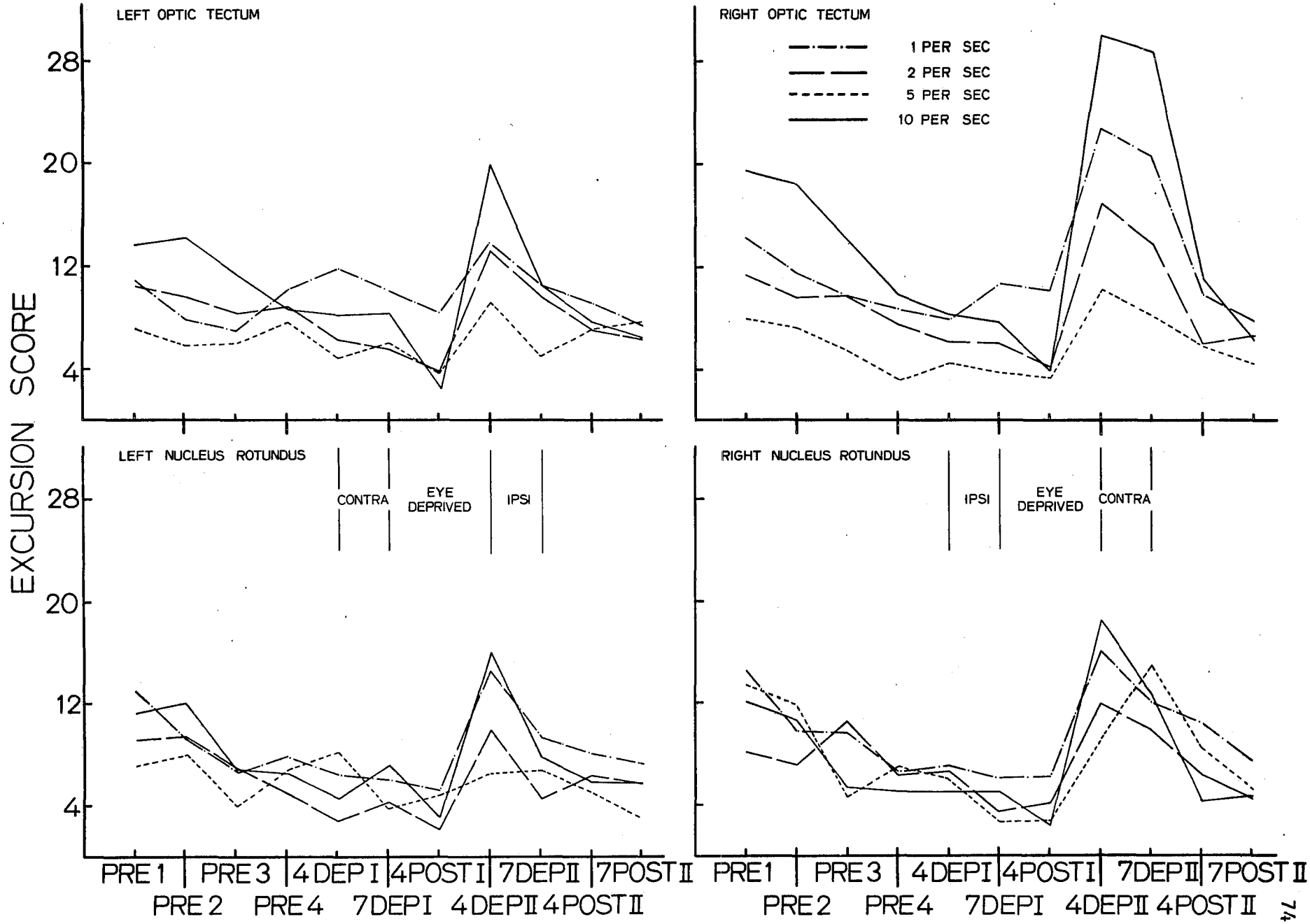

A
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
CONTRA
4POSTI
4DEPII
7DEPII
IPSI
4POSTII
7POSTII
B
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
CONTRA
4POSTI
4DEPII
7DEPII
IPSI
4POSTII
7POSTII
EYE DEPRIVED
EYE DEPRIVED
C
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
IPSI
4POSTI
4DEPII
7DEPII
CONTRA
4POSTII
7POSTII
D
PRE 1
PRE 2
PRE 3
PRE 4
4DEPI
7DEPI
IPSI
4POSTI
4DEPII
7DEPII
CONTRA
4POSTII
7POSTII

FIGURE 22.

Excursion scores for bird 85. Pattern deprivation was first, followed by deprivation of light.



DIFFERENCE WAVEFORM

recognizable effects on the EEG. Figures 23 and 24 illustrate the spontaneous brain activity recorded during the former and latter types of occlusion, respectively. Apart from the lack of deprivation-related effects on the EEG of bird 85, it is interesting that darkness did not reduce the eyeblink related evoked potential in this bird to the same degree it did in the other two animals in Experiment II.

FIGURE 23.

EEG samples from bird 84 recorded in the dark and in the light. A. and F. 7POSTI. B. 4DEPI. C. and H. 7DEPII. D. and J. 4 POSTII. E. and K. 7POSTII.

The six traces in each group represent (from top to bottom): the left optic tectum, the left nucleus rotundus, the right optic tectum, the right nucleus rotundus, the left EOG, and the right EOG. The upper two traces in B, C, and H are from structures which were contralaterally deprived of light. The animal was not exposed to light on 4DEPI, hence there are no "LIGHT" samples for that session.

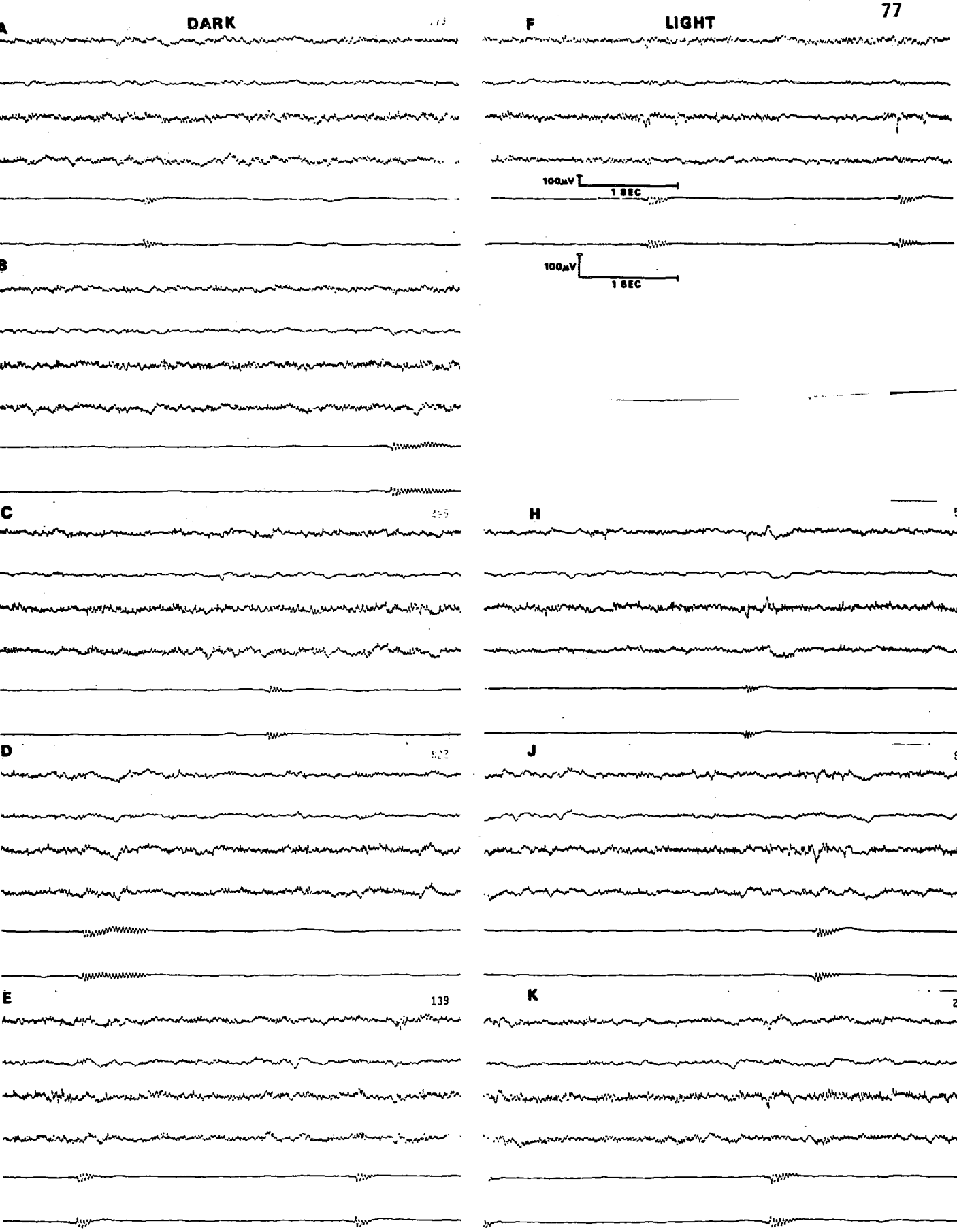
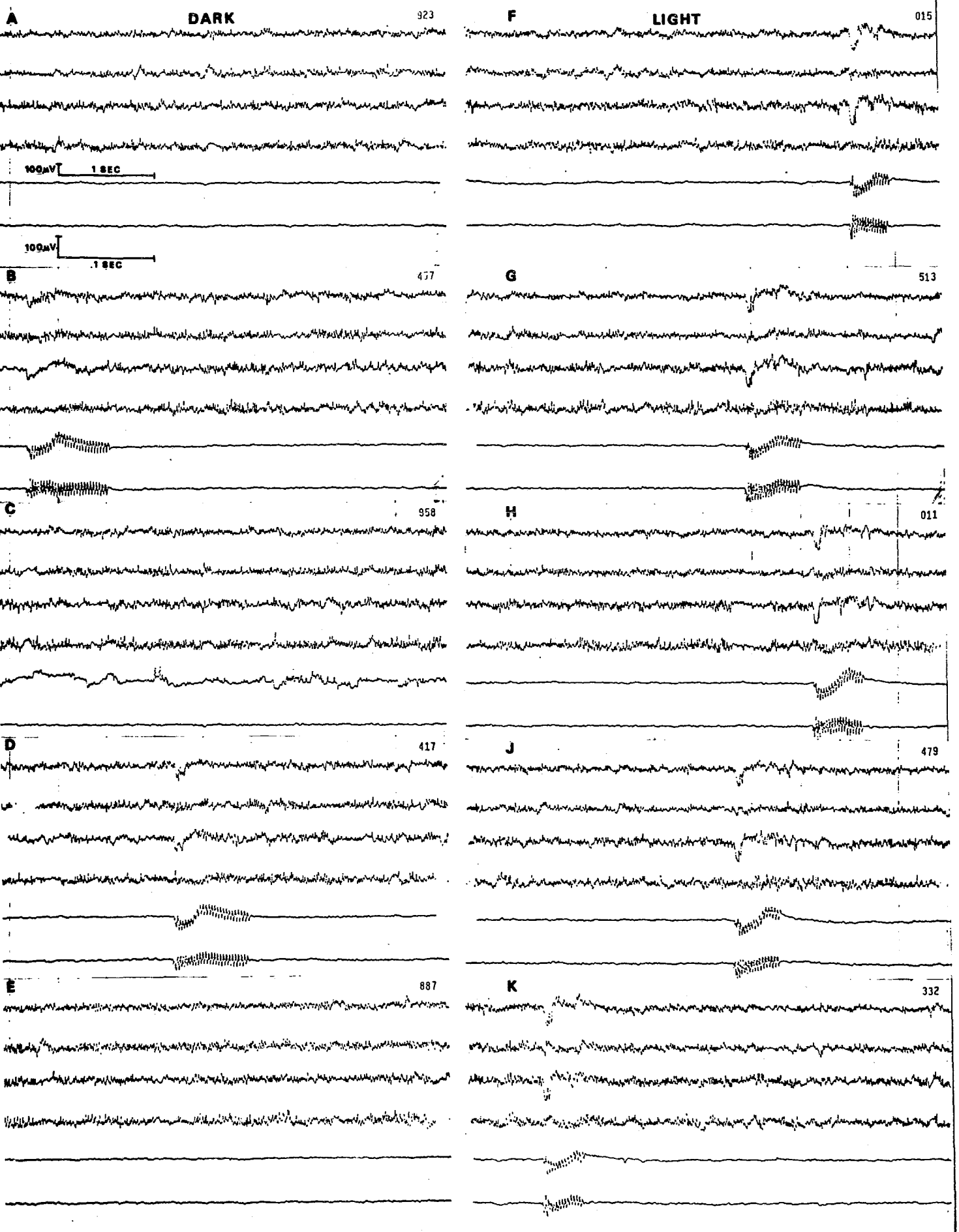

A
DARK
F
LIGHT
100μV
1 SEC
100μV
1 SEC
B
C
H
D
J
E
K

FIGURE 24.

EEG samples from bird 85.

A. and F. PRE 4. B. and G. 4DEPI. C. and H. 7DEPI. D. and J. 4POSTI. E. and K. 7POSTI.

Each group of six channels is ordered as it is in Fig. 23. The upper two samples in B, C, G, and H were recorded from structures which were contralaterally deprived of patterned input. This bird was the only one to show large eyeblink-evoked responses in the dark.


A
DARK
923
100μV
1 SEC
100μV
1 SEC
B
477
C
958
D
417
E
887
F
LIGHT
015
G
513
H
011
J
479
K
332

DISCUSSION

The studies reported here found that deprivation of light input to one eye of a pigeon increases the amplitude and changes the wave-form of the visual evoked potential of both the contralateral optic tectum and nucleus rotundus, while increasing the amplitude of the ipsilateral rotundal response. When the occlusion procedure is repeated on the other eye, the responses of both the contralateral and ipsilateral tecta and rotundi change in shape, but only the structures contralateral to the covered eye show an amplitude increase. Monocular elimination of patterned input has no clear effect, though there is some suggestion that the amplitudes of the evoked potentials of the contralateral structures increase with pattern deprivation. Subsequent unilateral deprivation of light however, resulted in response amplitude increases and waveform changes in structures contralateral and ipsilateral to the covered eye. This suggests that there are persisting effects from previous deprivation.

It might be argued that the response changes found with total deprivation of light are not due to the restriction procedure but rather to the prolonged dark adaptation of the occluded eye, particularly since it is known that response amplitude increases with dark adaptation (Galifret, 1966). This is unlikely for two reasons. First, there were twenty minutes of dark adaptation for both occluded and nonoccluded eyes prior to testing. Second, because the optic fibers of the pigeon visual system are completely crossed the changes seen in the responses of the

structures ipsilateral to the covered eye cannot be accounted for by dark adaptation alone. Indeed, for this same reason it is unlikely that the changes in the responses are due solely to any direct type of peripheral effect in the visual system, since such an effect would alter only the contralateral, and not the ipsilateral, evoked potentials.

The changes in the responses ipsilateral to the covered eye represent the most unexpected findings in Experiment II and imply that the pigeon is not functionally "split-brained" but instead that there is considerable interdependence between the two lobes. Presumably the ipsilateral effect in the first light-deprivation procedure is due to altered input coming from the optic lobe that is contralateral to the deprived eye, most probably by means of the tectal commissures. Nevertheless, it is unclear why only the ipsilateral rotundus and not the tectum showed a response change with the first occlusion. The problem arises because there appears to be no input from the contralateral optic lobe to the nucleus rotundus which does not pass through the optic tectum (Ebbesson, 1970). Therefore, if the rotundal effect is due to altered input from the contralateral optic lobe such input should have an effect on the optic tectum also. Examination of the difference waveforms of Figure 16 indicates that there is some reason to believe that such an effect took place for the left tectum on sessions 4 DEP I and 7 DEP I, since the waveform for those sessions differ from the preceding and succeeding waveforms. However, the amount of difference appears to small to conclude definitely that an effect is present.

A related question asks why the ipsilateral effect was much more noticeable during the second deprivation procedure when both the tectum

and rotundus showed changes in response shape, rather than during the first procedure when the only change was an increase in rotundal response amplitude. Since each ipsilateral optic lobe in the second deprivation procedure had been subjected to previous contralateral deprivation, it may be that the prior contralateral procedure left residual effects in the lobe that re-emerged with subsequent ipsilateral deprivation. This line of reasoning suggests that because the ipsilateral response changes seen in the bird that was deprived of light after having been deprived of patterned input were similar to those seen in the two birds which were deprived of light during both occlusions, contralateral pattern deprivation may also have left residual effects which appeared with ipsilateral light deprivation. It might be suggested that the ipsilateral effects are due not to the occlusion but to changes in the general arousal state of the animal. Such changes in arousal would be expected to be accompanied by changes in the EEG -- a "flattening" for increased arousal, an increase in slow wave activity for a decrease in arousal (Schaub, 1963). Careful inspection of our EEG data, however, gave no indication of such events, nor did the behavior of the birds suggest altered arousal.

The increased evoked response amplitudes found in the present study suggest increased sensitivity of the nervous system to stimulation and resemble the findings of investigations on the effect of unilateral tactual deprivation on bilateral tactual sensitivity (Heron & Morrison, unpublished paper; Aftanas & Zubek, 1964). Researchers occluded a small area of the forearm with a plastic cup for seven days, and found an increase in the tactual acuity of the occluded area and of the homologous, non-occluded area on the contralateral arm, but no change on a non-homo-

logous, contralateral area. These results were interpreted in terms of a disuse supersensitivity model in which deafferentiation is produced by elimination of sensory input rather than by surgical intervention. Monocular deprivation of visual input in the pigeon may be producing changes in the nervous system similar to those seen in disuse supersensitivity.

Several studies could be carried out paralleling those which have examined hyperexcitability phenomena to investigate this possibility. One such approach might involve the administration of stimulating and depressing chemical agents to visually deprived animals. If supersensitivity effects occur with sensory deprivation it is expected that the reaction of the nervous system to these substances would be an exaggeration of its normal reaction. Another procedure would be to repeat the present study but record evoked responses at many points in time during deprivation to see if the increased sensitivity of the visual system is preceded by a period of depressed sensitivity as noted by Spiegel and Szekely (1955) in their study of the partially deafferented cortex.

SUMMARY

The research reported in this thesis found that:

1. The EEG of the pigeon optic tectum and nucleus rotundus is characterized by high-voltage fast activity of variable frequency intermixed with potentials of 2 to 5 Hz. There is little EEG synchrony between contralateral structures, though some synchrony is present between ipsilateral structures. EEG recordings made in the light and in the dark do no appear to differ.

2. The positive component of the evoked response of the pigeon decreases in amplitude as the frequency of stimulation is increased from 1 to 5 flashes per second. At 10/sec. however, the amplitude increases. The amplitude of the response's negative peak declines continuously over the same frequency range.

3. Deprivation of light input to one eye increases the amplitude and changes the waveform of the visual evoked potential of the contralateral optic tectum and nucleus rotundus and increases the amplitude of the response of the ipsilateral rotundus.

4. When the light deprivation is repeated, with the previously uncovered eye occluded, the evoked responses of both the contralateral and ipsilateral tecta and rotundi change in shape but only the contralateral structures show a clear response increase.

5. The effect of monocular deprivation of patterned visual input on the evoked response is unclear, though there is some suggestion that the amplitude of the potentials of the contralateral structures increase.

6. Recordings of the EEG made in the dark and in the light reveal no change related to either deprivation of light or of patterned input.

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