EFFECTS OF CORTICAL STIMULATION ON LEARNING

by

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INTRODUCTION

In 1932, Hess developed a technique whereby electrodes could be permanently implanted in the brains of intact, awake animals. The electrode mountings were small, caused no apparent discomfort, and allowed the animals complete freedom of movement. Although several other investigators independently devised their own techniques (Loucks, 1933; Chaffee and Light, 1934), subsequent work has shown Hess' method to be the most reliable (Delgado, 1955a). This development has enabled neurophysiologists and psychologists alike to stimulate the brains of relatively normal animals in a variety of behavioral situations. The present study involves the use of the technique as modified for rats by Olds and Milner (1954). The investigation is concerned with the effects of cortical stimulation delivered to the animal at various stages in the learning process.

Although a vast literature is available on the motor, autonomic, and electroencephalographic effects of electrical stimulation of the brain, relatively few studies have been concerned with the relationships between brain stimulation and learning. Even less numerous are studies dealing with the effects of cortical stimulation on learning, the topic with which we are concerned here. A better understanding of the problem may be had, however, if we first review the relevant experiments dealing with the reticular activating system, and the structures which make up the limbic system or rhinencephalon.

The Reticular Activating System

A number of reports have become available during the past 25 years demonstrating that lesions placed in the basal diencephalon and anterior midbrain frequently caused a state of sleep (Bremer, 1935, 1938; Nauta, 1946). Bremer concluded from his observations, that the sleep resulted from deafferentation of the cerebral cortex. Olfactory and visual connections remained uninterrupted in Bremer's preparations, however, so a complete sensory deafferentation was not produced. Furthermore, subsequent work (Lindsley, Bowden, and Magoun, 1949) has demonstrated that large basal diencephalic lesions which spare the sensory relay nuclei of the thalamus and sensory afferent pathways are very effective in producing E.E.G. changes characteristic of sleep. Thus it appears that the sleep which followed lesions of the brain stem in Bremer's preparations did not necessarily result from deafferentation but rather, as Lindsley, Bowden, and Magoun suggested, from the removal of a more specific facilitating influence from the reticular formation. Moruzzi and Magoun (1949) were able to demonstrate that in addition to the classical sensory pathways to the cortex, excitation is conducted to the cortex by way of a second parallel system in the core of the brain stem, a system which has since come to be called the reticular activating system. These results were enlarged upon later by Starzl, Taylor, and Magoun (1951) who showed that a major portion of sensory potentials were conducted to the frontal and sensory-motor regions. More recently, Magoun's group has shown that all sensory modalities send collaterals into the reticular activating system which serves as a secondary, non-specific sensory pathway (French, Verzeano, and Magoun, 1953).

With the accumulating interest in the reticular activating system during the past five years (see e.g. the review by Jasper, Gloor, and Milner, 1956), it is not surprising that attempts have recently been made to discover what effects stimulation of this system would have on learning. As yet only a few such studies have appeared in the literature; the results obtained in these pioneer efforts, however, and the many problems they suggest, should encourage other workers.

Milner (1954) first investigated the effects of stimulation of various portions of the reticular activating system on performance in a shuttle-box situation in which the rat could only avoid electrical shock to the feet by running to the opposite side of the box within a predetermined interval. The animals were trained to respond only after a certain period of time had elapsed and without the benefit of any external signal or stimulus. After the habit was acquired, and a stable response pattern established, shock to the feet was discontinued so that the effects of intracranial stimulation could be observed (i.e., a change of rate could occur without the animal's receiving punishment). Milner reported that brief bursts of electrical stimulation in the more rostral portions of the reticular activating system, prior to and during the response, caused a decrease in response rate. An increase in response was noted, however, when the electrodes were in the posterior tegmentum.

This work was soon followed by several other related investigations. Chiles (1954) trained six cats in a modified Skinner box to press a bar for food reward. After a plateau in performance had been attained, electrodes were implanted in the reticular activating system and a suitable time allowed for post-operative recovery. Following this, 48 additional days of testing were given. On alternate days the animals were given intermittent bursts of electrical stimulation during the interval for which they were in the test situation.

Chiles obtained two interesting results. First, a decrease in response rate occured when the rats were stimulated. Second, although response rate fell, the variance was significantly higher for the group on the days they were stimulated. Chiles suggests that diencephalic stimulation provides distracting sensory stimuli and thus impairs performance. He feels that extraneous responses are caused by these stimuli and produce a corresponding increase in variance.

The next two studies with which we are concerned here dealt with the disruptive effects on memory that electrical stimulation of the reticular activating system may cause. Glickman (1957) utilized the one-trial learning technique developed by Hudson (1950) in order to study the effects of stimulating the reticular activating system with respect to the consolidation of memory traces. Essentially, Hudson's procedure requires the rat to acquire an avoidance response in a single exposure to the test situation. Following a period of pretraining, a copper food tray is inserted through a striated pattern into the animal's living cage. After the rat has eaten for one minute, it receives a 250 volt condensor discharge, the copper tray serving as anode. In Glickman's procedure, the experimental group received a short burst of electrical stimulation to the reticular activating system at varying intervals following the shock. This stimulation, during the period supposedly required for the consolidation of memory traces (Duncan, 1949) produced deficits in avoidance behavior on a subsequent trial. The results are interpreted as meaning a failure of learning, the intracranial stimulation apparently interfering with the memory for the noxious event.

Mahut (1957a, 1957b) also investigated the effects of reticular stimulation upon consolidative processes in more complex behavioral situations. In the first experiment, the effects of stimulation upon the formation of a learning set in the closed-field test (Rabinovitch and Rosvold, 1951) were investigated. It was found that the animals which received stimulation after each trial made more errors than the control group. Further testing showed that the effects of stimulation were reversible, for when stimulation was discontinued the differences between the groups disappeared. This finding was extended by Mahut in a second study using conditional response learning on a simple T-maze. Here again the differences between the stimulated and control animals were significant, for the experimental group was responding no better than chance after all the members of the control group had met criterion. Electrical stimulation was then discontinued and the experimental rats reached criterion in the same number of trials as the control animals required to learn the problem originally. Finally, a third experiment was performed to investigate the effects of stimulation of the reticular activating system after a learning set had been acquired by the animal; retention of the previously learned skills were found not to be disturbed.

Mahut proposed the following hypothesis as an explanation for her data. If learning is presumed to be dependent upon a number of cortical neurones whose connections become well established through repeated excitation (Hebb, 1949), then stimulation of the reticular activating system may be considered to have a disrupting effect on the consolidation of these connections. The disruption of a given pattern of cortical firing, following reticular stimulation, would render some cells unavailable for participation in the necessary phase sequences. Once the connections among the cortical neurones are well established, however, reticular activating system stimulation becomes ineffective as an interfering influence.

Although Mahut's findings are in accord with other results (Milner, 1954; Chiles, 1954; Glickman, 1957), the present writer feels that the interpretation she offers is not in accord with current neurophysiological conceptions. Direct stimulation of the reticular activating system produces a desynchronized electroencephalogram (E.E.G.) resembling the pattern commonly associated with attention or "set" (Moruzzi and Magoun, 1949; Magoun, 1952, 1954; Ingram, 1952). Certain patterns of desynchronization, in turn, are interpreted as conditions favorable for learning (Hebb, 1949; Lindsley, 1951, 1957). Thus, Lindsley (1957) has suggested that stimulation of the reticular activating system renders the cortex more receptive to incoming messages, enhancing the speed and efficiency of learned responses. Mahut (1957b), then, postulates that electrical stimulation disrupts the learning process, while others suggest such stimulation would have a facilitating effect. Mahut's suggestions, however, are only tentative and other experiments along these lines are needed before more durable hypotheses can be offered.

It is interesting to note that although the intralaminar nuclei of the thalamus are usually considered to be part and parcel of the reticular activating system (Jasper, 1949; Nauta and Whitlock, 1954; Ellingson, 1956) several objections to this view have been raised (Magoun, 1954; Morison, 1954). Hebb (1955, 1958) has pointed out that certain semantic and operational difficulties frequently arise from the naming of "systems", and that the reticular activating system as a unitary entity, may turn out to be no more than a convenient fiction. Certainly Milner's study (1954) and the next investigation to be considered are striking examples of the different effects that can be obtained from stimulation of various parts of the reticular activating system.

Fuster (1957, 1958), working in Lindsley's laboratory, has shown that stimulation of the core of the brain stem at the level of the mesencephalon can improve performance on a certain task. Six rhesus monkeys with implanted electrodes were tested on a modification of the Wisconsin General Test Apparatus. In the test situation, the discriminanda were briefly illuminated by a tachistoscopic light, preceded by a burst of intracranial stimulation. Fuster interprets the improved performance which followed electrical stimulation as indicating that brain stem excitation facilitates cortical activity thus making the cortex more receptive to the arrival of sensory impulses. Although Fuster's results are at odds with other reports in this area, we have seen that several authors believe that reticular activating system stimulation should increase attentiveness and facilitate performance (Hebb, 1955; Lihdsley, 1951, 1957; Milner, 1957). To the best of the writer's knowledge, Fuster's experiments represent the first verification of this hypothesis. Yet the concept of an optimal level of excitation (Hebb and Thompson, 1954) included the postulate that over-arousal (i.e. beyond optimum) will interfere with adaptive behavior (Hebb, 1955). Milner, (1954), Chiles (1954), Glickman (1957), and Mahut (1957b) have made use of this view in interpreting their own results.

The problems and inconsistencies apparent in a comparison of these studies are puzzling indeed, although perhaps it is too early to expect a consistent picture to emerge from so few data. Rather than look to these early investigations as definitive works, they may better be regarded as studies suggestive of re-interpretations of the existing literature and as stimuli for further experiments. The interference with learned behavior reported by Milner (1954), Chiles (1954), Glickman (1957), and Mahut (1957a, 1957b) may be related to the arrest reactions reported by Hunter and Jasper (1949) and Grossman (1958). If such a relationship were present, an interpretation other than that usually offered could be made. Generally, the investigators under discussion in this section have attributed the deficits they observed to an impairment in conceptual processes. If a disorganization of motor behavior is caused by the stimulation, however, the analyses these investigators have offered appear incomplete. It is suggested here that learned behavior involves at least two related, but separate, central processes. On the one hand there are the "ideational" or conceptual processes. Frequantly, these are called cell-assemblies, memory traces, trace systems, S-S relationships, or more simply, meaning. (See Hilgard, 1956 or Osgood, 1953 for complete reviews of this topic). It also seems likely, however, that still other patterns of excitation become established centrally which are directly responsible for integrated motor movements; for the present let us call these "central motor processes." Given then, these two types of on-going central processes (with the possibility of additional processes of an associational nature), it may be possible to interfere with the central motor component - yet leaving the memory per se intact. None of the studies reviewed has systematically attempted to distinguish between the intellectual and motor components of the behavior they are studying. (The distinction, however, bears directly upon the present experiment and we shall return to this point in the discussion.)

It is possible that several factors contribute to the apparent discrepancy between these two findings, i.e. interference and facilitation. Differences in electrode locus within the reticular activating system could easily produce such contrasting effects, as Milner's (1954) study has shown. It is also reasonable to assume that the probability of observing an effect like facilitation is dependent - apart from electrode placement - on a fortuitous choice of both experimental task and stimulation frequency. Further studies assessing the effects of these variables are needed in order to correctly interpret the role of the reticular activating system in learning situations.

The Limbic System

Paralleling the recent development of interest in the reticular activating system, a growing number of investigations by neurophysiologists and psychologists have appeared dealing with rhinencephalic or limbic structures. In this section we will consider those studies in which portions of the limbic system are electrically stimulated, and the resulting effects on motivation and learning are observed. The definition of limbic systems which will be used here is the one outlined by Pribram and Kruger (1954) which includes the amygdaloid and septal nuclei, the hippocampus, the cingulate, entorhinal and frontotemporal cortex. In addition, the suggestion (Papez, 1937; Fulton, 1955; Olds 1955b) that parts of the hypothalamus be included functionally with limbic structures will be accepted.

Much of the recent work in this area has supported the views of Papez (1937, 1958) and MacLean (1949), that the limbic system is concerned in emotional behavior. Electrical stimulation of the hypothalamus (Cohen, Brown, and Brown, 1957) and of the points in the crus of the fornix, and the inferomedial portion of the hippocampal gyri (Delgado, Roberts, and Miller, 1954;

Delgado, 1955b; Delgado and Burstein, 1956; Delgado, Rosvold, and Looney, 1956; Miller, 1958) have elicited fear responses which in turn can serve as a basis for learning.

Taken together, the results of these studies are in opposition to the earlier work of Masserman (1941, 1942) who reported that hypothalamic stimulation was incapable of providing the motivation necessary for the acquisition of a conditioned response. As Cohen, Brown, and Brown (1957) have pointed out, however, the differences between Masserman's training techniques and those of later workers may account for some of the discrepancies which were noticed. A correct avoidance response in Masserman's experiments required the animal to jump to a small elevated platform; yet no pre-training period was given to allow for response selection. In contrast, by selecting an instrumental response appropriate to the arousal state produced by intracranial stimulation (Delgado, Roberts, and Miller, 1954; Cohen, Brown, and Brown, 1957), it was demonstrated that the motivational properties of centrally produced emotionality could provide the basis for conditioning.

In addition to the literature on avoidance conditioning, data are now also available on the positive reinforcing effects of intracranial stimulation. Numerous reports from several laboratories (Olds and Milner, 1954;

Olds, 1955a, 1955b, 1956a, 1956b, 1958; Brady, 1956) have amply demonstrated the existence of this startling phenomenon. In general, the evidence supports the view that basomedial forebrain stimulation can act as a reward and create drive states not unlike those elicited by the more classical external manipulations (e.g. food deprivation). Extensive reviews of this literature are available (Sheer, 1957; Zeigler, 1957) with perhaps more theoretical speculation than the data warrant (Olds, 1955a, 1955b). The basic methodology, however, is important for the present investigation and is similar to that employed in several of the studies included in this review.

Apart from these studies on "reward systems", there exists but one systematic investigation of the effects of limbic system stimulation on learning. In a study similar in design to that employed by the present writer, Correll (1957) investigated the effects of bilateral hippocampal stimulation on the acquisition and extinction of an instrumental response. In his study, cats were trained to run a short alley and press a lever in order to obtain food reward. After each animal had received 42 trials (6 trials per day for 7 days), it was placed on an extinction schedule. No differences in response speed were apparent between the stimulated and non-stimulated groups during the acquisition training. The group of animals that was stimulated both in the acquisition and extinction trials, however, took significantly longer to extinguish than the group stimulated only during acquisition. Correll concludes that the greater resistance to extinction displayed by this group demonstrates that hippocampal stimulation increases "reaction potential" and thus acts as an increment in drive. He also suggests that hippocampal stimulation increases arousal level, possibly because of rhinencephalic connections with the reticular activating system (Sloan and Jasper, 1950; Green and Arduini, 1954), and thus increases drive level.

Assuming that Correll's suggestion is valid, one is led to wonder why he failed to observe the emotional behavior reported in previous studies in which the hippocampus was stimulated (e.g. Delgado, Roberts, and Miller, 1954). Correll himself has suggested two possibilities for this difference. First, his electrodes were located in more posteroventral portions of the hippocampal formation. This explanation, attributing different properties to various regions within the hippocampus, is supported anatomically by the evidence of Nauta (1956) and behaviorally in a recent study by Kimura (1958). Kimura, for example, showed that discrete bilateral lesions in the posterior hippocampus interfered with the acquisition and retention of an avoidance response; lesions in the anterior hippocampus did not interfere with such learning. Secondly, the intracranial stimulation used by Delgado, Roberts, and Miller (1954) was administered through monopolar electrodes. Such stimulation would involve a much larger region than Correll would have excited with his bipolar electrodes.

The wide range of behavioral alterations which follow surgical manipulation of limbic structures make it difficult to offer any general statements with regard to the functions of the system. Perhaps the participation in so many aspects of behavior is to be expected from a set of structures with such diverse connections. Yet in spite of the number of autonomic and somatic responses which are influenced by various components of the limbic system (Kaada, Pribram, and Epstein, 1949; Kaada, Andersen, and Jansen, 1954; Anand and Dua, 1955, 1956a, 1956b), removal of these components does not significantly interfere with the animal's homeostatic balance (Gloor, 1955). Perhaps the effects of stimulation in the studies we have reviewed in this section should be considered as enhancing the normal activity of limbic structures, enabling them to take the lead in the organization of complex somatomotor responses. Under normal conditions, the function of these structures is undoubtedly more subtle.

One restraining influence on the present writer, which prevents excessive speculation on the role of limbic structures in motivation and learning, is a consideration of the older literature on the participation of the cerebral cortex in these complex functions (Bard, 1934; Bard and Mountcastle, 1948; Morgan and Stellar, 1950; Young, 1943). Thus, in 1950, Morgan and Stellar urged that emotional responses are not subcortically mediated, and are dependent upon the cortex. Four years later, however, one of these authors (Stellar, 1954) felt compelled to conclude that subcortial centers <u>are</u> responsible for the control of basic motivation. More recently, it has been suggested (Lindsley, 1951, 1957; Hebb, 1955) that levels of motivation are related to the neuronal activity of the reticular activating system. Frequently, several older issues may appear to be resolved by a new theoretical viewpoint, but more often a fresh approach points to a whole set of different problems. The importance of these conceptualizations, then, is in the interest they generate and in the experiments they suggest. Although the data already available appear too voluminous to grasp, perhaps more directed efforts along new lines will enable us to be more precise about the mechanisms responsible for behavior.

The Cortex

It is heartening to find, in the midst of learned meetings devoted to subcortical functions, that an occasional voice is raised reiterating the importance of the cerebral cortex for integrated behavior. Lashley (1954, p.434), having just concluded an eloquent plea for the significance of the cerebral cortex in behavior processes, asks : "What role, if any, can the reticular system play in the complicated dynamic functions of perception?" Bremer (1954), at the same meetings, pointed out that all wakeful behavior was dependent upon a continuous "facilitatory dynamogenesis" of cortical and diencephalic neuronal networks. Yet in recent years comparatively little has been done concerning the effects of cortical stimulation on learning. This is surprising when one considers the vast earlier literature on cortical extirpations - *a priori*, one might expect cortical stimulation to be an equally valuable complementary method - and the fact that the first studies of the effects of intracranial stimulation on learning did concern the cortex (Loucks, 1935). With implanted electrodes, however, it is just as easy to get at subcortical structures, and in the large number of current studies using the method almost all the attention has been directed away from the cortex.

Historically, the studies of Loucks (1935, 1938; Loucks and Gantt, 1938), originating in the Pavlovian Laboratory at Johns Hopkins, were the first and best known investigations of this sort. In an attempt to define the functional components of the conditioning mechanism, Loucks developed his own technique for the electrical stimulation of the central nervous sytem in intact unanesthetized animals (Loucks, 1933). Although this method still remains widely discussed (Zeigler, 1957), Clark and Ward (1937) have conclusively demonstrated that extraneous sensory stimuli were created by the electrical stimulation (e.g. audible buzzing). Since the core of Loucks' work depended upon intracranial stimulation for the production of conditioned (CS) or unconditioned (US) stimuli, the artifacts inherent in the earlier method render it unsatisfactory as a basis for theoretical discussion.

Several recent investigations (Loucks, 1955, in press; Doty, Rutledge, and Larson, 1956; Rutledge and Doty, 1955), however, have clearly demonstrated that true cortical conditioning is possible. Two interesting results in connection with these studies should be mentioned. First, the integrity of a conditioned response (CR) to a cortical CS appears to be dependent upon the columnar (or vertical) aspect of cortical tissue rather than the tissue adjacent to the electrode (Loucks, 1955). Second, ease of establishment of a CS-CR relationship is dependent upon the nearness of the electrodes to the midline, that is, the median longitudinal fissure (Doty *et al*., 1956).

In addition to these experiments using electrical stimulation of the cortex as a CS, a series of papers have appeared in recent years by Gengerelli and his coworkers studying the effects of cortical stimulation on maze performance (Gengerelli and Cullen, 1954), and on the learning of a black-white discrimination (Gengerelli and Cullen, 1955; Gengerelli and Mower, 1956). Gengerelli interprets the data in terms of a model he formulated earlier (Gengerelli, 1934). Essentially, the premises are that the rate of neuronal firing and the duration of the volleys are fundamental variables in the learning process. Thus, in Gengerelli's studies the differential effects of two rates of brain stimulation (75 per second and 300 per second), with other stimulus parameters held constant, are investigated; the general prediction is that the higher rate of stimulation will facilitate learning. Although several interesting possibilities are suggested by these experiments, serious flaws in the procedures and design make them difficult to interpret.

Milner (1954), for example, used a high frequency generator similar to Gengerelli's (1948, 1951) and found that serious distortions of signal strength occurred with the movements of the animals or even movements of other objects in the room. Tests in which the positive or aversive effects of electrical stimulation could be determined are also conspicuously absent in Gengerelli's reports. Perhaps the most notable lack, however, is the absence of histological data. None of the reports provide information as to the exact locus of stimulation.

Rosvold and Delgado (1956), in an experiment designed to investigate some of the consequences of prefrontal lobotomy, stimulated portions of the frontal lobes of monkeys during delayed-alternation and visual discrimination testing. Although performance on the visual discrimination task was not impaired by stimulation, rather striking impairments, similar to those which follow prefrontal lobotomy, were observed in the delayed-alternation task. While electrical stimulation of the white substance of the frontal lobe, the medial surface of the cortex, and the orbital surface did not impair performance, those animals with electrodes in the caudate were incapable of performing better than chance on the delayed-alternation task while being stimulated. Prior to the introduction of stimulation, the performance of these animals was virtually perfect.

It should be noted that a frequent consequence of caudate stimulation is temporary arrest (Smith, 1945; Heath and Hodes, 1954; Heath, Leach, et al., 1954; Forman and Ward, 1957). Rosvold and Delgado (1956) also observed a decrement in activity to result from stimulation of the caudate; in fact, the most severe deficits in performance were observed in the animals in which activity was decreased. Although they attempt to integrate their data with the literature on prefrontal lobotomy, Rosvold and Delgado (p.371) conclude that the data on the effects of cortical stimulation are too sparse to attempt an explanation in terms of "...any one process such as excitation, desynchrony, inhibition or absence of activity..."

Along with these animal studies dealing with cortical stimulation, a number of neurosurgical observations are also relevant to the present study. In addition to the production of motor movements and sensations by stimulation of portions of the human cerebrum (see e.g. Rasmussen and Penfield, 1947, 1948), activation and interference of more complex processes have also been noted. The application of weak electrical current to Broca's area or to parieto-temporal cortex, for example, may interrupt or distort the flow of speech (Penfield, 1952; Penfield and Rasmussen, 1950). Electrical stimulation of the anterior and inferior temporal lobe frequently elicits specific recollections from the epileptic patient with temporal lobe lesions (Penfield, 1952, 1958; Bickford, Mulder, et al., 1958; Roberts, in press). Thus, stimulation of cell aggregates in temporal cortex can recreate, for the patient, phenomena which have a strong reminiscent quality.

Stimulation of points in the posterior part of the middle temporal gyrus has recently been observed to interfere with immediate and long-term recall (Bickford, Mulder, et al., 1958). The duration of amnesia thus produced was found to be dependent upon the strength of the stimulating current used and frequently outlasted the period of stimulation. As Bickford points out in the discussion of his paper, these results suggest that the amnesic response is due to localized interference--perhaps either through the activation of cortical neuronal assemblies which are incompatible with proper functioning or through an as yet unknown suppressor mechanism. In any case, the reticular activating system is probably only minimally involved in the phenomenon since the conscious state of the patient was not affected and he could converse sensibly during stimulation. Direct application of electrical current to the cortex, then, has been observed to interfere with memory and learning processes and it is to this topic that we now turn our attention.

The Aim of the Present Investigation

As we have seen from a review of the literature on intracranial stimulation, there is much which remains to be done in this field. The most striking deficiency, perhaps, is that so few workers have systematically explored the introduction of stimulation at different stages of the learning process. In order to correctly interpret the consequences of stimulation in learning situations, it is advisable to examine its effects early in learning, while the necessary neural trace systems are being established, as well as after a certain degree of habit strength has been attained.

The research to be presented consists of three related experiments; the first deals with the effects of cortical stimulation on the acquisition of a habit, the second with the effects of such stimulation when introduced after the habit has been acquired, and the third with the effects of stimulation on extinction of the habit.

METHOD

The subjects were one hundred adult male hooded rats from the Royal Victoria colony. All animals weighed between 195-225 grams at the time of operation.

The operations were performed under aseptic conditions using nembutal anesthesia (0.88cc/Kg.). The animal was placed in the Johnson-Krieg stereotaxic instrument (Krieg, 1946) modified with an electrode carrier, and a midline incision was made in the scalp. The skull was then exposed with the help of a small muscle retractor and the site for implantation chosen. A small hole was made in the skull with a dental drill and the electrode lowered into place. The electrodes themselves have been previously described (Olds and Milner, 1954; Olds, 1955b); essentially, they consisted of two strands of enameled nichrome wire 0.005 inches in diameter cemented into a lucite block. As only the cortex was to be stimulated, very short electrodes varying in length from 1.0 to 2.25 millimeters were used.

After completion of the testing schedule, the animals were killed with ether and immediately perfused with physiological saline followed by 10 per cent formalin. The brains were removed and, after fixing for several days frozen sections were cut at 40 micra and stained with cresyl violet to determine the depth and position of the electrode tips. In the majority of the cases, however, the electrode locus could be seen on the top of the cortex and the brain sections saved were only from this region.

Apparatus : a wooden Skinner box (11" x 9" x 5") having a wire mesh top and equipped with a Gerbrands food delivery mechanism was used. The current used for stimulation was obtained from the 60-cycle power line, through a bell transformer, and adjustable between 0 and 12 volts r.m.s. A flexible lead, supported from the ceiling by elastic strands, was dropped through the wire mesh top of the Skinner box and clipped onto the plastic block containing the electrode. The circuit was designed so that when the bar was pressed two seconds of electrical stimulation, regulated by a Hunter timer, was given to the rat. Simultaneously, a pellet fell into the food cup and the press was registered on a counter. Further presses during the two-second interval did not activate either the food delivery mechanism or the stimulator.

The animals were first put into individual cages and placed on a 24-hour food deprivation schedule for 10 days prior to the formal testing session. After this, surgical operations were performed on the experimental and operate control animals of the acquisition study. The animals were allowed to recover for two or three days before they were tested. In the performance and extinction phases of the study, the operations were performed as indicated in Figure 4.

All animals that received stimulation were first tested outside the experimental situation in order to choose a voltage that did not cause motor movements. A plywood box (7" x 9" x 4") was used for this purpose. In general, with most of the cortical loci that were involved, the animals proved quite resistant to motor effects or seizures. The range of voltages that were used for all the animals in the study was from 1.5 to 8.0 volts (see Tables 1 and 2). This observation is in accord with the high cortical threshold (8.5 volts) to self-sustained after-discharge induced by electrical stimulation reported by Gangloff and Monnier (1957). Finally, a voltage once chosen was not varied during the course of the experiment.

EXPERIMENT 1: EFFECTS ON ACQUISITION

In the experiment dealing with the effects of cortical stimulation on the acquisition of the habit, 50 animals were used as subjects. Of this number, 20 served as normal controls. The remaining animals underwent the operative procedures described above. Of these 20 were in the experimental group; 10 served as operate controls and received no stimulation during the course of the experiment.

The animals were placed in the Skinner box for 15 minutes daily on 10 successive days without any previous adaptation to the apparatus. The subjects in the experimental and operate control groups had the flexible lead attached to their electrodes on all testing sessions. Each animal received a food pellet after each bar-press, but as the rats in the experimental group depressed the bar they also received, at the same time, a two-second burst of electrical stimulation (as described above).

Results

The operate and non-operate controls were combined in a single control group after it was found that their scores did not differ significantly. Furthermore, the experimental animals readily fell into two distinct sub-groups on the basis of response rate and electrode locus. All animals of the experimental group with electrodes in the cortex (N=12) had a very low rate of response, while those animals with electrodes in the white matter (N=8) behaved similarly to the control group. (This point is discussed at greater length in the next section.) The mean number of bar-presses per day made by the experimental group with cortical placements is shown in Figure 1. Although the response rates for the two groups did not differ significantly on day 1, the stimulated animals made fewer responses than the controls on the remaining test sessions; this difference increased from day to day and was significant at the .01 level on each of the last seven days (Mann-Whitney U-Test: Mosteller and Bush, 1954).

Early in the experiment it was observed that the stimulated animals frequently did not eat all the pellets they earned, which is of course quite unlike the behavior of normal animals in such a situation. Records were then kept of the number of pellets left in the food cup after each 15 minute session. These data are shown in Figure 2. The number of pellets left is expressed as a percentage of the total number obtained in each 15 minute session. Again a Mann-Whitney U-Test was used to assess the differences between the scores of the two groups. The difference between the groups, based on the combined scores of each animal for the first three and the last three days, was significant at the .001 and the .01 levels respectively. Thus the stimulated animals press less, yet leave more pellets than the control animals.

Histological Results

Because of the short length of the electrodes, it was occassionally difficult to arrive at a precise determination of the site of implantation. For example, nicks in the cortex, inflicted during the removal of the skull, were difficult to differentiate in some cases from the electrode track. Drawings were made at the time of operation based on the coordinates used for the implantation and rechecked on removal of the brain; these were very helpful in determining the electrode locus. Figure 3 shows the electrode loci for all the experimental animals.

In 15 of the 20 animals it was possible to do complete histology and accurately verify the electrode locus. Table 1 lists the animals for which this was done and their mean response rates. In addition, the cortical area (Krieg, 1946) in which the electrode most clearly appears and its depth (presented in terms of cortical layers) are given. Three of the 15 animals (2, 5, and 6) were found to have small infections which extended from the region of the electrode to the hippocampus and were not included in any of the statistical analyses. Fourteen of the rats had electrodes in the cingulate (Krieg's areas 23, 29c), two in the occipital (area 18), and four in the frontal region (areas 4, 6, and 10). There was not any relationship, however, between the cortical area in which the electrode was situated and the effects of stimulation on acquisition.

The depth of the electrode, on the other hand, did appear to be a significant variable. Five of the electrodes (rats 1, 3, 4, 7, and 8) had passed through the cortex and touched the corpus callosum. Interestingly enough, none of these animals showed impairment from the stimulation and the group has a slightly higher response rate than that of the control animals (see Table 1). Due to the small number of animals with electrodes in the corpus callosum, no really definite statements can be made. It does appear, however, that stimulation of the corpus callosum exerts a facilitating effect on performance and we shall return to this question in the discussion.

Table 1

Response Rates for the Experimental Animals in the Acquisition Study with the Voltages Used and Histological Data.

Rat	Cortical Layer	Cortical Area	Mean Response Rate	Volts
1	cc	29c	33.0	2.5
2	cc	29c	73.7	4.0
3	cc	29c	49.4	4.0
4	cc	29c	39.7	5.0
5	cc	29c	62.1	4.0
6	cc	29c	35.1	6.0
7	cc	18	15.3	8.0
8	cc	18	60.1	2.5
9	3	29c	7.5	2.5
10	4 *	29c	6.4	2.5
11	2	23	9.4	2.5
12	2 *	10	1.9	1.5
13	3	10	0.6	1.5
14	3 *	29c	1.5	2.5
15	4	29c	3.3	2.5
16	4 *	29c	2.3	4.5
17	4	29c	3.0	5.0
18	4 *	23	7.4	4.0
19	3	10	4.0	3.0
20	3	6	5.4	1.5

cc = corpus callosum

* = locus approximated

Test for Affective Quality of Stimulation

The results clearly show that stimulation can depress the rate of response. But the possibility exists that this depression in response was due to painful effects resulting from cortical stimulation. Though Olds (1956a) has recently mapped out some of the areas of the rat brain with regard to the reinforcing effects of stimulation, data on the effects of cortical stimulation are not available.

Although it was not obvious from the general behavior of the experimental animals that stimulation had painful effects, it was considered advisable to test this possibility. Eighteen sated normal animals of the same weight and stock were tested for 10 days in the Skinner box to obtain their operant levels. As can be seen in Figure 1, the mean response rate for the stimulated animals is not significantly different from that of the operant group. This is in agreement with earlier experiments (Burns, 1957) which showed that stimulation of the occipital and frontal cortex is neutrally reinforcing.

EXPERIMENT 2: EFFECTS ON PERFORMANCE AND EXTINCTION

The results of the first experiment then, indicate that cortical stimulation has deleterious effects on habit acquisition. The second experiment was designed to find out whether it had any effect on performance after the habit is fairly well established, and any effects on the extinction of the habit.

Procedure

Thirty-four animals completed the experimental procedures outlined in Figures 4 and 5. After being on a 24 hour feeding schedule for 10 days, each rat was placed in the Skinner box for 15 minutes a day on ten successive days (Stage I). Electrodes were then implanted in all the animals of the experimental group (Stage II). Post-operative recovery for all experimental animals was excellent and no ill effects from the operation were noted. After the four-day recovery period, the animals again spent 15 minutes a day for the next six days in the Skinner box (Stage III). During this period, the response rate returned to the preoperative level. For the next six days the experimental group received stimulation on alternate days, i.e. on days 2, 4, and 6 (Stage IV). As in the acquisition study, the stimulation was given for two-seconds each time the bar was pressed.

In all of the test sessions described so far, each bar-press delivered a food pellet to the animal. In the final phase of the study (Stage V in Fig. 5), the animals' responses were no longer rewarded with pellets. Thus the effects of stimulation on response rate during the extinction of the bar-pressing habit could be observed. Half of the experimental group (N=10), was stimulated on days 1, 3, and 5 of the extinction period (Fig. 5, Exp'tl Gp. "B"). The remaining experimental animals (Gp. "A") received no stimulation.

The surgical and histological procedures described earlier (as well as the apparatus) remained the same during this phase of the study. In addition to recording response rate, however, records were taken of the number of pellets left in the food cup, and the incidence of defecation and urination.

Results

The mean number of bar-presses per day for the two groups during the training trials are shown in Figure 4. The groups were closely matched and no significant differences between them were apparent during the first three stages of the experiment.

The effects of stimulation during the performance of a learned habit are shown in Figure 5. As described earlier, the experimental group was stimulated on days 2, 4, and 6 during Stage IV. A t-test for independent means was used to test the significance of the differences between the two groups. As in the acquisition experiment, the experimental animals made significantly fewer responses than the control animals on the days that they were stimulated (day 2, $p < .01$; day 4, $p < .05$; and day 6, $p < .05$). Further analysis showed that the experimental animals made significantly fewer responses ($p < .05$) on days 2, 4, and 6 than on days 1, 3, and 5 (Sign test: Mosteller and Bush, 1954). It should be noted, however, that only a reduction in response rate is observed and not the complete disappearance of the habit. No significant difference between the groups was observed on days 1, 3, and 5. Moreover, judging from the increase in response rate which the experimental rats showed on the days that they were not stimulated, it seems that the impairment produced by stimulation is reversible.

As in the first experiment, the rats in the experimental group continue to display the curious phenomenon of "pellet-leaving"; yet the effect was not as marked as in the acquisition study. On the first day of Stage IV (a day on which the experimental rats had not yet received stimulation), both groups of animals consumed all the pellets they obtained as can be seen in Figure 6. A marked difference was apparent on day 2 ($p < .005$, Mann-Whitney V-Test) the first day on which stimulation was given. The differences grew smaller on days 3 through 6 and were not statistically significant (see Fig. 6).

However, the experimental group left significantly more pellets ($p < .01$) on the days during which they were stimulated than on the days they were not, in addition to leaving significantly more pellets than the controls ($p < .05$). A comparison of the number of pellets left by both groups on days 1, 3, and 5 revealed that the experimental animals, even in the absence of electrical stimulation, continued to leave more pellets than the controls though this difference only approached significance ($p < .10$).

The results of the extinction study are shown in Figure 5. One of the more puzzling results is the disproportionate increase in response rate that occurs in group "A" on the first day of extinction. This difference in response rate cannot be due to any error in matching; the mean response rate for group "A" on the first six post-operative days (Stage III) was not significantly different from that of group "B" ($\bar{X}_A=75.6$, $\bar{X}_B=76.7$). Further, the means for the two groups on day 6 of Stage IV were not significantly different ($\bar{X}_A=67.9$, $\bar{X}_B=70.7$). Apart from day 1, on which group "A" had a higher response rate than both group "B" ($p < .03$) and the control animals ($p < .16$), the rates for the three groups are very similar during the remaining five days of the extinction period. Stimulation of the animals during this stage of the experiment has no apparent effect on the number of bar-presses; the rate of extinction of group "B" is similar to the rate of extinction of the control animals (Fig. 5).

A closer analysis of the data, however, revealed that stimulation was having differential effects on the members of group "B". Five of the animals did show the typical decrement in response rate for the days on which they were stimulated while the remaining five showed an increase in response rate on these days (Fig.7). The differences between their mean response rates for the extinction days on which they were stimulated were significant at the .05 level (Mann-Whitney U-Test). It can be seen from Figure 7 that the animals whose response rates were depressed on the days that they were stimulated (group "B-Low"), pressed significantly less than group "B-High" and also group "A" on days 1, 3, and 5 of Stage V.

The percentages of earned pellets that remained uneaten by groups "B-Low" and "B-High" during the performance phase (Stage IV) were put to statistical analysis to determine whether the two groups also differed in this respect. It was found that the animals in group "B-High" rarely left pellets, the differences between the two groups being significant at the .05 level (Mann-Whitney U-Test). Thus, the same animals that showed an increase in response rate on the days on which they were stimulated during extinction also differed from the other stimulated animals in that they failed to leave pellets during the performance phase of the study (Stage IV). Yet, there was no difference between these groups in the earlier phases (Stages III and IV) of the study.

None of the groups differed in incidence of defecation and urination at any time.

Histological Results

Histology was performed on the animals of the experimental group and the placements are shown in Figure 8. Table 2 gives a more detailed description of the anatomical data. As in the acquisition study (see Table 1), there was no correlation between the cortical area in which the electrode was situated and the effects of stimulation on response rate. The electrode of one animal (18) was far forward in the head of the caudate nucleus. Four animals (rats 1, 2, 3, and 4) had the tips of their electrodes in the corpus callosum. The remaining animals had electrodes which were clearly in the cortex. The three animals with electrodes in the callosum behaved in the same way as those of the acquisition study with callosal electrodes in that their response rate was not depressed on the days during which they were stimulated. Unfortunately, they were not members of group "B" during Stage V and therefore no data are available on the effects of callosal stimulation during extinction.

The five animals comprising group "B-High" were found to have electrodes in or close to cingulate cortex. Olds (1956a) has found electrical stimulation of this tissue to be positively reinforcing, and this may help explain the increase in response rate displayed by this group on days 1, 3, and 5 of Stage V (Fig. 9); it does not, however, explain why these rats should have a depressed response rate when stimulation was introduced in Stage IV (Fig. 7).

Table 2

Response Rates for the Experimental Animals in the Performance Study with the Voltages Used and the Histological Data.

Rat	Cortical Layer	Cortical Area	Mean Response Rate Non-Stimulated Days (1, 3, 5)	Mean Response Rate Stimulated Days (2, 4, 6)	Volts	Current
1	cc	4	49.6	62.6	5.5	Not Obtained
2	cc *	29c	103.6	122.3	4.5	" "
3	cc	29c	52.0	59.6	5.0	200 µA
4	cc	18	92.3	96.3	4.0	140 µA
5	3	10	59.3	37.6	2.0	Not Obtained
6	4	10	26.0	15.3	3.5	" "
7	3	10	65.0	64.6	4.0	" "
8	5	10	63.0	60.3	6.0	" "
9	2	10	59.6	54.6	6.0	" "
10	2	10	67.0	35.3	2.0	" "
11	3	29c	102.6	80.3	5.0	" "
12	4	4	54.3	39.3	2.5	" "
13	5	29c	140.3	105.3	5.0	" "
14	4 *	29c	106.6	65.3	5.0	" "
15	3	29c	76.3	73.3	2.5	" "
16	5	18	81.3	66.3	5.0	200 µA
17	3	18	78.0	68.0	5.0	175 µA
18	cn	10	67.6	21.6	5.0	200 µA
19	3	10	100.3	47.3	5.0	200 µA
20	3	10	68.6	46.6	4.0	160 µA

cn = caudate nucleus

cc = corpus callosum

* = locus approximated

DISCUSSION

What sort of a general picture, then, can one obtain from the results of the experiments just presented? First, during the acquisition of the bar-pressing habit, and after the habit has been acquired, cortical stimulation depresses the animal's rate of response. This finding is similar to the reports of other workers (Milner, 1954; Chiles, 1954; Mahut, 1957) obtained with stimulation of subcortical systems with diffuse cortical connections. In contrast, the effects observed here were obtained with stimulation of discrete areas of the cerebral cortex. The second finding is that coincident with this reduction in response rate, the animals failed to eat all the pellets they received (Figures 2 and 6).

We may consider the experimental animals as operating on a partial reinforcement schedule. Certainly the fact that a proportion of their responses was unrewarded is obvious from the number of pellets left uneaten in the food cup. In fact, in the first two days of the acquisition study, the ratio was of the order of 25 rewards per 100 presses. In the second experiment, when stimulation was introduced after the habit had been acquired (Stage IV), the ratio was approximately 90:100. Both these conditions meet the definition of partial reinforcement suggested by Jenkens and Stanley (1950, p. 194):

"...reinforcement given at least once but omitted on one or more of the trials or after one or more of the responses in a series."

At first glance, the rate of learning for the experimental group in the acquisition study favors partial reinforcement as the most parsimonious explanation of the results of this study. Numerous investigations have shown that response strength builds up more slowly under various schedules of partial reinforcement than under 100 per cent reinforcement (Humphreys, 1939; Finger, 1942a, 1942b; Sheffield and Temmer, 1950; Dufort and Kimble, 1956; Lewis, 1956). These differences in rates of learning between continuously and partially reinforced groups, however, were never so great as those observed in the present study (Fig. 1). Moreover, with training, partially reinforced animals <u>do</u> learn; the experimental animals in the acquisition phase of the present experiment did not.

The partial reinforcement analysis could, perhaps, be applied to the extinction data. Comparisons of response rate (e.g. between group A and the controls, Figs. 5 and 7) are difficult, however, since the habit strength of the groups varied at the onset of extinction procedures. Thus, since response-persistence in the absence of reinforcement is related to pre-extinction habit strength (Sidman, 1955) it is difficult to make a precise analysis. Group A (Fig. 7) may be displaying greater resistance to extinction as a function of partial reinforcement during Stage IV. The acquisition data, however, make it impossible to assume that the response rate of the experimental animals was due to partial reinforcement. It would therefore be unreasonable to suggest one hypothesis for the latter phase of the research and yet another for the earlier phase. Especially when cortical stimulation produces the same deficit in both experiments: a depression of response rate and a tendency to leave pellets.

What alternative interpretations can be offered to account for these results? One could argue, for example, that since the reduction in response rate was accompanied by a failure to eat all the pellets which were obtained, the retarded rate of learning of the stimulated animals was due to a loss of drive. Simply stated, such a view suggests that cortical stimulation renders the animals less hungry. If the effect of the stimulation is to reduce drive, however, it is hard to see why the experimental animals in the performance phase of the study (Stage IV) left so few pellets. It seems clear now that any interpretation of motivational states must include the existence of organized central processes which initiate, direct, and sustain behavior (Hebb, 1949; Lindsley, 1957). Viewed in such a light, an interference with the central "motor processes" mentioned earlier which are directly concerned with response sequences would interfere with the persistence and direction which are typical of motivated behavior.

One could also argue that the degree to which a habit is susceptible to interference is dependent on the level of organization that the trace system has achieved. Thus, during the acquisition of the habit, the central processes which subserve learned behavior may be less resistant to interference from cortical stimulation than they are after a certain level of habit strength has been attained. It is difficult, however, to understand why the physiological effects of cortical stimulation should vary with the degree of habit strength when the data from the second experiment indicate that a significant impairment occurred during stimulation even after the habit was well established.

This, then, brings us to the hypothesis favored by the present writer as an interpretation of the results. Presumably, a number of neural trace systems, some predominantly perceptual and others predominantly motor, are involved in the acquisition and performance of learned behavior. A complex interplay of such trace systems must precede the occurrence of correct response patterns in the Skinner box, a sequence of patterns which are much more involved than at first appears (Denny and Adelman, 1955). A chain of responses becomes established which results in obtaining <u>and</u> eating a pellet.

The hypothesis being offered proposes that the interfering effects of cortical stimulation observed in the present study are in the motor rather than the perceptual phase sequences. Although the data from the acquisition study give no definitive answer as to the nature of the deficit (all we can say is that the animals did not acquire the habit), this interpretation gains support from the findings of the performance and extinction studies. When stimulation is introduced after the habit has been acquired, a reduction in response rate occurs. But this only happens on the days during which the animals are stimulated. On the alternate days, the response rate for the experimental group is not significantly different from that of the control animals.

Similarly, the data obtained from the animals stimulated in both performance and extinction (Stages IV and V, Fig. 5) support this hypothesis. After those animals for which cortical stimulation appeared to have a reinforcing effect are removed from the sample (group "B-High"), the response rate for the remaining five animals (group "B-Low", Fig. 7) is seen to fall on the days during which they are stimulated. On the alternate days during which they are not stimulated, however, the response rate is very similar to that of the controls. Whether one takes the position that extinction represents the acquisition of a new response (Guthrie, 1935; Culler, 1938; Briggs, 1954; Johdai, 1955) or a Pavlovian kind of inhibition (c.f. Razran, 1939 or Hilgard and Marquis, 1940) is immaterial here. The effects of cortical stimulation during extinction appear to interfere only with the animal's performance while it is being stimulated and not with the rate of the extinction process.

Physiologically, it is not surprising that behavioral interference is so frequent a consequence of intracranial stimulation. The work of several investigators (Sholl, 1956; Olds, 1958) has made clear that stimulation of the cerebrum undoubtedly fires cells belonging to different functional groups, which under normal physiological conditions are never simultaneously active. These considerations force the conclusion that the experimental conditions used in the majority of the experiments described do not produce even an approximation to the asynchronous and selective firing characteristic of normal neuronal activity (Hebb, 1954).

Yet, facilitating effects have been observed to follow stimulation of certain subcortical areas (Milner, 1954; Fuster, 1957, 1958). In the present experiment there was some indication that the animals with electrodes in the corpus callosum were performing better than those with electrodes in the cortex. These differences, however, did not reach an acceptable level of statistical significance.

Mogenson (1958), though, has also observed a similar trend towards improved performance to occur in his animals which had electrodes in the corpus callosum. In his study, periods of electrical stimulation administered several hours before testing in a complex maze situation resulted in slightly faster running times and fewer errors.

While electrical stimulation of the cortical grey matter probably produces a refractory state in the area surrounding the electrode (Burns, 1957), stimulation of the corpus callosum may be thought of as producing effects which more closely resemble normal neuronal firing. Stimulation of a commissural system like the corpus callosum which has diffuse cortical connections may be thought of as having "second order" effects, i.e. a number of neurones in the network are (i) excited directly by the electrical current, and (ii) then carry trains of impulses to large and separate areas of the cortex. Hebb (1955, 1958), Lindsley (1957), and Milner (1957) have considered the nonspecific cortical bombardment provided by the reticular activating system as exerting a facilitatory effect on the cortex; perhaps the corpus callosum plays a similar role.

In closing, perhaps a parallel can be seen between the interference with behavior which was observed in the present study, and the interference with speech and thought processes which are frequently observed by neurosurgeons (Bickford, Mulder, et al., 1958; Penfield, 1952, 1958; Roberts, in press). Although again, several interpretations of these phenomena can be offered, it would appear that stimulation of the cortex affects a complicated neuronal network and interferes with the temporal integration of central processes which are required for organized behavior.

SUMMARY

(i) Electrical stimulation of the cortex delivered during the acquisition of a simple habit (bar-pressing for food in a Skinner box) severely depressed response rate and interfered with consummatory processes.

(ii) Stimulation of the cortex after the habit had been acquired also depressed response rate but only on the days during which stimulation was administered; the interference with consummatory behavior was much less marked during this stage of the learning process.

(iii) A similar reduction in response rate as a result of cortical stimulation was also observed during extinction of the habit.

(iv) Animals with electrodes in the corpus callosum displayed a tendency towards improved performance under stimulation conditions.

(v) The interpretation of the results suggests that electrical stimulation affects the complex integration of cortical motor processes required for correct performance.

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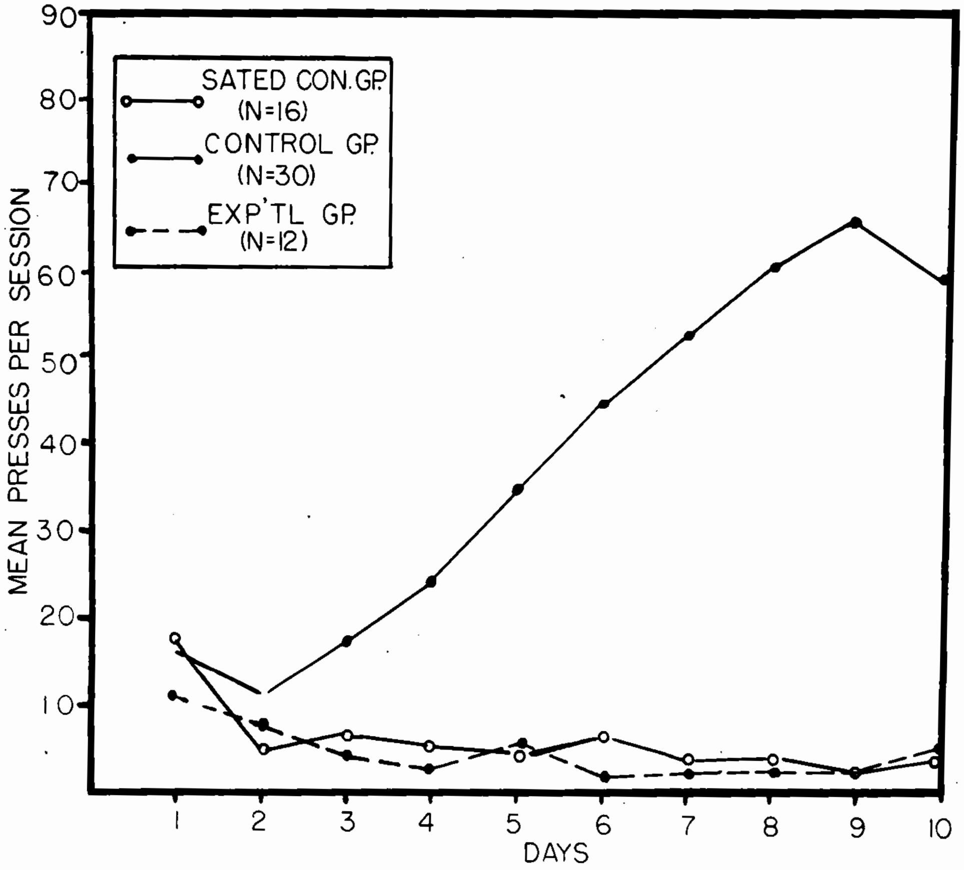

Figure 1. The mean number of responses made per day by each of the groups during acquisition.

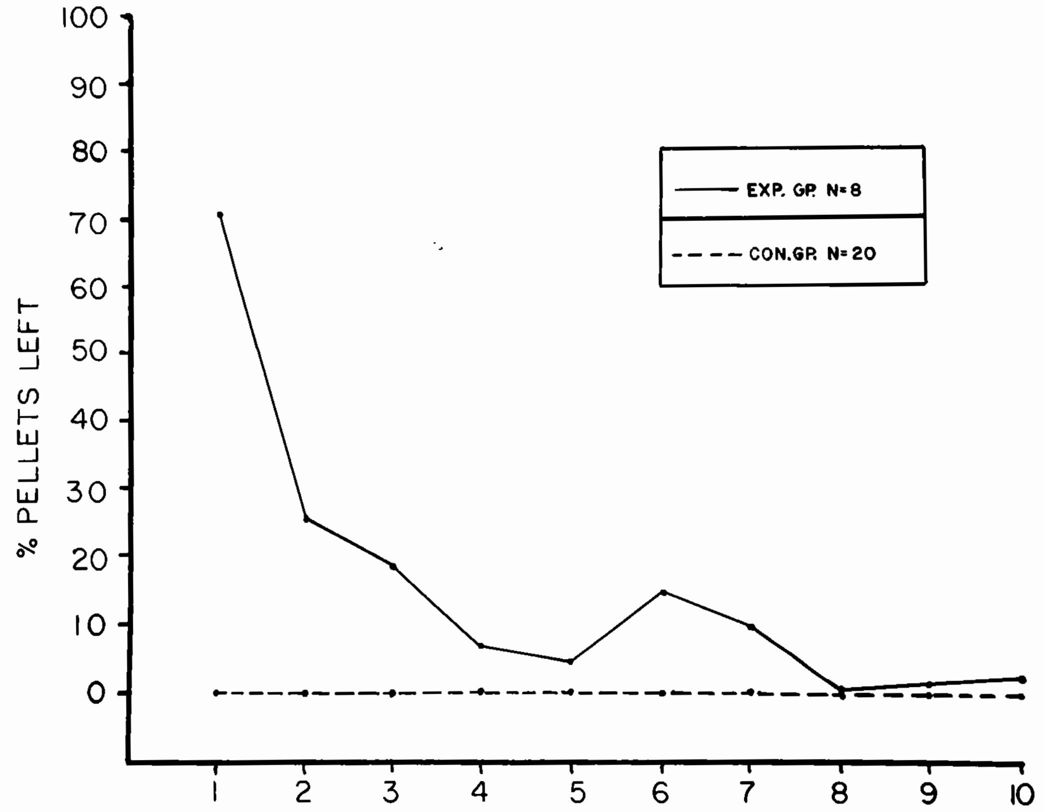

Figure 2. The percentage of pellets left on each of the testing days by the two groups. Note: the number of pellets left by the control group over the ten day period was 28 out of a total of 9,290 which were obtained (approx. 0,30%). This explains the comparatively flat curve drawn for this group.

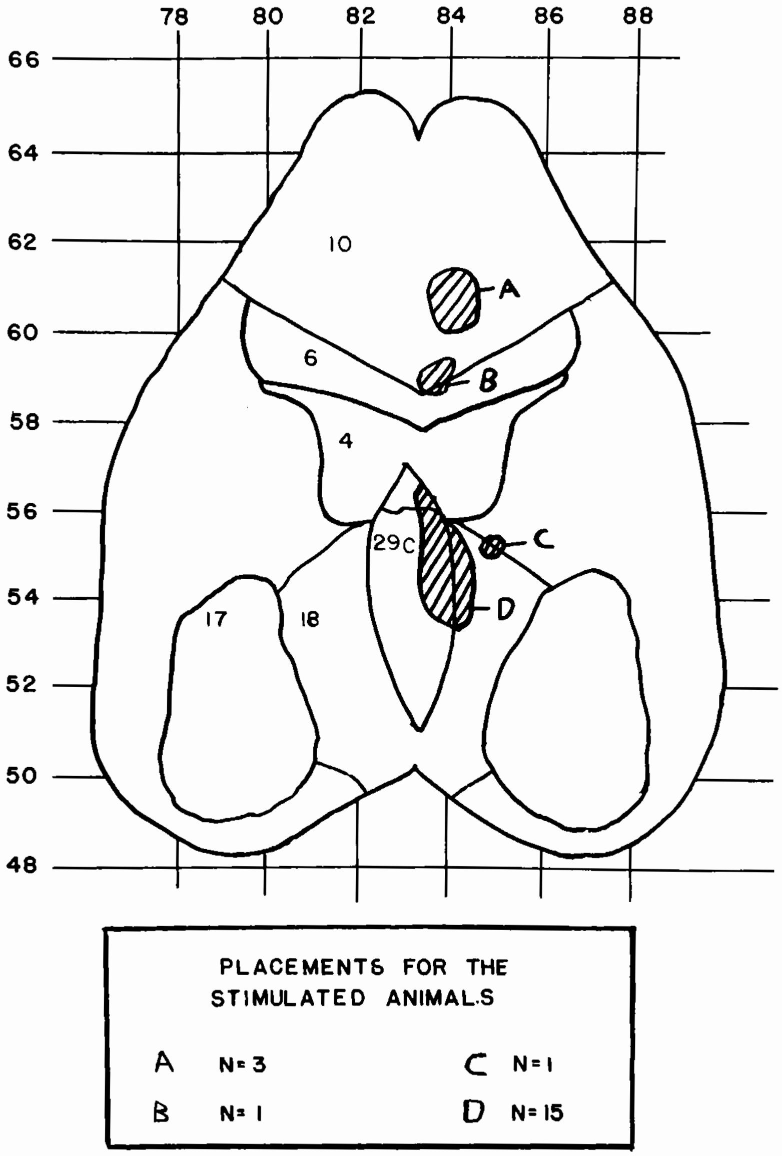

Figure 3. Electrode placements for the experimental group : Acquisition study.

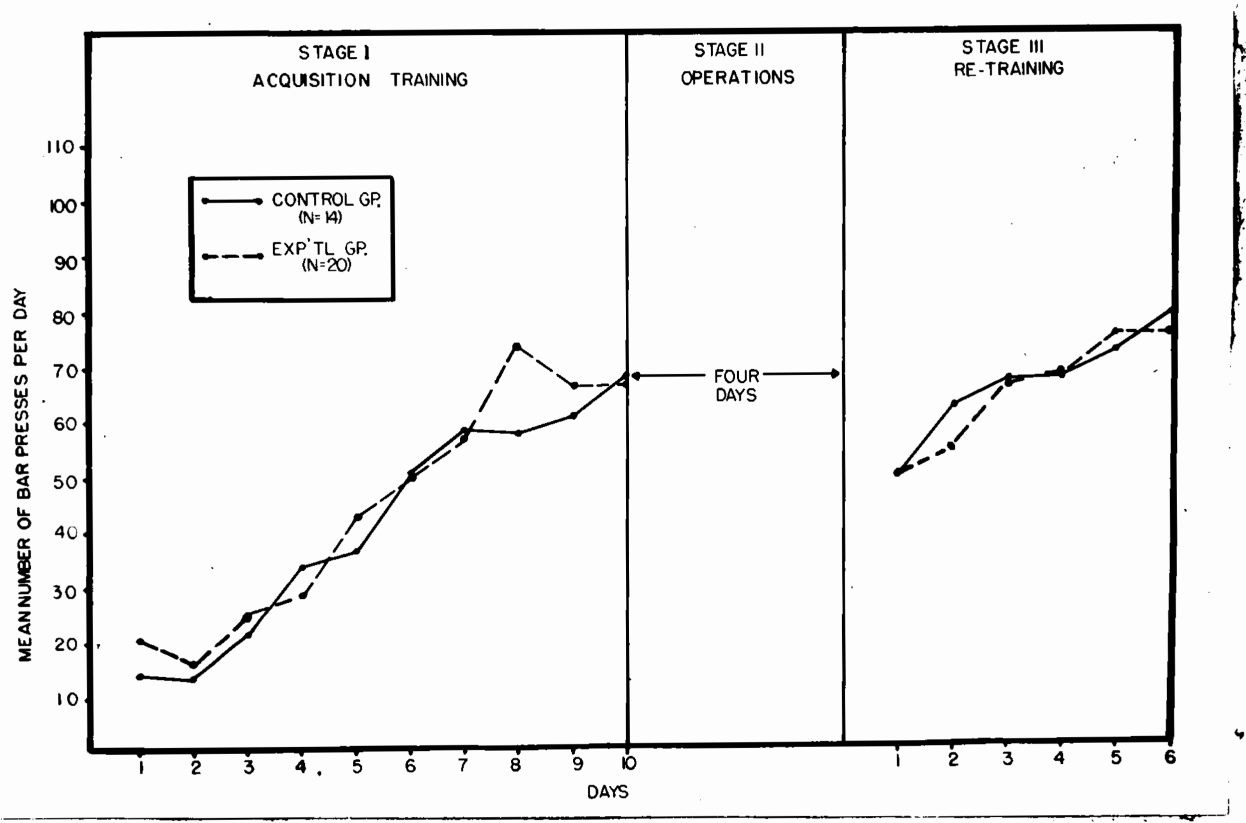

Figure 4. The mean number of responses per day made by the two groups during training and prior to the introduction of stimulation.

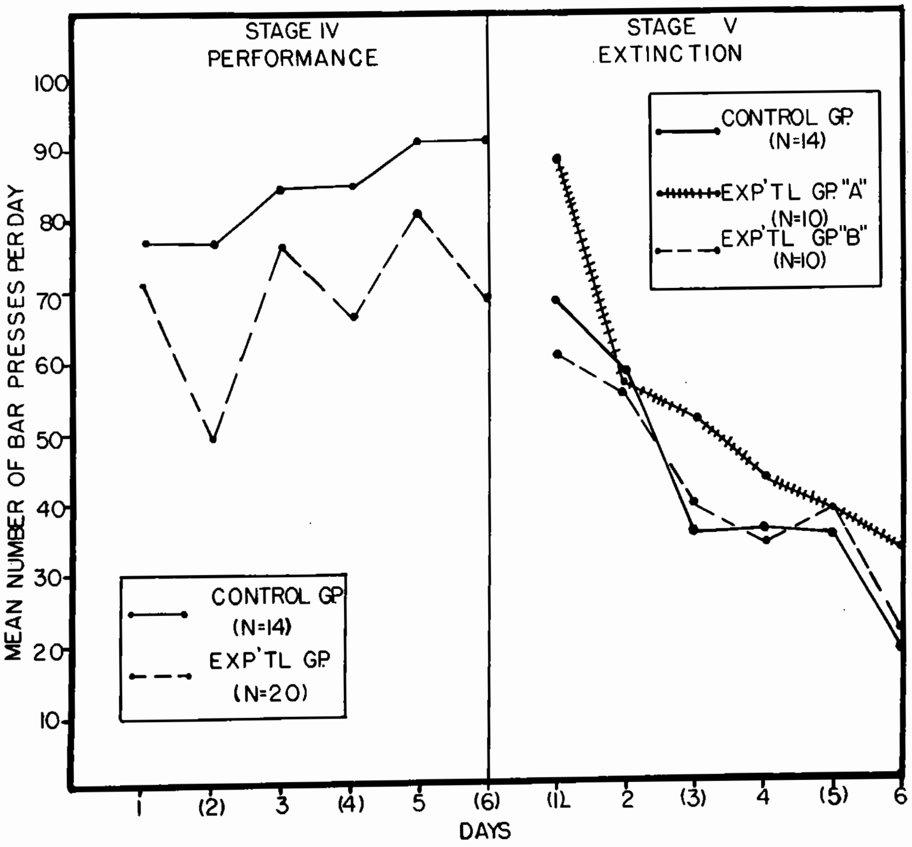

Figure 5. Response rate during performance and extinction with stimulation given on the days enclosed in parentheses. Group A, not stimulated during extinction; Group B, stimulated during extinction.

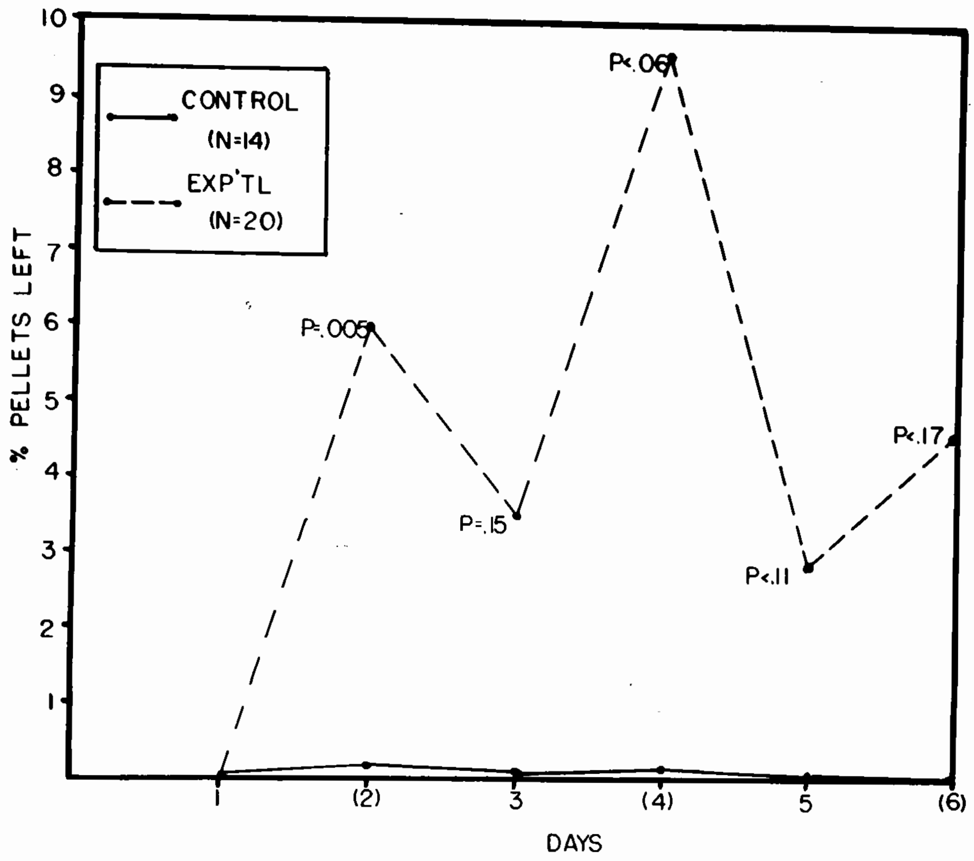

Figure 6. The percentage of pellets left by the two groups during performance (Stage IV, Figure 5); stimulation days in parentheses.

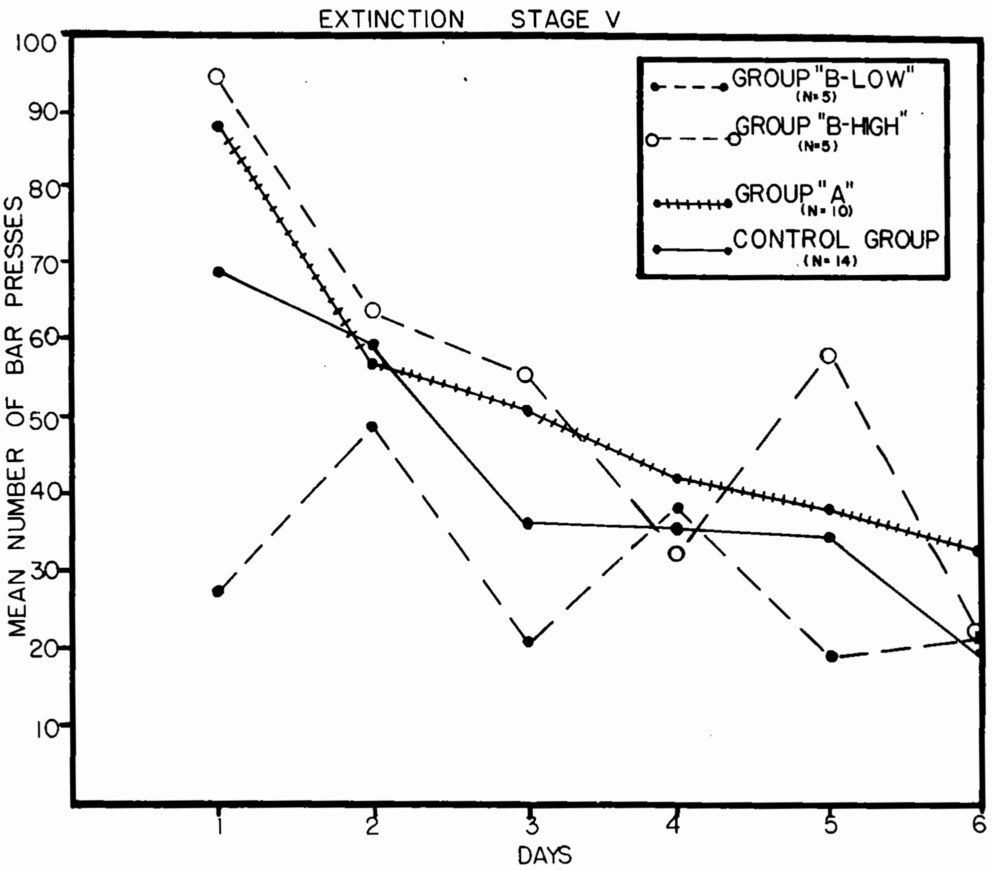

Figure 7. The mean number of responses per day made during extinction (Stage V, Figure 5).
Group "B-Low" - Animals with electrodes in the cortex.
Group "B-High" - Animals with electrodes in the cingulate.
Group A - Animals stimulated during Stage IV but not Stage V.
Control Group - Animals that were never stimulated.

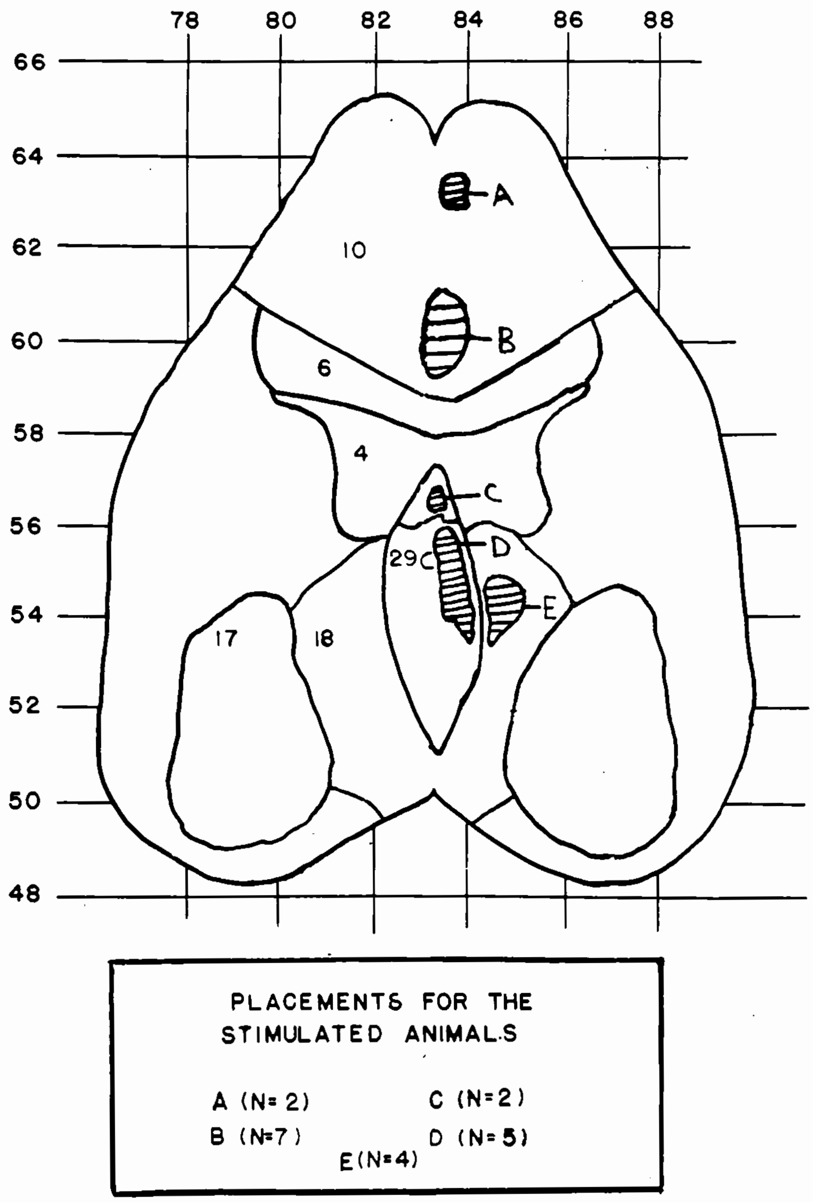

Figure 8. Electrode placements for the experimental group : Performance and extinction study.