INFORMATION TO USERS

This material was produced from a microfilm copy of the original document. While the most advanced technological means to photograph and reproduce this document have been used, the quality is heavily dependent upon the quality of the original submitted.

The following explanation of techniques is provided to help you understand markings or patterns which may appear on this reproduction.

1. The sign or "target" for pages apparently lacking from the document photographed is "Missing Page(s)". If it was possible to obtain the missing page(s) or section, they are spliced into the film along with adjacent pages. This may have necessitated cutting thru an image and duplicating adjacent pages to insure you complete continuity.

2. When an image on the film is obliterated with a large round black mark, it is an indication that the photographer suspected that the copy may have moved during exposure and thus cause a blurred image. You will find a good image of the page in the adjacent frame.

3. When a map, drawing or chart, etc., was part of the material being photographed the photographer followed a definite method in "sectioning" the material. It is customary to begin photoing at the upper left hand corner of a large sheet and to continue photoing from left to right in equal sections with a small overlap. If necessary, sectioning is continued again — beginning below the first row and continuing on until complete.

4. The majority of users indicate that the textual content is of greatest value, however, a somewhat higher quality reproduction could be made from "photographs" if essential to the understanding of the dissertation. Silver prints of "photographs" may be ordered at additional charge by writing the Order Department, giving the catalog number, title, author and specific pages you wish reproduced.

5. PLEASE NOTE: Some pages may have indistinct print. Filmed as received.

Xerox University Microfilms
300 North Zeeb Road
Ann Arbor, Michigan 48106

75-12,687

LESLIE, Robert C., Jr., 1935-
MIXED FIXED RATIO DRO PERFORMANCE IN NORMAL
AND CAUDATE LESIONED RATS.

The City University of New York, Ph.D., 1975
Psychology, physiological

Xerox University Microfilms, Ann Arbor, Michigan 48106

MIXED FIXED RATIO DRO PERFORMANCE
IN NORMAL AND CAUDATE LESIONED RATS

by

ROBERT C. LESLIE, JR.

A dissertation submitted to the Graduate
Faculty in Psychology in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy, The City University
of New York.

1975

This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

January 21, 1975
date

Robert L Thompson
Chairman of the Examining Committee

January 22, 1975
date

Florence L. Denmark
Executive Officer

Prof. Donald E. Mintz

Prof. Eliot Gardner

Prof. Robert L. Thompson, Chairman
Supervisory Committee

The City University of New York

Abstract

MIXED FIXED RATIO DRO PERFORMANCE IN NORMAL AND CAUDATE LESIONED RATS

Robert C. Leslie, Jr.

Advisor: Professor Robert L. Thompson

Rats with small bilateral lesions of the caudate nucleus and unoperated rats were trained on a mixed schedule of reinforcement (Mixed Fixed Ratio, Differential Reinforcement of Other Behavior or Mix FR DRO) to provide a behavioral description of lesion effects characterized as "perseverative", "failures of inhibition", "inability to shift from one mode of response to another", or "failure to discriminate response produced stimuli". Mix FR DRO schedules alternate FR reinforcement periods with DRO non-reinforced periods, neither component being exteroceptively cued. These schedules were considered representative of the kind of task on which caudate lesioned animals are impaired because they required (1) discrimination of non-reinforcement periods solely on the basis of cues generated by the animal's own responding, and (2) the requirement during DRO periods of not responding, i.e., "inhibition".

Experiment I studied post-operative acquisition and steady state performance at four levels of the ratio (FR 1, 4, 8, and 16) in three groups of rats: anterior caudate, posterior caudate, and unoperated rats. Radio frequency lesions were found to be small when compared to lesions in other studies (e.g., Chorover and Gross, 1963; Kirkby and Kimble, 1968).

The primary findings were (1) the groups did not differ with respect to total responses in DRO, number of runs in DRO, or number of responses per initial run in DRO; (2) in all groups total responses per DRO, num-

ber of responses per initial run in DRO, and median latency of first response in FR were increasing functions of ratio size, thus extending to mixed schedules Boren's (1961) finding regarding resistance to extinction as a function of ratio size; (3) median latencies of first response in FR were longer following reinforced runs than following DRO periods; (4) mean number of responses in initial runs in DRO emitted by the unoperated rats approximated run lengths reported by Mechner (1958a) and Platt and Senkowski (1970), but standard deviations of run length distributions were not linear functions of ratio size as found by Platt and Senkowski (1970). It was not possible therefore to employ the linear generalization of Weber's Law to provide discriminability functions for number of responses as a stimulus as had Platt and Senkowski. No further attempt was made to obtain discriminability functions using transforms of the data or some formulation other than Weber's Law. It was pointed out that in contrast to Mechner's (1958a) procedure the Mix FR DRO schedule does not maximize differentiation of run lengths.

Experiment II compared pre- and post-operative performance of Mix FR 1 DRO in anterior caudate, posterior caudate, and unoperated rats. Since only two posterior animals completed the experiment it became essentially a two group design. Radio frequency lesions were again relatively small, but showed greater anterior to posterior extent than in Experiment I. The principle findings were that (1) during both first and last five post-operative sessions anterior caudate rats emitted about twice as many responses and runs per DRO period as did unoperated rats; (2) during the first five post-operative sessions number of responses per initial run in DRO did not differ between groups, but over the last five post-operative sessions number of responses per

initial run was about 50 percent greater in the anterior group than in the unoperated rats; (3) both post FR and post DRO median latencies of the first response in FR periods were two to four times longer for anterior rats than for unoperated controls over both first and last five post-operative sessions; (4) in all groups post FR latencies were longer than post DRO latencies.

That Experiment II showed a lesion effect while Experiment I did not agreed with previous findings that effects of relatively small caudate lesions are more likely seen in comparison of pre- and post-operative performance than in acquisition (Thompson, 1959). It was also suggested that the greater extent of lesions in Experiment II and the possible obscuring of lesion effects at higher ratios in Experiment I may have contributed to the discrepancy between experiments.

It was suggested that for purposes of further study the caudate lesion effect could be considered as arising from reduced control of behavior by conditioned reinforcers in proportion to the degree of discontiguity between such stimuli and primary reinforcement.

ACKNOWLEDGEMENTS

This work was supported by a City University of New York Faculty Research Grant (Robert L. Thompson, principle investigator) and by funds from the Biopsychology Program, Hunter College of the City University of New York.

Particular gratitude is expressed to Professor Thompson for his guidance and support throughout the course of this project and throughout the author's graduate career.

The author also wishes to thank the members of his dissertation and final examination committees: Professors Donald E. Mintz, Eliot Gardner, Sally Louise Hobson, and Everett Wyers, for their truly helpful suggestions and comments.

Thanks are also due to the following fellow graduate students, faculty and staff at both Hunter College of the City University of New York and the State University of New York at Oneonta for typing, technical aid and moral support: Ellen Brown, Foster L. Brown, Julie Sage Day, Ron Embling and his staff, Dirk Houben, Frank Le Fever, Terry and David Malcolm, Agnes Riley, Michael Potegal, Dorothy Siegel, John Van Esen, John Van Laer, Miriam Vedder, and H. Philip Ziegler.

Finally, the author wishes to thank his wife, Mary, for her patient support and understanding.

TABLE OF CONTENTS

Page

LIST OF TABLES x

LIST OF FIGURES xi

INTRODUCTION 1

- Initial remarks 1
- Performance of caudate lesioned animals on experimental learning tasks 2
- Caudate lesions and activity in rats 5
- Caudate lesioned rats on spatial alternation and related tasks 7
- Caudate lesioned rats on avoidance learning tasks 14
- Caudate lesioned rats and schedules of reinforcement 18
- Behavioral characterization of caudate lesion effects in rats 20
- Mixed fixed-ratio schedules 23
- The present study 27

EXPERIMENT I 29

- Method 29
- Results 40

EXPERIMENT II 101

- Method 101
- Results 103

DISCUSSION 123

- Initial comments 123
- Acquisition and steady state behavior on Mix FR DRO 124
- Effects of caudate lesions 130
- Characterization of caudate lesion effects 134
- Concluding remarks 140

Page

APPENDIX I 141

APPENDIX II 181

REFERENCES 187

LIST OF TABLES

Table Page

1 55

2 66

3 126

4 132

LIST OF FIGURES

Figure Page

1 39

2 42

3 44

4 46

5 48

6 53

7 57

8 60

9 62

10 64

11 70

12 72

13 74

14 77

15 79

16 82

17 84

18 87

19 89

20 91

21 93

22 95

23 97

24 99

25 105

Figure Page

26 107

27 109

28 111

29 114

30 116

31 118

32 121

INTRODUCTION

Initial Remarks

The caudate nucleus of mammals is a subcortical forebrain structure considered to play an important role in the regulation of locomotion, postural adjustment and emotional behavior (Jung and Hassler, 1960; Denny-Brown, 1962; Martin, 1967; Divac, 1968b; MacLean, 1972). The nature of this role, defined in terms of experimental studies of animal behavior and learning, has not yet been determined (Laursen, 1963; Divac, 1968b).

One current view of caudate nucleus function is that it is primarily inhibitory (e.g., Mettler, 1942; Fox, Kimble, and Lickey, 1963). Support for this view comes from several sources. Clinical studies of both human beings and animals have associated destruction of caudate and related tissue with the appearance of involuntary motor signs such as chorea, athetosis, and cursive hyperkinesia (Mettler, 1942; Denny-Brown, 1962; Martin, 1967; Liles and Davis, 1969). Chorea is characterized by jerky and rapid movements affecting the limbs, face, and trunk, while athetosis consists in slower, writhing movements of the same parts (Smith, 1965). Cursive hyperkinesia, a term coined by Mettler (1942), refers to the fact that animals with bilateral lesions of the caudate are frequently observed to walk or run in a forward direction without pause, and with apparent disregard of obstacles (Martin, 1967). Martin (1967) has suggested that such signs represent the "release" of reflex activity from normal inhibitory regulation.

The results of experimental investigation with animals have also contributed to the assignment of an inhibitory function to the caudate. For example, electrical stimulation of the caudate nucleus has been found to produce a number of reliable behavioral effects which vary with the parameters of stimulation (Jung and Hassler, 1960; Laursen, 1963; Grossman, 1967; McLennan, Emmons, and Plummer, 1964). One of the behavioral effects which most strongly suggest an inhibitory function is the so-called "arrest" response which refers to the cessation of on-going behavioral activity when caudate stimulation is superimposed (e.g., Mettler and Mettler, 1942; Rosvold and Delgado, 1956; Buchwald, Wyers, Lauprecht, and Heuser, 1961; Rubenstein and Delgado, 1963; Kitsikis, 1968; Stamm, 1969; Deadwyler and Wyers, 1970).

Additional support for an inhibitory theory of caudate function has come from studies employing experimental lesions. Animals with caudate lesions have been reported to exhibit hyperkinesia, sometimes taking the form of "obstinate progression" or "compulsive" locomotion, e.g., continual pacing back and forth in cages, uninterrupted bouts of running (Mettler and Mettler, 1942; Davis, 1958; Dean and Davis, 1959; Whittier and Orr, 1962).

Performance of Caudate Lesioned Animals on Experimental Learning Tasks

The majority of studies concerning the behavior of caudate lesioned animals has involved performance on experimental learning tasks. Caudate lesioned animals have been reported either to fail or to be slow to learn delayed response and delayed alternation tasks (e.g., Battig, Rosvold, and Mishkin, 1960, 1962; Rosvold and Delgado, 1956; Gross, Chorover, and Cohen, 1964; Divac, Rosvold, and Szwarcbart, 1967; Divac, 1968). On active and passive avoidance tasks caudate lesioned animals have been reported to be unable to retain or relearn appropriate responses (e.g., Thompson, 1959; Thompson and Mettler, 1963; Fox, Kimble, and Lickey, 1964; Kirkby and Kimble, 1968). On fixed interval and DRL schedules of reinforcement caudate lesioned animals tend to respond more frequently than do normal animals (e.g., Thompson, 1963a; Thompson, Ayers, and Mettler, 1962; Schmaltz and Isaacson, 1968; Hansing, Schwartzbaum, and Thompson, 1968).

It has also been shown that animals with caudate lesions exhibit greater resistance to extinction on mixed fixed ratio-extinction and variable interval schedules of reinforcement (Thompson, 1963b; Unger, 1966; Butters and Rosvold, 1968).

Many of the studies cited above, especially those involving positively reinforced responding, have been taken as providing evidence that the primary function of the caudate nucleus is indeed inhibitory, in that the lesioned animal engages in premature responding when required to delay, or commits errors of perseveration. Perseveration errors may involve the emission of previously reinforced responses under conditions in which these responses are either no longer reinforced or followed by aversive stimulation.

Since the behavioral changes resulting from caudate lesions are similar in many respects to those changes resulting from damage to the frontal lobes, it has been suggested that these two areas (the caudate nucleus and the frontal lobes) along with other structures comprise a system subserving the same basic function(s) (Rosvold and Szwarcbart, 1964; Rosvold, 1968). Rosvold (1968) has hypothesized that the caudate nucleus and the prefrontal cortex function as part of a system for effecting "correction in response mechanisms" and postulates two such mechanisms. One has a supposedly inhibitory function which serves to determine whether or not a given response will occur (e.g., Go, No-Go alternations); the other serves a spatial function which influences where a response will occur (e.g., spatial alternation tasks).

That the caudate nucleus may subserve behavior which involves the discrimination of spatial position is suggested by the slowness with which caudate animals often learn spatial alternations and reversals (Rosvold, 1968; Potegal, 1969; Divac, 1971). It is further suggested by the failure of caudate lesioned monkeys to correct errors made while attempting to track a moving target or to correct errors induced by the prismatic displacement of the visual image (Bossom, 1965, 1972; Bowen, 1969).

Divac (1968b) contended that the caudate nucleus may perform the same behaviorally defined role as the cortex, but on a different "level," depending upon the position of the species in the evolutionary scale. He suggested that there exists a "level" of phylogenetic development in which the striatum is the "highest" formation of the brain. For example, Divac noted that the striatum of some birds and reptiles appears to represent the most phylogenetically recent, integrative central nervous structure. According to Divac, the striatum is more "primitively" organized than the cortex, and is more or less "dominated" by the cortex depending upon the degree of corticalization in a species. Critical to Divac's hypothesis are two theories: (1) that the caudate nucleus is "coupled" to all sensory systems and is thus in a position to integrate complex motor and autonomic functioning, and (2) that the caudate is functionally heterogeneous and topographically related to the cortex in a manner which reflects that heterogeneity.

If, as Divac (1968b) suggested, the striatum of some species does subserve some of the same behaviorally defined roles as the cortex in other species, an understanding of caudate nucleus function would be considerably advanced by extending investigations to a wider range of species. The majority of studies of caudate nucleus function have employed species with relatively well developed cortices, e.g., monkeys, cats, and dogs. Only recently have substantial caudate lesion effects been reported for the rat, a species whose caudate nucleus, if Divac is correct, might subserve many of the same functions attributed to certain cortical areas of "higher" mammals.

Caudate Lesions and Activity in Rats

One of the recent studies to investigate the effects of caudate lesions in rats was that of Whittier and Orr (1962) who investigated the effects of bilateral electrolytic lesions on several measures of general activity. It was found that caudate lesions resulted in the development of pronounced cursive hyperkinesia. In running wheels caudate rats exhibited longer continuous bouts of running with fewer pauses than did either cortically lesioned or sham operated controls. In some cases the number of revolutions in the running wheel for a sixteen-hour period was quadrupled compared to preoperative measures. This increased activity was sustained for about thirty days post-operatively, at which time activity was observed to return to the preoperative level. It was found, however, that if the amount of caudate tissue destroyed was less than 20 percent, hyperactivity was not observed. Also, when observed in cages or on table tops, the rats engaged in continuous ambulatory and cursive behavior showing little or no tendency to pause. These findings are similar to the reports of hyperkinesia in caudate lesioned cats (Mettler and Mettler, 1942). Whittier and Orr (1962) also reported that caudate lesioned rats evidenced "transient" aphagia and adipsia; aphagia lasting for mean of 1.8 days following surgery, and adipsia lasting for a mean of 3.8 days following surgery.

On the basis of these findings, Whittier and Orr (1962) concluded that the destruction of caudate tissue in the rat interfered with the animal's ability to shift from one mode of behavior to another. They thus concurred with Mettler (1955) and Mettler and Crandell (1959), who contended that caudate lesions produced a condition which they labelled "proharesis," or an inability to initiate behavior.

In a study which investigated the effects of caudate lesions on mating and maternal behavior in rats, Kirkby (1967) measured the relative amount of time spent in various behaviors by both lesioned and unoperated rats. The caudate rats appeared to spend more time than the unoperated rats in what Kirkby called "activity behavior," and also in self grooming and manipulating pups, than did the controls. The nature of the "activity behavior" was not specified.

Green, Beatty, and Schwartzbaum (1967) reported that when tested one week following surgery, caudate rats exhibited a higher rate of shuttling activity in a shuttle box during a preshock adaptation period prior to avoidance training than did cortical and sham operated controls. Tests carried out four to six weeks following surgery, however, revealed no differences between the caudate rats and the controls.

Albert and Bignami (1968) recorded the number of between trial crossings in a shuttle box during active avoidance learning and found that during the first three days of post-operative testing both caudate and cortically lesioned rats made more crossings than operated controls. By the seventh post-operative session, however, no inter-group differences were evident.

Borst, Delacour, and Licouban (1970) reported failing to find any evidence of increased activity in caudate lesioned rats based on either observation of general behavior or the number of spontaneous alternations, when compared with operated, but unlesioned animals.

Lynch (1970) included a caudate lesion group in a study concerned with the effects on activity of lesions placed in either the dorsal or ventral forebrain of the rat. The caudate rats showed no signs of increased activity in stabilimeter cages under either deprived or free feeding conditions.

While two of the studies mentioned failed to report increases in activity following caudate lesions, this can probably be accounted for in terms of the different measurement procedures employed. In general, the literature appears to suggest that rats, as well as other species, do evidence cursive hyperkinesia following bilateral caudate damage and that this increased activity tends to dissipate with time after surgery.

Caudate Lesioned Rats on Spatial Alternation and Related Task

The body of work concerned with the effects of caudate lesions on learning tasks also suggests that the types of deficits produced in rats are similar to those produced in other species. Chorover and Gross (1963) investigated the effects of caudate lesions in rats on the acquisition and retention of an alternation task in which rats were reinforced whenever a response on one of two levers in an operant chamber was preceded by a response on the other lever. An error was recorded whenever the same lever was pressed twice in succession. Training was continued until a criterion of 80 percent correct had been run. During both acquisition and retention the caudate rats committed more errors and thus needed more trials to reach criterion than did either cortical or unoperated controls. The caudate rats, however, did not take longer than either of the two control groups to learn a Hebb-Williams maze. Chorover and Gross concluded that the performance of the caudate rats on the alternation task was not due to a general behavioral disfunction, and that adequate performance by rats of spatial alternation requires an intact caudate nucleus.

Gross, Chorover, and Cohen (1965) compared caudate lesioned rats to rats with anterior cortex, posterior cortex, dorsal thalamic or hippocampal lesions, as well as with unoperated controls. In this experiment reinforcement was delivered whenever a rat responded on a left-hand lever followed by a single press on a right-hand lever. Any departure from the press-left, press-right sequence produced a five-second time out and counted as an error. The training criterion was 80 percent correct in four out of five days, or 13,000 trials, whichever occurred first. On both acquisition and retention the caudate group made more errors and took more trials to reach criterion than the posterior cortex or unoperated groups. General behavioral impairment was again discounted on the basis of a Hebb-Williams maze performance. The investigators also discounted hyperactivity as a source of errors on the grounds that hyperactivity in caudate lesioned rats is only temporary, and that latencies measured in their own study did not suggest hyperactivity. Direct measurements of activity were not undertaken in their study, however. It was also concluded that the errors on the sequential bar pressing task did not appear to result from response perseveration in that there were no differences in the number of successive responses on any one bar. The authors suggested that the deficit produced by the caudate lesions was specific to the performance of chained response sequences, admitting, however, that performance on the

Hebb-Williams maze, which was not impaired, could also be construed as involving chained responding.

In view of an hypothesis to be discussed later, it is worth noting that the chaining of responses required in the bar-press sequence might require greater dependence on response produced stimuli than would the negotiation of a maze which presumably offers a greater variety of exteroceptive stimuli.

Unger (1966) pointed out that the task employed by Chorover and Gross (1963) permitted the rats to make only one type of error; i.e., the error of an additional response on the bar most recently pressed. Unger used a procedure developed by Mechner (1958) in which a fixed ratio (FR) requirement was in effect on the first lever in a two lever sequence. The rat had to complete the FR on one lever before being reinforced for a single response on the second lever. This procedure permitted errors involving premature switching from lever one to lever two, as well as errors involving continued responding on either lever. Using FRs of 3, 5, and 10, Unger found that caudate lesioned rats tended to undershoot the FR requirement; that is, switch prematurely. It was also noted that the caudate lesioned rats, having once switched to the second lever, tended to make more consecutive responses on that lever than did the controls. Thus, both "anticipatory" and "perseverative" errors were committed by the caudate animals. Unger concluded that an inhibitory failure could not be called upon to account for both types of errors.

Commenting on the Chorover and Gross (1963) study, Mikulas and Isaacson (1965) interpreted the results as indicative of a failure of inhibition resulting from caudate damage. They tested their hypothesis with a delayed response task in which rats had to respond by entering one of four goal boxes after a ten-second delay following the presentation of a light over the entrance to the correct goal box. None of the caudate lesioned rats was able to achieve the criterion performance of four out of five correct responses within the imposed limit of 200 trials, as opposed to operated and unoperated controls. When errors were analyzed, it was found that the caudate rats tended to approach that goal box which had been approached most frequently on previous trials. Furthermore, the caudate rats demonstrated a preference for the outermost of the four goal boxes.

In a second study Mikulas (1966) ran normal and caudate lesioned rats in a spatial alternation task in a T-maze. In one condition the correct arm was indicated by the presence of a light; in another condition the light was omitted. In both conditions the caudate rats required more trials to achieve criterion than did normal controls. The caudate rats made fewer errors in the light condition than in the no light condition, however. Mikulas interpreted this latter finding to indicate that the presence of the cue light served to "distract" the perseverative tendencies which resulted from the caudate lesion.

Schwartzbaum and Donovick (1968) compared the performance of caudate lesioned rats with septal and sham operated rats on both spatial alternation and brightness discrimination tasks. The apparatus used was a shuttle box with two lever manipulanda at either end. In the brightness discrimination task the rats were required to press the lever underneath the brighter of two lights and then shuttle to the other pair of levers and repeat the task. Sixty such trials (30 in each compartment of the shuttle box) were run, with the rats required to shuttle between each trial. The other task, spatial alternation, involved simply shuttling between each side of the box and pressing either of the two levers at a given end in order to receive reinforcement. No significant differences were found between the three groups on the spatial alternation task, but on the brightness discrimination task the caudate rats made significantly more errors. Most of these errors involved continued responding at one end of the shuttle box, i.e., failures to shuttle. The authors noted that these results appeared to be inconsistent with reports that caudate lesioned rats fail on spatial alternation, but not on brightness discrimination. They pointed out, however, that the brightness discrimination task which they employed actually could be construed as a spatial alternation task with added visual cues, and that the errors made by the caudate rats were probably more consistent with failure to alternate than with a failure of brightness discrimination. From a close inspection of the description of their procedure, it appeared that the so-called brightness task may well have presented the rats with a spatial alternation problem somewhat more difficult than the alternation task without visual cues.

In an attempt to explore further the role of the caudate nucleus in the performance of tasks involving the discrimination of spatial position, Potegal (1969) studied the performance of caudate and medial thalamic rats on both a spatial alternation task and on the acquisition of a simple position habit. The spatial alternation task involved the formation of what was termed an "absolute position habit" in which location of the correct goal box was not correlated with exteroceptive cues arising from either within or without the maze. To learn such a position habit an organism must rely upon cues arising from its own bodily position, i.e., proprioceptive, kinesthetic, tactile or vestibular stimuli. The simple position task, on the other hand, involved the formation of a "relative" position habit in which the location of the correct goal box could be determined through the utilization of extra and intra-maze stimuli. The caudate lesioned rats were found to make more errors than sham operated controls on the "absolute" position task, while performing as well as the controls on the "relative" position task. Potegal observed that while the caudate rats evidenced some degree of response stereotypy on the alternation task, this occurred later than the incidence of a higher error rate. Potegal concluded that the response stereotypy was secondary to whatever factor produced the errors, and not the source of the errors. Having equated tasks for difficulty, Potegal suggested that since the caudate rats performed poorly on a task requiring "absolute" spatial organization as opposed to "relative" spatial organization, the lesions may have interfered with the rats' ability to utilize proprioceptive, kinesthetic, or vestibular cues.

Potegal's (1969) experiment was also of interest in that he found a positive correlation to exist between the location of the caudal boundary of the caudate lesion and the degree of impairment. This suggested that there exists a critical locus in the posterior caudate which, according to Potegal, may be in the region of the transverse limb of the anterior commissure.

Borst, Delacour and Licouban (1970) found that rats with lesions in the anterior caudate nucleus entered significantly more blind alleys than cortical or sham operated controls in a maze which required a left-right-left-right alternation. The anterior caudate rats performed as well as the control animals on another maze task in which all of the turns were to the right. In a second part of the same experiment a medial caudate lesion group was found to perform as well as the controls. Contrary to Potegal (1969), these findings suggested the anterior caudate to be more critical than either the medial or posterior portions. It was concluded that the nature of the impairment resulting from the anterior lesion involved either a failure of short-term memory or a failure to inhibit instrumental responses.

Divac (1971) compared rats with medial caudate lesions with rats which had lesions of either the frontal poles or the medial cortex, on a task involving a series of four spatial reversals. It was found that both the caudate and the medial cortex groups made significantly more errors than did the rats with frontal pole lesions. It was further found that the number of errors made by the caudate rats on the first three reversals was significantly greater than either of the other two groups.

It seems clear from the literature just reviewed that caudate lesions interfere in some way with a rat's ability to perform tasks which involve spatial alternation. Whether this impairment is properly construed as a failure of inhibition (i.e., response perseveration), an inability to discriminate, store, or otherwise use information arising from proprioceptive stimuli, or some other impairment of spatial organization is not clear. There is also some suggestion that the locus of caudate lesions may be an important variable. That this may be the case should not be surprising in view of the heterogeneity of function found in the caudate nucleus of other species (e.g., Rosvold, 1968; Divac, 1968b).

Caudate Lesioned Rats on Avoidance Learning Tasks

Lesions of the rat caudate nucleus have also been found to affect performance on a variety of active and passive avoidance problems. Green, Beatty and Schwartzbaum (1967) found that rats with caudate lesions attained lower asymptotic levels of performance in terms of percent successful avoidance responses in a shuttle box than either septal, hippocampal, or sham operated rats. Interestingly, the investigators point out that a criterion measure of performance (e.g., nine out of ten successful avoidance responses) would not have revealed any performance differences between the caudate and other groups.

Kirkby and Kimble (1968) conducted a series of experiments in which they investigated the behavior of caudate rats on a variety of avoidance tasks. In one type, a passive-avoidance problem, caudate, cortical and unoperated control rats were first given ten days of training in a Y-maze in which entry into one arm was reinforced by access to a water bottle. On the fifth trial of the eleventh day the rats were shocked upon approaching the water bottle. On subsequent trials it was observed that while the approach latencies to the water bottle were significantly increased over the pre-shock levels for all groups, the caudate rats showed significantly shorter latencies than either of the other two groups. In a second experiment similar groups of rats were permitted a single opportunity to drink from a water cup apparatus before shock was administered on the second trial. In this situation the mean number of contacts with the water cup did not differ significantly between any of the groups. On the basis of these two tests it was concluded that caudate rats were unable to inhibit an approach response if they had been "well-trained" previously, but could inhibit if not previously "well-trained."

In a third experiment Kirkby and Kimble (1968) studied acquisition of one-way active avoidance in a shuttle box, and found that caudate rats required more trials to reach the criterion of successful avoidance, and also made more failures to avoid and/or escape shock than either hippocampal or unoperated controls. The authors concluded that the warning buzzer produced arousal which was abnormally enhanced in the caudate rats due to the absence of normal inhibitory responses. The caudate rats were thus judged to have been "disorganized" and unable to acquire appropriate avoidance responses as rapidly as the control rats.

In seeming contrast to the results reported by Kirkby and Kimble (1968), and by Green, Beatty, and Schwartzbaum (1967), Albert and Bigmani (1968) failed to find evidence of a caudate deficit in active avoidance. They found that caudate rats made the same proportion of successful avoidance responses in a two-way shuttle box situation as sham operated controls. Furthermore, mean avoidance response latencies for these two groups were not significantly different.

Winocur and Mills (1969) reported a failure to find any significant differences in trials to achieve criterion performance in a one-way active avoidance task between caudate, cortical, and sham operated rats. They did find, however, that rats with caudate lesions required three to four times as many trials to attain criterion on a passive avoidance task. Since the passive avoidance tests had been conducted using the same animals and apparatus used for the active avoidance task, a second experiment was performed in which different caudate, cortical and sham operated controls were compared on passive avoidance without prior training. In this second experiment no differences in median approach latencies were found between any of the groups.

Winocur and Mills (1969) concluded that rats with caudate nucleus lesions are able to acquire a passive avoidance response when required to inhibit an unlearned response, but not when required to inhibit a response which had been previously reinforced. With respect to passive avoidance, these conclusions appeared to support those reached by Kirkby and Kimble (1968). Winocur and Mills felt, however, that a simple response inhibition hypothesis did not provide an adequate explanation. Pointing to a study by Olton and Isaacson (1968) concerning the nature of active avoidance in shuttle boxes, they argued that spatial cues play an important role in the acquisition of appropriate avoidance behavior; i.e., animals learn where shock will be delivered. Thus, they suggested the acquisition of successful avoidance may be construed as learning a spatial discrimination. Winocur and Mills suggested that since the rats in their first passive avoidance test had previously acquired successful active avoidance in a shuttle box, the learning of the passive avoidance task required them to "reorganize" spatial information, and it may have been this requirement rather than an inability to inhibit approach responses which was the critical factor underlying the poor performance.

This view seems particularly important in the light of the literature previously reviewed, which strongly indicates a role for the caudate nucleus in tasks involving spatial alternation or reversal, and which also casts doubt on the adequacy of an inhibition explanation (e.g., Gross, Chorover, and Cohen, 1965; Potegal, 1968).

Another interesting point can be made in connection with the Winocur and Mills (1969) study. Kirkby (1970), commenting on that study, indicated that their report that caudate lesioned rats were not impaired in the acquisition of active avoidance was inconsistent with a substantial part of the current literature (e.g., Thompson and Mettler, 1963; Green, Beatty, and Schwartzbaum, 1967; Konorski, 1967; Kirkby and Kimble, 1968). In response to Kirkby's comment, Winocur and Mills (1970) argued that in those studies which reported failures to acquire avoidance, the lesions had been directed at the postero-ventral portions of the nucleus and were sufficiently large to have encroached on other structures. According to Winocur and Mills (1970), lesions restricted to the antero-dorsal caudate did not result in failure to acquire active avoidance responses (e.g., Winocur and Mills, 1969; Albert and Bigmani, 1968).

Two studies, however, have subsequently reported finding caudate rats unable to acquire active avoidance when the lesions were reasonably well confined to the antero-dorsal part of the caudate nucleus. Neill and Grossman (1970) placed lesions in either the dorsal or ventral areas of the anterior caudate in rats and found that these animals made significantly fewer successful avoidance responses than operated or unoperated controls. Mitcham and Thomas (1972) compared the performance of rats with either antero-dorsal caudate or substantia nigra lesions with both operated and normal controls on one- and two-way active avoidance, and on passive avoidance. In one experiment in which the rats were first trained on one-way active avoidance and then on a passive avoidance task, they found that the caudate and nigral rats took significantly more trials than controls to reach a criterion of nine out of ten successful avoidance responses. The nigral rats were also found to take significantly more trials than the caudate rats. Identical groups were run on the passive avoidance problem, and it was again found that the caudate and nigral rats took more trials to reach the criterion than did any of the controls. No differences between the caudate and nigral rats were found on the passive avoidance task.

In the second part of their study Mitcham and Thomas (1972) trained different groups of antero-dorsal caudate, nigral, and control rats in a two-way active avoidance task. They found that while the control groups all showed negatively accelerated acquisition curves, neither the caudate nor the nigral rats ever achieved better than 20 percent successful avoidance responses. Mitcham and Thomas concluded that the results of their study were consistent with Kirkby's (1970) findings, to the effect that lesions of the caudate nucleus do produce deficits in rats on both one- and two-way active avoidance.

Caudate Lesioned Rats and Schedules of Reinforcement

Relatively few studies concerned with the effects of caudate nucleus damage in rats have employed the use of the free operant with traditional schedules of reinforcement. In one such study Allen, Mitcham, and Byrd (1972) compared the performance of caudate lesioned rats with operated and normal control animals on a schedule of unsignalled (Sidman) avoidance. They found that while the caudate rats acquired the bar press avoidance response they received higher steady state shock rates than did either of the control groups. Distributions of interresponse times (IRTs) showed that the caudate performances were characterized by sustained post-shock bursts followed by long pauses of no responding until the delivery of shock. Interresponse times per opportunity (IRTs/Op) functions provided no evidence that the caudate rats acquired the sort of temporal discrimination which accompanies successful avoidance on such schedules. The investigators suggested that the caudate lesions may have interfered with the acquisition of normal timing behavior.

Hansing, Schwartzbaum, and Thompson (1968) found that rats with bilateral caudate lesions emitted more responses during the first half of the interval on a fixed interval one minute (FI 1) schedule than either unilateral caudate rats or unoperated controls. These results were taken to indicate an impairment of normal response inhibition.

In contrast to Hansing, Schwartzbaum, and Thompson (1968), Unger (1966) failed to find any differences in the temporal patterning of responses on a fixed interval one minute schedule between rats with caudate lesions and either cortically lesioned or unoperated controls. Unger found, however, that the caudate rats took longer to extinguish responding than did the rats in the other two groups. The inconsistency between Unger's and Hansing's _et_ _al_. results can probably be accounted for in terms of locus of lesion placement. Unger's lesions were directed considerably more anteriorally than were the lesions made by Hansing _et_ _al_.

On a multiple schedule which alternated a variable interval one minute component with an extinction component Unger (1966) found that caudate lesioned rats emitted significantly more responses during the extinction component than did any of the controls. Unger also examined the performance of caudate lesioned rats on a concurrent schedule of reinforcement in which a three minute variable interval was in effect on one lever, and a thirty second variable interval was in effect on another lever. In terms of the number of reinforcements obtained, the performance of the caudate rats could not be distinguished from that of the control animals. The fact that the caudate lesioned rats performed as well as the controls on both the fixed interval schedule and the concurrent schedule led Unger to reject the hypothesis that caudate deficits are primarily perseverative in nature. Referring to the alternation study discussed earlier in the present paper (page 9), Unger argued that the impairment evidenced by caudate rats is related to the requirement of having to shift from one mode of responding to another. It was this requirement that both the alternation task and the multiple schedule appeared to have in common.

Finally, two studies have investigated the performance of rats with caudate nucleus lesions on schedules involving the differential reinforcement of low rates of responding (DRL). Meyer (1967) trained caudate rats on DRL schedules of 5, 10, and 15 seconds and failed to find any differences in the mean number of responses emitted between the caudate rats and either frontal or sham operated controls. Schmaltz and Isaacson (1968), using a DRL 20 second schedule, found their caudate rats emitted more responses during the first ten of forty retention sessions than were emitted by rats with frontal lesions. The discrepancy between these two studies can probably be resolved in terms of lesion placement. Meyer's lesions were placed relatively more dorsally than those placed by Schmaltz and Isaacson. Another difference is that the DRL interval employed by Schmaltz and Isaacson was longer than that used in Meyer's study.

Behavioral Characterization of Caudate Lesion Effects in Rats

Studies of the effects of caudate nucleus lesions in rats appear to be consistent with findings outlined earlier in this discussion concerning the effects of caudate nucleus lesions in other species. Similar explanations have been offered to account for the behavioral changes observed; e.g., failure to inhibit previously trained responses, tendency to perseverate, inability to shift from one mode of response to another or to initiate behavior, interference with normal utilization of spatial cues, etc. Some of these attempts to account for the role of the caudate nucleus in behavior represent little more than summary descriptions of the behavior itself, or seem to be the products of the kind of reasoning which Gregory (1961) has criticized; namely, that in the absence of an explicit knowledge of the exact role of a brain area as a functioning part of a circuit, the area is inappropriately assigned a function on the basis of behavioral changes resulting from the destruction of that area. The assignment of an inhibitory function to the caudate nucleus seems to provide a fairly good example of this kind of reasoning. Dews' (1958) advice concerning the analysis of pharmacologic and other physiological effects on behavior seems particularly relevant here. Dews suggested that experimental investigations concerned with behavior and physiological processes should strive to provide operational analyses. Rather than appeal to unspecific processes, investigations should attempt to determine to what extent alterations in the physiological substrate produce changes on dependent variables which are like changes produced by other experimental operations with which we may be more familiar.

With the intention of just such a behavioral analysis of caudate nucleus function, Thompson (1963a) analyzed the typical deficits suffered by caudate lesioned animals in terms consistent with current reinforcement theory. Thompson pointed out that caudate lesioned animals are typically impaired when tasks require the bridging of a delay interval or contain an extinction component. If extinction is considered a behavioral equivalent of inhibition, it is thus suggested that caudate lesions increase resistance to extinction.

Thompson (1963a) also suggested, as did Miles (1964), that on those tasks which include a delay requirement (e.g., delayed alternation, DRL schedules) animals may learn to bridge such delays through the acquisition of adventitiously chained "timing" or mediating behavior. The development of such chains can be conceived in several ways. Schoenfeld and Farmer (1970) pointed out, for example, that whenever a response class (R) is specified, its complement (not-R) is also necessarily defined. If it is assumed that an organism is always doing something, then any decrease in the probability of R must be accompanied by an increase in the probability of not-R. According to such a view, the ability of an organism to delay or inhibit behavior involves increases in the frequency of "other" behavior.

The development of such chains of "other" behavior has been regarded as important to the acquisition of optimal performance on schedules of reinforcement in which periods of non-reinforced lever pressing (i.e., extinction) alternate with periods of reinforced responding, or when continued responding during periods of non-reinforcement has the effect of postponing occasions for reinforcement. This appears to be especially true when neither reinforcement nor non-reinforcement periods are correlated with exteroceptive stimuli (Wilson and Keller, 1953; Ferster and Skinner, 1957; Weiss, Laties, Siegel, and Goldstein, 1966).

It has been further argued by behavior theorists that in the absence of correlated exteroceptive stimuli, the emission of "other" behavior necessarily comes under the control of response produced stimuli, i.e., proprioceptive, kinesthetic, or tactile stimuli (Keller and

Schoenfeld, 1950; Ferster and Skinner, 1957; Thompson, 1963a).

If the development of chains of "other" behavior, occasioned or controlled largely by response produced stimuli, is a requirement characteristic of the kind of learning tasks on which caudate lesioned animals are most often impaired, then it may be that caudate damage interferes in some way with the discrimination of response produced stimuli. This view would seem consistent with Potegal's (1969) argument that caudate lesions interfere with spatial "ego-centric" localization; i.e., localization on the basis of stimuli arising from the animal's bodily orientation. It would also appear consistent with those studies in which caudate lesioned animals have been described as unable to shift from one response mode to another. In such studies the occasions for shifting were seldom exteroceptively cued (e.g., Gross, Chorover, and Cohen, 1965; Unger, 1966).

In short, it seems reasonable to suggest that the behavioral deficits displayed by animals with caudate lesions, characterized as failures of inhibition, inability to shift from one response to another, disruption of "ego-centric" localization, may all reflect a disruption in the normal proprioceptive or kinesthetic control of sequential behavior.

Mixed Fixed-Ratio Schedules

The principle difficulty with such a hypothesis is the relative invisibility of response produced stimuli. This problem has led some theorists to dismiss appeals to response produced stimuli as unproductive (e.g., Kelleher, 1966). Current operant technology does, however, provide several ways in which the role of response produced stimuli may be reflected. Among these are schedules known as mixed schedules in which two or more schedule components are alternated in random manner, but are not exteroceptively cued. When exteroceptive cues are provided the schedules are called multiple schedules (Ferster and Skinner, 1957).

Ferster and Skinner (1957), for example, described a mixed fixed ratio schedule in which a short FR is randomly alternated with a long FR (e.g., Mix FR 30 FR 180). After repeated exposure to such a schedule pigeons were observed to pause, while responding on the long FR, at a point approximating the number of responses required by the short FR. In view of the lack of exteroceptive cues Ferster and Skinner argued that such pauses were occasioned by stimuli arising from the bird's own behavior.

On the basis of Ferster and Skinner's (1957) account of Mix FR schedule performance it became apparent that number of responses emitted in FR runs could be conceived as a stimulus. Mechner (1958a), concerned with the concept of "internal cohesion" of FR runs, developed a "counting" schedule in which completion of an FR on one lever was reinforced when the rat "reported" such completion by pressing a second lever. This procedure enabled Mechner to describe FR responding on the first lever in terms of the probability that a rat would terminate an FR run having already emitted N responses. Berryman, Wagman, and Keller (1960) pointed out that in Mechner's procedure cumulated frequency distributions of the run lengths on the first lever could be regarded as psychophysical functions. Platt and Senkowski (1970) used a modification of Mechner's procedure to obtain such psychophysical functions with the intent of further describing the role of number of responses as a stimulus. In their procedure completed FR runs were "reported" by the interruption of a photo beam across the food hopper rather than by a response on a second lever. Platt and Senkowski found that both means and standard deviations of the run length distributions at different FR requirements were linear increasing functions of the FR requirement. Thus the linear generalizations of Weber's Law ($\Delta I = KI + C$) was used to describe the relation of the standard deviations to the means.

Ferster and Skinner (1957) also described a mixed schedule in which FR reinforced periods were alternated randomly with periods of extinction (Mix FR Ext). As with all mixed schedules no exteroceptive stimuli were correlated with either component. The end of an extinction period was signalled by an arbitrary event, e.g., the lights in the experimental chamber were turned out for three seconds. The end of an FR reinforcement was, of course, signalled by the delivery of reinforcement. As with other types of Mix FR schedules it was assumed that in order to determine which contingency was in effect an animal would normally emit the number or responses called for by the FR. The presentation of reinforcement indicated that the period had been a reinforcement period, while no reinforcement indicated that extinction was in effect. Animals were presumed to discriminate extinction on the basis of stimuli arising from their own lever pressing behavior.

Although steady state responding on Mix FR Ext schedules has not been investigated thoroughly parametrically, some typical performances have been described by Ferster and Skinner (1957) and by Weissman (1960). After prolonged exposure to the schedule, a pattern of responding emerged in which, following a non-reinforced run, the animal stopped responding until the end of the extinction period was signalled.

The pattern of responding generated by Mix FR Ext schedules resembled the kind of discriminated extinction reported by Bullock and Smith (1953) where animals were exposed to daily sessions in which the first

40 lever presses were each reinforced and in which the 41st response initiated a one hour extinction period. The number of responses emitted during extinction decreased to a limiting value by the tenth session. The Bullock and Smith procedure represented a prototypical Mix FR Ext schedule and was interpreted as demonstrating the discrimination of extinction based upon response-correlated stimulation.

Further evidence for the development of discrimination based on response correlated stimuli was presented by Pribram (1958). Pribram's procedure also represented a prototypical mixed schedule in that FR 40 and FI 4 minute components were alternated in the absence of correlated exteroceptive stimuli. Differential rates of responding in the two components (i.e., high rates in FR 40 and lower rates in FI 4 minutes) demonstrated the discrimination. Interestingly, Pribram reported that normal monkeys and monkeys with inferetemporal lesions showed differential responding on the schedule while monkeys with anterofrontal lesions did not.

Thompson (1963a, 1963b) studied the effects of caudate lesions in monkeys on behavior maintained by Mix FR Ext schedules. He observed that lesioned animals typically made an initial run in extinction, paused, but then made additional runs in the same extinction period with greater frequency than did control subjects. Such an observation might well have tempted characterization as a failure of inhibition, inability to switch from one behavior to another, or a short term memory deficit. In any event, Thompson pointed out that the behavior generated by Mix FR Ext schedules fell into the class of behaviors typically disturbed by caudate lesions. This was not surprising since the Mix FR Ext schedule could be presumed to require both the discrimination of extinction without the aid of exteroceptive stimuli, and the occasioning of other behavior (i.e., not lever pressing).

The Present Study

The Mix FR Ext schedule thus presented itself as a procedure by which to describe, in both operational and functional terms, the effects of caudate nucleus lesions in rats. Furthermore, the schedule could be programmed on a single lever. Caudate lesions in the rat necessarily involve damage to internal capsular fibers of passage and can produce motor impairment independent of caudate destruction. It was thus considered desirable to employ a schedule which, in addition to the inclusion of an extinction component and the creation of a situation in which extinction would be discriminated on the basis of response produced stimuli, did not involve locomotion between two or more levers.

The study comprised two experiments. Experiment I investigated post-operative acquisition and steady state characteristics of responding at four levels of the FR requirement: FR 1, 4, 8, and 16. Experiment II compared pre-operative performance with post-operative retention at one level of the FR requirement, FR 1.

On the basis of pilot work it was decided to employ a differential reinforcement of other behavior (DRO) contingency for the non-reinforced component of the mixed schedule rather than simple extinction. DRO involves the presentation of a stimulus contingent upon the emission of "other" behavior defined as an interval of time during which no responses, i.e., lever presses, occur. This contingency was instrumented in order to minimize the possibility of adventitious reinforcement during the non-reinforced component of the mixed schedule as well as to place a premium on not responding once an initial run had been emitted.

As the literature showed the caudate nucleus of some species to be functionally heterogeneous, it was decided to include two caudate groups, one with anterior lesions and another with posterior lesions, rather than a single caudate group and a non-caudate lesioned control (Divac, 1968b; Rosvold, 1968).

In addition to providing a further description of caudate lesion effects, the data to be obtained from an unoperated normal control group were intended to provide a needed parametric study of behavior maintained by Mix FR DRO schedules, and an extension to mixed schedules of Boren's (1961) study of resistance to extinction as a function of the size of the fixed ratio.

EXPERIMENT I

Method

Subjects. Twenty-two experimentally naive, male, Long Evans hooded rats, approximately 60 days old at time of delivery, were obtained from Rockland Farms, Inc., Gilbertsville, Pennsylvania.

The animals were individually housed in stainless steel drawer type cages measuring approximately 17.5 cm wide by 24.5 cm deep by 17.5 cm high. The cages were located in a small room measuring approximately 1.8 by 1.8 meters, illuminated by a frosted 60 watt light bulb. The light-dark cycle in the room was not strictly controlled. The light was turned on at approximately 8:00 a.m., and turned off from between 5:00 and 7:00 p.m., depending upon the schedules of other experimenters. Temperature in the room varied between 70 and 80 degrees Fahrenheit throughout the course of the study.

Animals were maintained on a diet of Purina Laboratory Pellets for rodents. While free feeding, food and water were available at all times.

Free feeding weights were determined when the animals were approximately 150 days old, by weighing each animal for seven consecutive days and then taking the mean of the last three days. All animals were then placed on food and water deprivation and reduced to 80% of their free feeding weights. At the onset of deprivation all food and water were removed from the cages for a period of 48 hours. Each rat was thereafter permitted seven grams of laboratory chow and two hours' access to a water bottle each day until 80% of weight was attained. During this time feeding occurred at approximately 8:00 a.m. each day.

When the 80% levels were reached, the rats were fed from one to four pellets of lab chow daily according to individual deviations from 80% weight. This regimen was maintained throughout the experiment except for a period of one week prior to surgery and during the post-operative recovery period. At the end of the recovery period free feeding weights were determined a second time.

Of the 22 rats initially received, two failed to acquire bar pressing during the first session of shaping and were discarded. Of the 20 rats which acquired bar pressing, two contracted respiratory infections and were discarded. Of the remaining 18, seven were assigned to the anterior caudate group (A), six to the posterior caudate group (P), and five to a normal control group (N). Animals in the normal control group did not undergo surgery of any kind. Three rats in group A and one rat in group P failed to recover from surgery.

An attempt was made to assign rats to groups on the basis of bar pressing rates during the final session of pre-operative training on simple FR 1, such that mean rates for the three groups would be approximately equal. Given that four rats failed to survive surgery this was only partly accomplished. Of the animals which recovered from surgery, four were assigned to group A, five to group P, and five to group N. Of these, one rat in group A died during testing on FR 16 (see Procedure), and one rat in group N died while activity measures were being taken, both of respiratory infection.

Apparatus. Training was carried out in three identical commercially manufactured operant conditioning chambers (Model 1111-L, Grason Stadler Co., Concord, Mass.). As the procedure required only one lever manipulandum in each chamber, the lever nearest the door of each chamber was removed. Aluminum plates were bolted in place over the emptied slots.

The space within each chamber measured approximately 28.0 cm high by 30.0 cm wide by 30.5 cm deep and was lighted by a 14 volt 0.2 ampere house light (Westinghouse No. 1815) located in the upper left corner of the chamber approximately 25.0 cm above the grid floor and 14.0 cm to the left of the dipper. An additional light (28 volt 0.04 ampere, Westinghouse No. 1819) was installed through a hole just above the dipper, protruding into the space above the dipper approximately 0.5 cm. The lever manipulanda were Gerbrands model G6312 rat levers (Ralph Gerbrands Co., Arlington, Mass.) which measured approximately 1.3 cm thick by 5.0 cm wide and protruded into the experimental space approximately 1.5 cm. Each lever was weighted to require a force of approximately 20.0 grams through a downward excursion of approximately 0.2 cm to activate an associated microswitch.

Each chamber contained a Gerbrands Liquid Dipper (Model GS-RH 24 VDC) which delivered a 0.1 cc mixture of one part sweetened condensed milk to one part water.

Each chamber also contained a three-inch speaker with an impedance of 45 ohms through which white noise was delivered by means of a single Grason Stadler model 901 noise generator. A ventilator fan in each chamber provided additional noise. With both fan and noise generator in operation, sound pressure level at a point approximately 3.0 cm directly in front of the dipper was measured at 65 decibels re .00002 Newtons per meter squared using the C scale on a General Radio Corporation type 1551 B sound level meter.

Each chamber was housed within a Grason Stadler model 1101 research chest which provided relative isolation from extraneous visual and auditory stimulation.

The three research chests were stacked, each on a separate wooden shelf, one above the other in a small experimental cubicle. Each shelf was bracketed separately to the wall of the cubicle in order to minimize the spread of vibratory stimulation from one chest to another. The door to the experimental cubicle was kept closed at all times, except when transporting animals.

Programming and control apparatus were housed in a hallway adjacent to the experimental cubicle. A hole chiseled in the wall separating the cubicle from the hall permitted the passage of connecting cables. This hole was stuffed with pieces of sponge rubber to aid in acoustic separation.

The programming and control apparatus consisted of conventional electromechanical relays and timers. Stepping switches (two bank 33 position) were used to program the alternation of reinforcement and extinction periods on the Mix FR DRO schedule. Responses were recorded on Gerbrands model C-3 cumulative recorders and on Scientific Prototype model 4084J serial printing counters (Scientific Prototype, New York, N. Y.). The printing counters were also used to record the latency of the first response in each reinforcement period being driven by Scientific Prototype model SS 4048J synchronous clocks at 10 pulses per second.

Data were additionally recorded on Grason Stadler model E3700 digital counters.

Activity measures were obtained using three tilt boxes designed to conform approximately to specifications described in Campbell and Lynch (1969). They were constructed of wire mesh cages approximately 36.8 cm long by 17.8 cm high and 17.8 cm wide and covered by hardware cloth. Each box was mounted on a central axle 0.8 cm in diameter and arranged so that movement of a rat from one side to the other would operate a microswitch mounted at one end of the box. The maximum possible excursion from the horizontal at either end of the box was limited to 0.3 cm. The microswitch was mounted on the same plywood base as the central axle, such that the arm of the microswitch protruded beneath the end of the cage by approximately 1.3 cm. The central axle was mounted so as to move freely through holes drilled in two L-shaped braces, one mounted at either side of the box. The floor of the box when horizontal was approximately 7.6 cm above the surface of the plywood platform. A third smaller, L-shaped brace was mounted under the box at the end opposite the microswitch to control the downward excursion of the box at the end. Only one pole of the STDP microswitch was connected through a pulse former to a counter, such that having once closed the microswitch, a rat had to cross to the side opposite the switch and then return in order to register another count. A test rat was placed in each box for a period of approximately one half hour during which times E attempted manually to tilt the box in such a way as to make the microswitch "bounce." E was not able to produce a "bounce" when switch closure occurred with a rat in the box.

Each tilt box was housed in a Grason Stadler chest model E3125A-3 containing one 14 volt 0.2 ampere light (Westinghouse No. 1815) housed directly over the center of each box. These chests were placed, each on a separate shelf and one above the other at the end of a small passageway in which several racks of relay programming equipment were maintained. Some of this apparatus was in operation during the period in which activity measures were taken. The passageway had three large windows, the shades of which were kept drawn at all times. In order to reduce further the amount of ambient light in the chests, a sheet of opaque material was taped over the window of each chest.

Preliminary training. Prior to surgery all animals underwent training consisting of two daily half-hour sessions of adaptation to the operant chambers, one day of magazine training and shaping, and six daily sessions on FR 1 (i.e., one bar press per reinforcement) of 34 reinforcements each. During the second adaptation session each rat was fed approximately 100 ml of the sweetened condensed milk mixture in order to accustom it to eating in the apparatus. Magazine training and shaping were carried out in a single session.

Water consumption. Measures of water consumption were taken for five days immediately preceding surgery and for eight days following surgery. Post-operative measurement of water consumption was discontinued after the eighth day in the interests of the rat's survival (see Results, p. 51). To accomplish this, the rats' regular water bottles were removed from their cages and replaced with calibrated drinking tubes. Tubes were filled with 100 milliliters of water and placed on the cages as a part of the normal animal care rituals each morning. On the following morning the tubes were removed and the amount of water remaining in each tube was recorded. The tube was then rinsed, refilled and replaced on the cage for the next 24-hour period. No attempt was made to estimate spillage.

Surgery. Clean stereotaxic techniques were employed using a small animal stereotaxic frame (David Kopf, Inc., Tujunga, Calif.). Rats were first injected with Taractan (Hoffman-LaRoche brand chlorprothixene) 12.5 mg per kg. of body weight and then anesthetized with sodium pentobarbital (50 mg per kg. of body weight). All injections were intraperitoneal. Taractan was used to potentiate the effects of the Nembutal and minimize respiratory congestion (Rye and Elder, 1966). During surgery ether was administered by means of a nose cone when necessary.

Animals were mounted in the stereotaxic frame with the tooth bar set 5 mm above zero. A longitudinal incision was made in the scalp and the dermal, muscle, and fascia tissue reflected. A 1 mm diameter dental burr was used to drill holes at the appropriate cranial sites. A Radionics RFG 4 radio frequency lesion maker (David Kopf, Inc.) was used to make the lesions with a Radionics thermistor probe electrode which permitted the monitoring of temperature at the electrode tip. The stainless steel electrode was 0.7 mm in diameter and was insulated with a nylon coating up to within 1.5 mm of the tip. Stereotaxic coordinates were determined using the Pellegrino and Cushman (1967) atlas. Anterior caudate lesions were directed 2.8 mm anterior to the bregma and 6.0 mm below the cranial surface. Posterior caudate lesions were directed 1.0 mm anterior to the bregma and 4.8 mm below the surface of the cranium. All lesions were directed 3.0 mm lateral to either side of the bregma. The electrode was lowered to the desired coordinates and current increased gradually until the metered temperature showed 60 degrees centigrade. Once this temperature was reflected on the meter, current was continued for approximately one minute and then turned off. The electrode was then withdrawn, and the scalp closed and sutured. The same electrode was used for all animals and was swabbed with hydrogen peroxide between operations. Each animal was injected intramuscularly with 150,000 units of bicillin (benzathene penicillin G) to prevent infection.

Post-operative acquisition of responding under Mix FR DRO schedules. Following recovery from surgery and return to the newly calculated 80% weight levels, each animal received 15 daily sessions on FR 1 of 34 dipper presentations each followed by 30 sessions on the Mix FR 1 DRO schedule. Following the 30 sessions on the mixed schedule each rat was returned to FR 1 and brought gradually to FR 4, the next appropriate FR level. This procedure was repeated for each level of the FR requirement (FR 1, 4, 8, and 16) such that animals received 15 sessions at each FR without intrusions of DRO periods followed by 30 sessions of Mix FR DRO. Following the 30 sessions on Mix FR 16 DRO, animals were returned to FR 1 and run a second time at that level, i.e., 15 sessions of FR 1 followed by 30 of Mix FR 1 DRO.

The Mix FR DRO schedule consisted of 66 periods, 33 each of FR reinforcement and DRO alternated according to a Gellerman (1933) series modified to conform to the characteristics of the stepping switch (see Appendix I, Table A for sequence). In addition to the 33 dipper presentations delivered on the FR, each session began with the first response producing a dipper presentation.

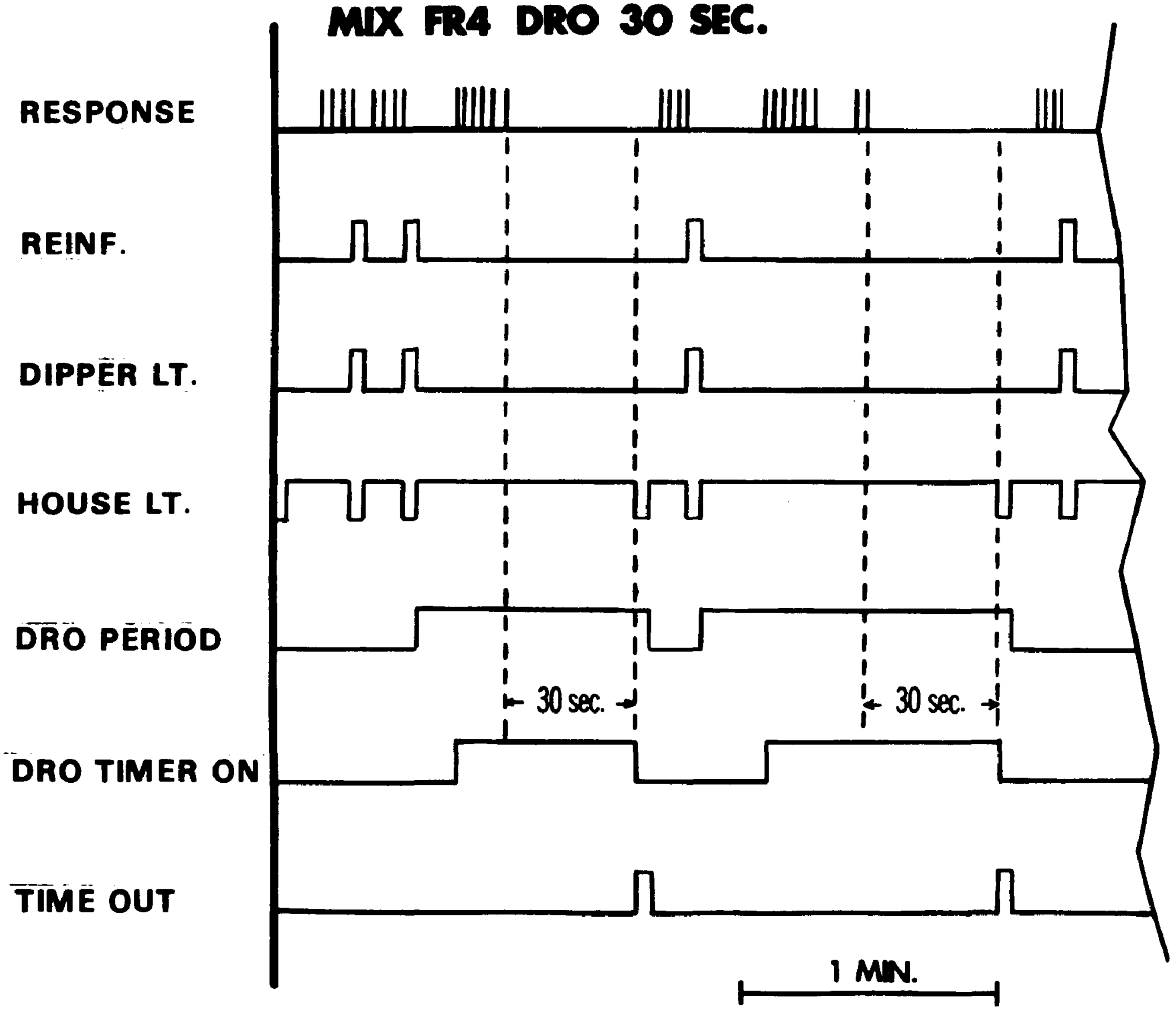
During testing at the FR 8 and FR 16 levels some rats exhibited relatively long interresponse times. In order to maintain the daily running schedule it was necessary to modify the session length criteria such that a session could be terminated whenever a rat completed at least 16 DRO periods and two hours had elapsed.

The DRO contingency was such that the first response in each DRO period initiated a 30 second timer which recycled with each subsequent bar press unless permiteed to time out and end the DRO period. Once in a DRO period, therefore, an animal had to respond at least once and than pause for at least 30 seconds to terminate the DRO.

The end of each DRO period was followed by a three-second time out during which the house light was extinguished and a DRO three-second contingency was in effect. The three-second DRO during the time out was employed to minimize further the probability that responding during nonreinforced segments of the schedule would be adventitiously reinforced. Figure 1 shows an event marker diagram of the Mix FR DRO 30 second schedule.

Activity measurement. Following completion of testing on Mix FR DRO schedules, all rats were assigned to tilt boxes such that an approximately equal number of rats from each group was run in each box, and only one rat from a group was run at a time. This assignment was only partially accomplished, since by the time activity measures were taken the sizes of the groups were unequal. Rats were run in the tilt boxes for six consecutive days, with the first day considered a "warm up" period. Each "day" consisted of approximately 12 hours with the box light on and 12 hours with the light off. The day began at about noon with the beginning of a light period. Rats were placed in the boxes at the beginning of the light period, at which time they were given one to four lab chow pellets according to weight and two hours' access to a water bottle attached to the cage directly over the central axis so as not to bias the tilt. During a 24-hour light-dark cycle the animals were disturbed only twice: once at the beginning of the light cycle for weighing and feeding, and two hours later when the water bottles were removed. Activity was measured in terms of the number of cage tilts (i.e., switch closures) per hour.

FIG. 1. Diagram of the Mixed Fixed Ratio DRO schedule with ratio requirement set at four responses. In all cases an upward excursion of the event marker indicates an "on" condition. For example, following the emission of the first four responses (top line), reinforcement (3 seconds'access to liquid dipper) is delivered, dipper light is on for three seconds, and house light is off for three seconds. Following the second delivery of reinforcement extinction is in effect (third line from bottom). The first response in extinction initiates the DRO timer (second line from the bottom), which resets with each of the six responses shown and does not time out until 30 seconds of no responding have elapsed. When the DRO 30 second contingency is satisfied, the three second time out condition is initiated (bottom line) during which the house light is again off and a DRO 3 second is in effect.


MIX FR4 DRO 30 SEC.

Histology. Following completion of the experiment, the lesioned rats were deeply anesthetized (75 mg. sodium pentobarbitol per kg. body weight) and then perfused through the heart with 50 cc saline solution followed by 50 cc 10% formalin. Rats were then decapitated and the heads placed in 10% formalin for a minimum of two weeks. The heads were then mounted in the stereotaxic frame for the purpose of blocking the brain tissue in a plane approximately parallel to the electrode track. This was accomplished by mounting a scalpel blade in the electrode carrier and drawing it in a coronal cut by means of the micromanipulator. After blocking, the brains were removed and stored in 10% formalin for several months, after which the blocks were dehydrated in alcohol and embedded in celloidin for sectioning. Sections 50 micra thick were taken with a microtome and every other section was stained with thionin and mounted. The stained sections were then projected onto reproductions of plates from the Pellegrino and Cushman (1967) atlas and the lesion outlines sketched. Estimates of the amount of caudate tissue remaining were made by selecting five representative sketches throughout the antero-posterior extent of the lesions, placing graph paper (eight squares to the inch) over them and counting the number of squares contained within the outlines of the intact caudate nucleus. Damage to neighboring structures was noted, but no attempt was made to estimate the amount of damage to these structures.

Results

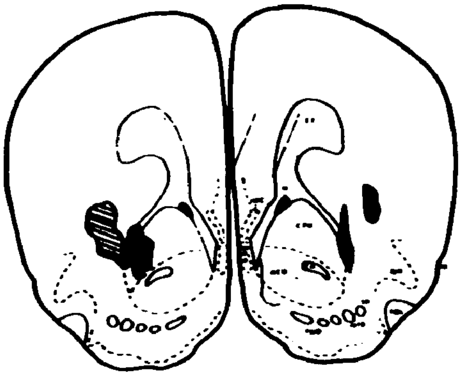
Histology. Figures 2 and 3 show composite reconstructions of caudate lesions for animals in the anterior and posterior groups respectively. Figures 4 and 5 show photographs of representative sections from each lesion. Estimates of percent of caudate tissue destroyed in

FIG. 2. Composite reconstructions of lesions in the Anterior Caudate group (N = 4) in Experiment One drawn on plates from the Pellegrino and Cushman (1967) atlas. The shaded areas represent individual lesions. The blackened areas represent the overlapping of two or more lesions. The number to the upper left of each drawing indicates millimeters anterior to bregma.

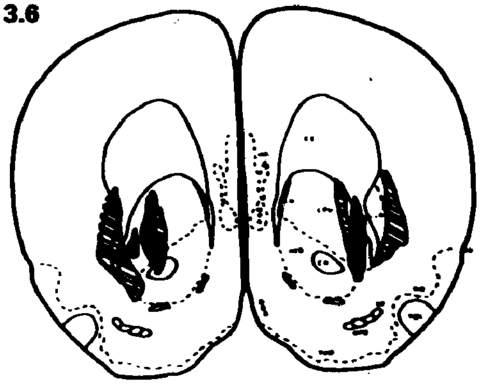
ANTERIOR

(EXPERIMENT I)

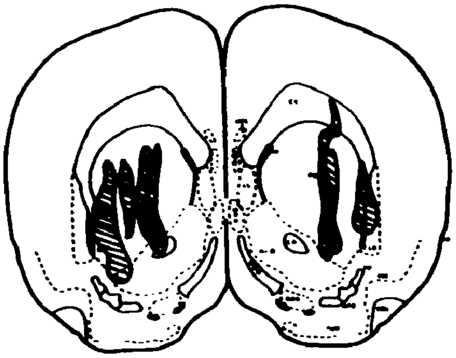
4.0



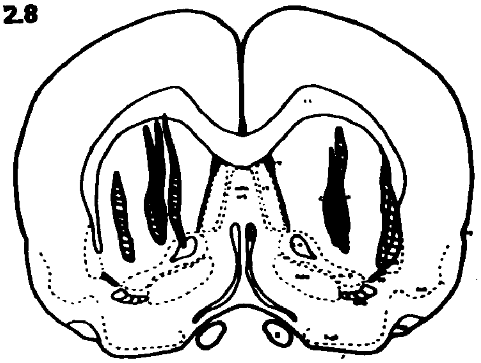
3.6



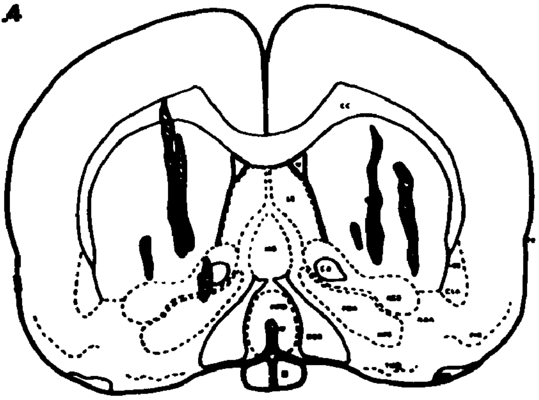
3.2



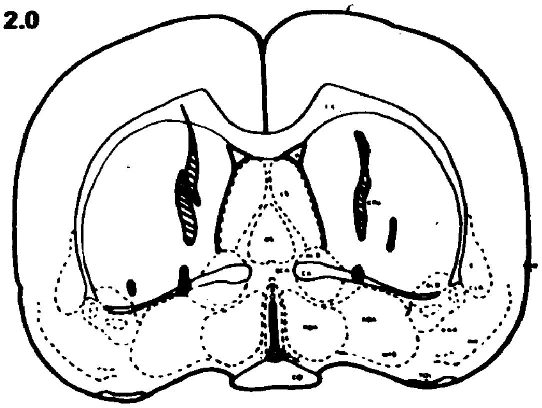
2.8



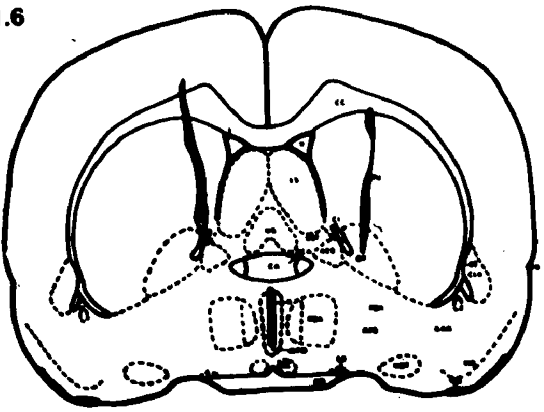
2.4



2.0



1.6



1.2

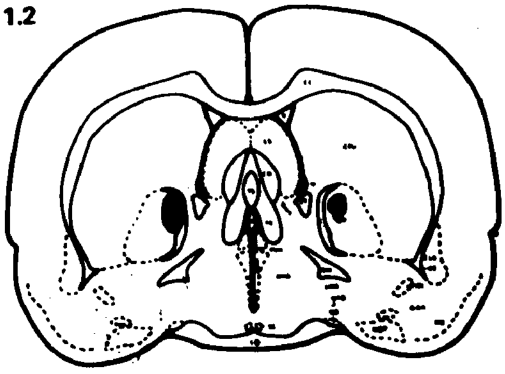


FIG. 3. Composite reconstructions of lesions in the Posterior Caudate group (N = 5) in Experiment One. Drawings were made on plates from the Pellegrino and Cushman (1967) atlas. The shaded areas represent individual lesions. The blackened areas represent the overlapping of two or more lesions. The number to the upper left of each drawing indicates millimeters anterior to bregma.

POSTERIOR
(EXPERIMENT I)

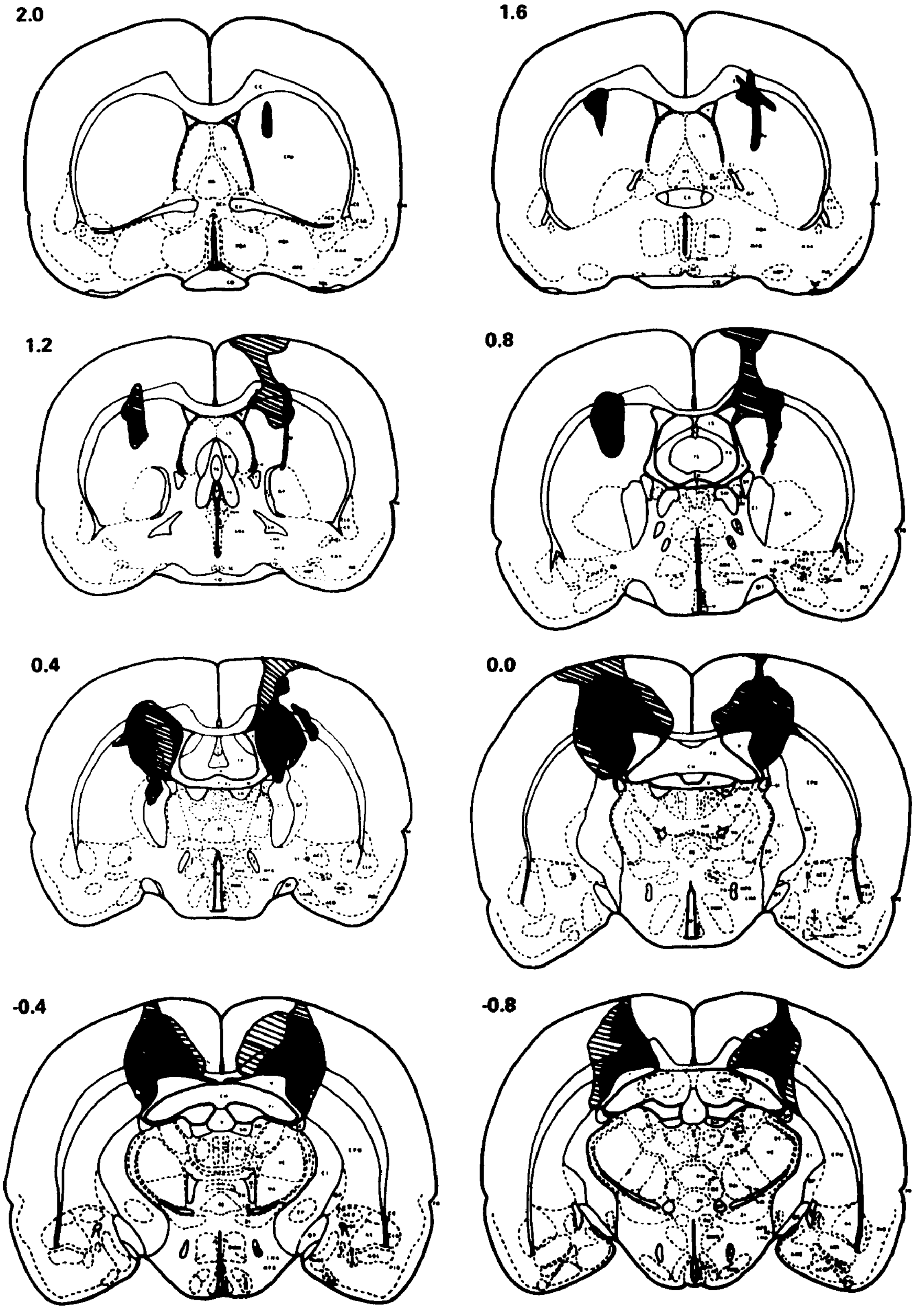
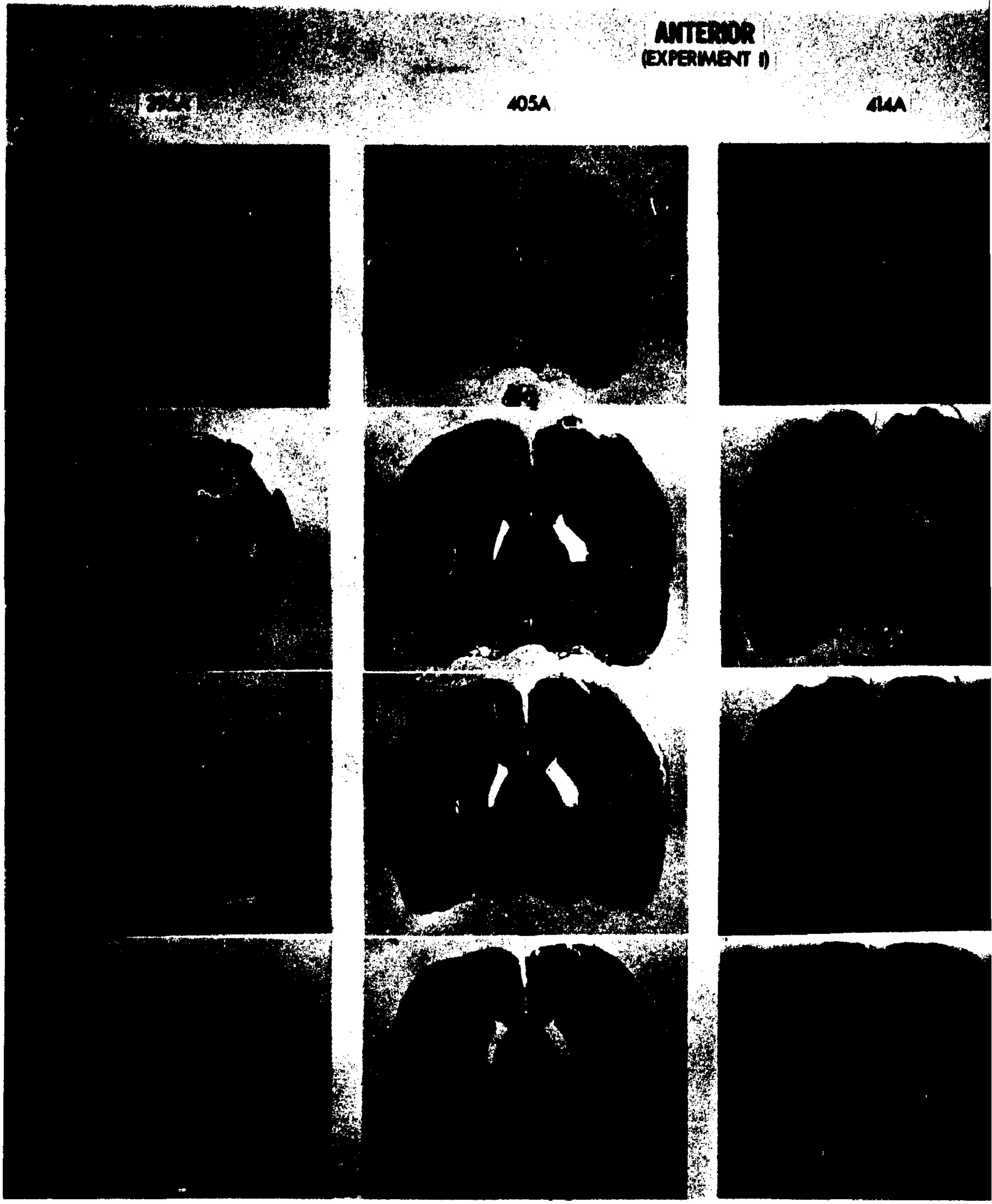


FIG. 4. Photographs of representative sections of lexions in the anterior caudate group (Experiment I).


ANTERIOR
(EXPERIMENT I)
405A
414A

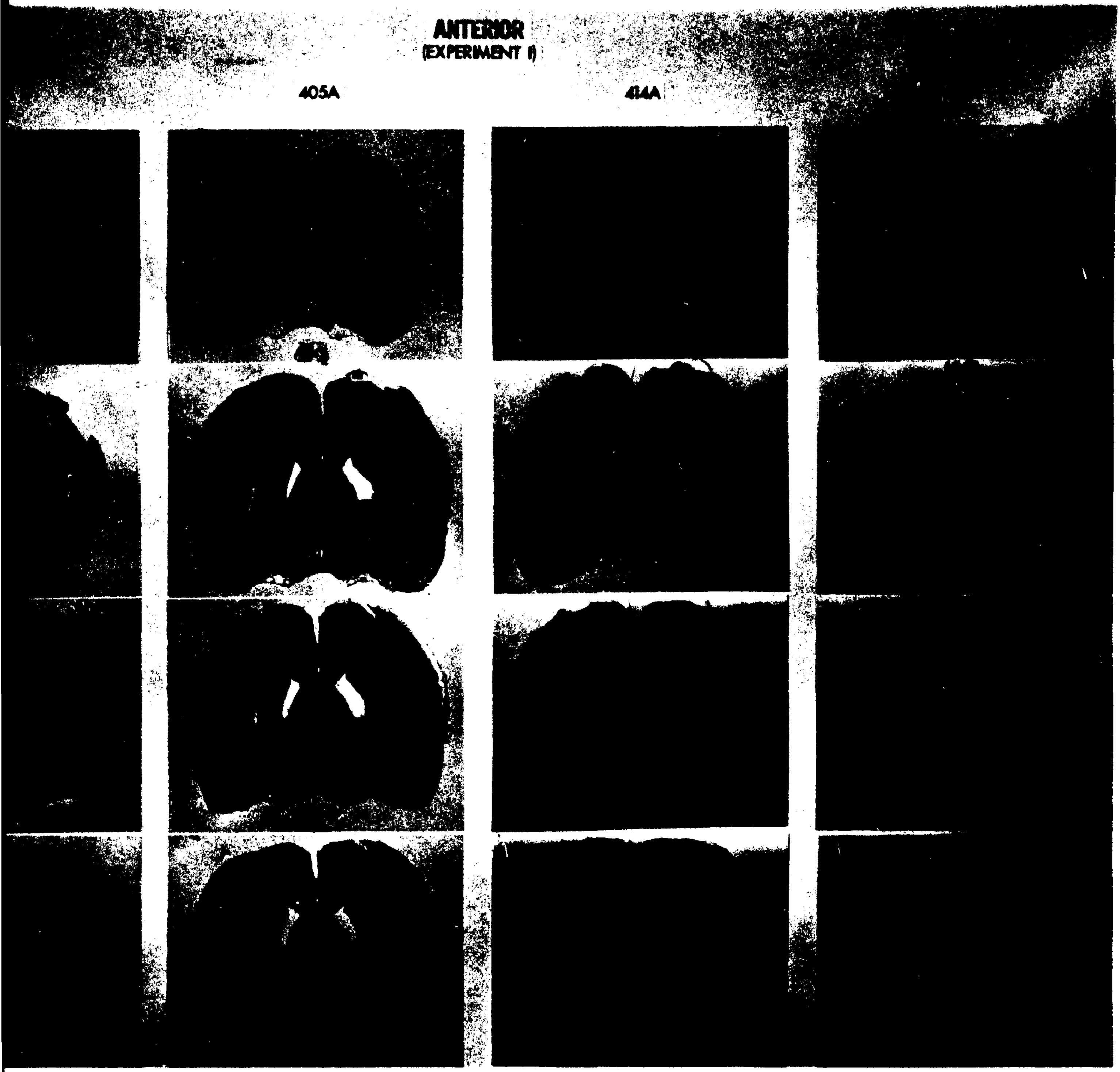
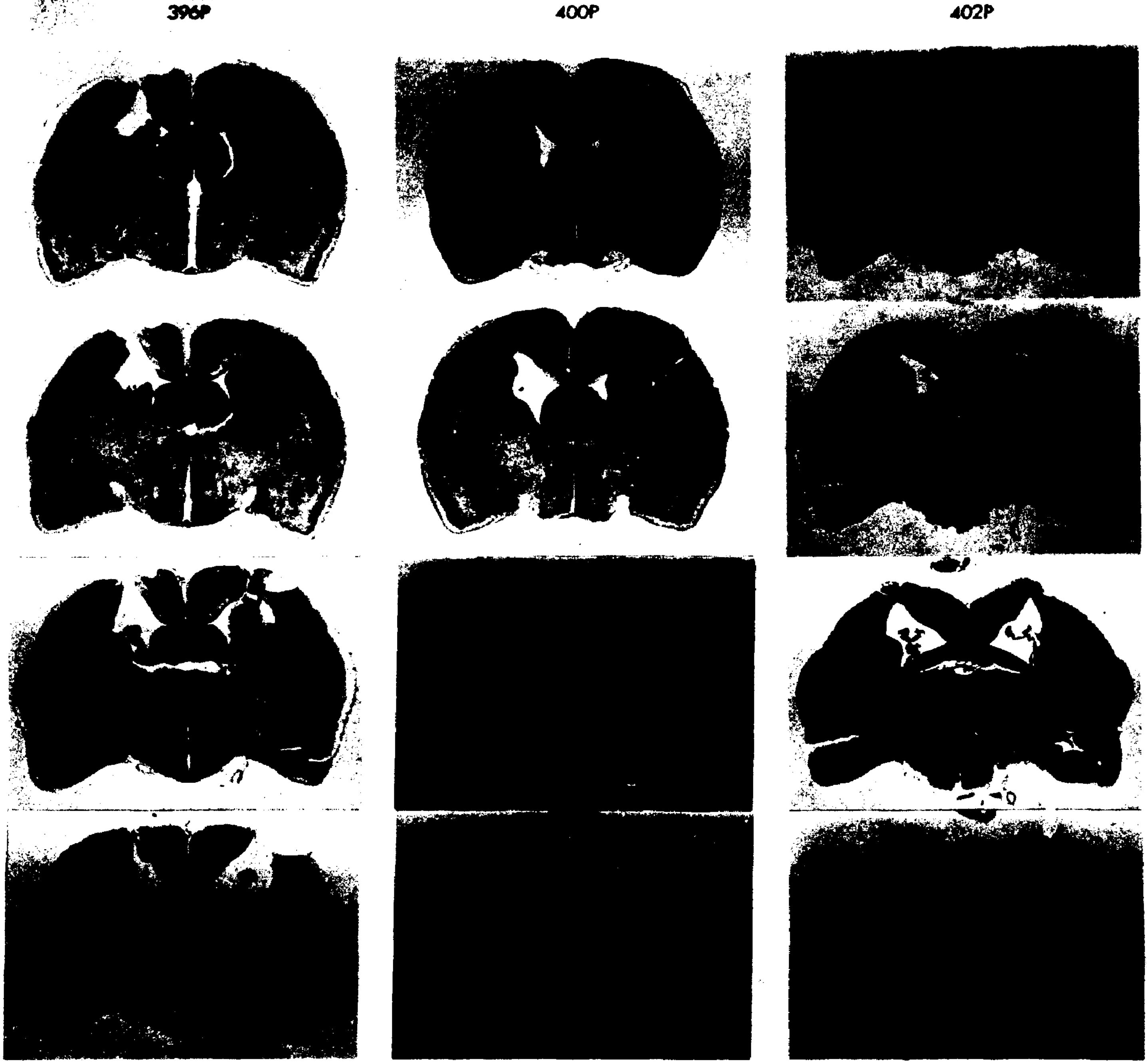
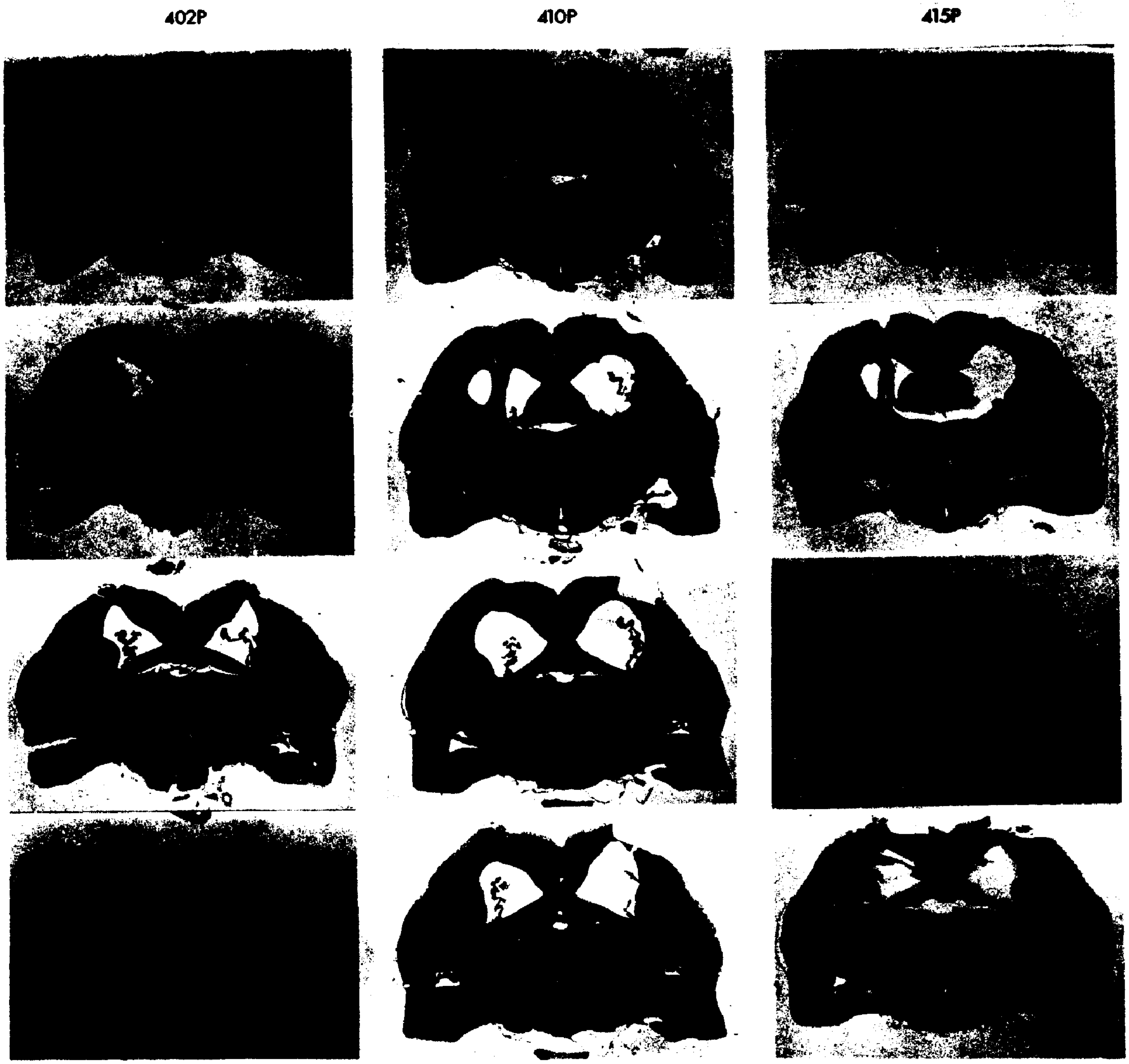

ANTERIOR
(EXPERIMENT I)
405A
414A

FIG. 5. Photographs of representative sections through the lesions of animals in the posterior caudate group (Experiment I).

POSTERIOR
(EXPERIMENT I)



POSTERIOR
(EXPERIMENT I)



the area of the lesions at five representative sections from each animal and plane of furthest anterior and posterior extent of lesions in each animal are given in Table B in Appendix I.

Examination of sections through the lesioned areas revealed dilation of the lateral ventricles in all animals. This dilation probably represented post-operative contraction of caudate tissue as described by Wolf and DiCara (1969). Estimates of percent tissue destroyed must be regarded with caution, therefore, and at best provide a basis for rank ordering.

The lesions in the posterior group were more dorsally placed than intended and damage to the corpus callosum was seen in all cases. In the cases of rats 402P, 410P, and 415P the lesions may have damaged the pia mater lining the lateral ventricles.

In the anterior group the lesions of rats 414A and 395A extended anterolaterally into the adjacent piriform cortex and claustrum. In rats 414A and 416A the lesions invaded to a very small extent the nucleus accumbens and globus pallidus.

There was an overlap in the posterior extent of anterior lesions with the anterior extent of posterior lesions (see Table B, Appendix I). For example, in rat 400P the left lesion extended 2.2 millimeters, anterior to bregma while the lesion in rat 414A extended posteriorally to 0.8 millimeters anterior to bregma. Rat 414A showed the greatest anterior-posterior extent (2.6 millimeters on either side) of either group. The median anterior-posterior extent for the anterior rats was 2.0 millimeters with a range from 1.6 to 2.6 millimeters. That for the posterior group was 1.7 millimeters with a range of 0.4 to 2.0 millimeters.

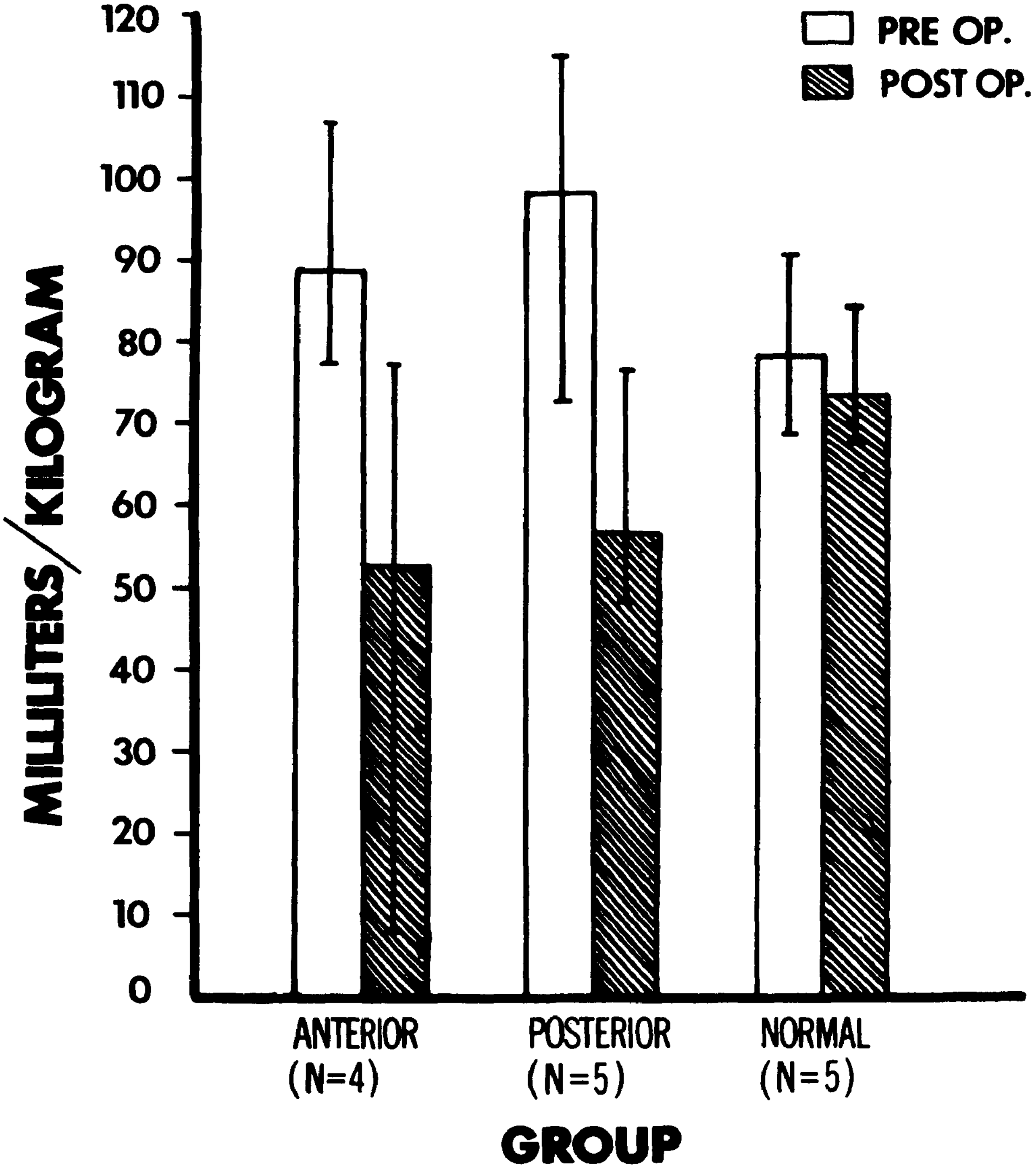
The lesions in both groups were approximately equivalent in size bilaterally within subjects with the exception of rats 395A and 400P. In the case of these two animals the appearance of greater ventricular dilation on one side suggested a larger lesion on that side. The amount of ventricular dilation may, however, have been influenced by proximity of the lesion to the ventricle as well as by lesion size. Some lesions were not bilaterally equivalent with respect to anterior-posterior placement (see Table B, Appendix I). For example, the right lesion of rat 414A was placed 0.4 mm more anterior than the left lesion. In rat 400P the left lesion extended to 2.2 anterior to bregma while the right lesion extended only to 1.6 anterior to bregma.

The lesions produced in the present experiment were small when compared with reconstructions and photographs of lesions in other studies. Based on inspection, the present lesions were approximately one-half to one-third the size of lesions reported by Whittier and Orr (1962), Chorover and Gross (1963), Gross, _et_ _al_. (1965), Unger (1966), Schmaltz and Isaacson (1968), Hansing, _et_ _al_. (1968), and Potegal (1969). The largest lesions in the present study (e.g., rat 395A) are approximately the size of the smallest lesions reported by Gross, _et_ _al_. (1965). Only one other study, Neill and Grossman (1970), reported lesions as small as those of the present study.

Water consumption. Figure 6 shows means and ranges of water consumed in milliliters per kilogram of body weight over five day periods both before and after surgery. Milliliters per kilogram consumed by individual animals over five pre-operative and eight post-operative days are shown in Figure A in Appendix I. It can be seen that water consumption decreased post-operatively for all groups. A series of *a priori* t tests revealed that the decreases for the anterior ($t = 4.42$, d.f. = 3, $p < .05$, two tail) and posterior ($t = 6.95$, d.f. = 4, $p < .05$, two tail) groups were significant while that for the normal rats ($t = 1.27$, d.f. = 4, $p > .20$, two tail) was not. An analysis of variance showed the location of lesion factor to be not significant ($F = 0.72$, d.f. = 2/11, $p > .50$).

Some non-quantitative observations of post-operative drinking behavior were worth noting. Two of the anterior rats, 395A and 414A, had difficulty drinking from the calibrated tubes which were used to measure water consumption. In the case of 395A it was observed that upon refilling and replacing the tube in the rat's home cage the animal approached the tube, but attempted to lick beneath rather than at the aperture through which water was available. Rat 395A normally made two or three such attempts before withdrawing from the tube. 395A accepted water when placed directly into its mouth with an eye dropper, however, By the end of the sixth post-operative day 395A's water intake was only 19.2 ml/kg, and it was placed back on its regular water bottle from which it appeared to drink without difficulty. The rat was thereafter permitted to continue drinking from its regular bottle and no further measures were taken of this animal.

FIG. 6. Means and ranges of amount of water consumed in milliliters per kilogram of body weight for each group over five days immediately preceding surgery and five days immediately following surgery, but not including the day of surgery. In each case the range is of the five-day means of each animal in the group.


120
110
100
90
80
70
60
50
40
30
20
10
0
MILLILITERS/KILOGRAM
PRE OP.
POST OP.
ANTERIOR (N=4)
POSTERIOR (N=5)
NORMAL (N=5)
GROUP

After the eighth post-operative day, rat 414A was also permitted to drink from its regular bottle and it also appeared to be able to drink more easily from it than from the calibrated tube. At this point all rats were returned to their regular water bottles in the interests of their continuing survival. While four out of five of the posterior rats also showed decreased post-operative drinking levels during the eight days of measurement, none of these four exhibited the difficulties which had been observed with rats 395A and 414A.

Body weight. As the post-operative decreases in water consumption shown by the two operated groups suggested possible metabolic changes the percent changes between the first and second determinations of *ad libitum* feeding weights were calculated and are shown in Table 1. It can be seen that three out of four anterior rats decreased in weight between the two determinations with rat 416A showing the greatest decrease: 9.7 percent. Only one anterior rat, 405A, showed an increase in weight. In the posterior group two rats showed increases, two showed decreases, and one animal did not change. All rats in the normal group increased in weight with rats 398N and 412N showing the greatest increases; 12.1 and 9.7 percent respectively. A non-parametric Kruskal-Wallis analysis of variance performed on the percent changes was significant ($H = 6.41$; $n = 5, 5, 4$; $p < .049$; two tail). Mann Whitney tests showed no difference between anterior and posterior groups ($U = 15$; $n = 5, 4$; $p > .548$; two tail). When anterior and posterior groups were combined they were found to differ significantly from the normals ($U = 4$; $n = 9$ & 5; $p < .05$; two tail).

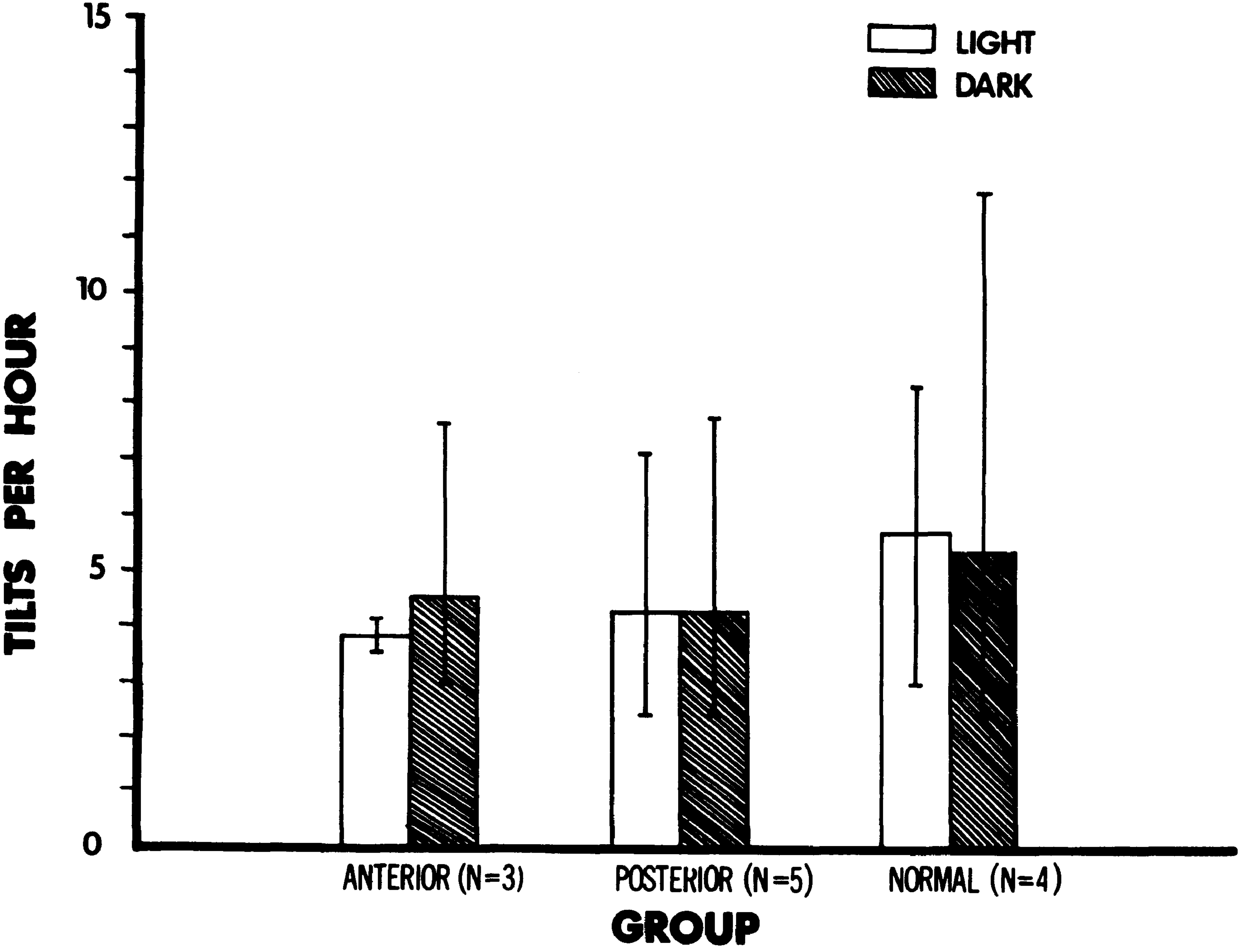
Activity. Figure 7 shows means and ranges of the number of cage tilts per hour for each group in both light and dark conditions over

TABLE 1

Percent Change in Weight Between First and Second _Ad Libitum_ Determinations, and First _Ad Libitum_ Weight in Grams

Rat and Group	First _Ad Libitum_ Weight	Percent Change
Anterior:		
395A	429	-1.9
405A	472	+3.6
414A	514	-4.1
416A	472	-9.7
Posterior:		
396P	486	0.0
400P	463	+0.4
402P	467	-0.9
410P	390	-1.0
415P	501	+1.6
Normals:		
397N	433	+3.0
398N	338	+12.1
403N	455	+2.0
407N	486	+0.8
412N	452	+9.7

FIG. 7. Means and ranges of number of tilts per hour for five days of activity observation for each group in experimental light and dark conditions. Observations were made approximately 11 months following surgery after testing on the Mix FR DRO schedules was completed. In each case the ranges shown are those of the five-day means for each animal in the group.



15
10
5
0
TILTS PER HOUR
LIGHT
DARK
ANTERIOR (N=3)
POSTERIOR (N=5)
NORMAL (N=4)
GROUP

the last five of six days following the completion of all testing on the Mix FR DRO schedules (i.e., about 11 months following surgery). Number of cage tilts per minute over days for each animal are shown in Figure B in Appendix I. The mean number of cage tilts per hour for the two operated groups was no different from that of the normal rats ($F = 0.400$; d.f. = 2/9; $p > 0.25$). There was also no difference in activity between the light and dark periods ($F = 0.02$; d.f. = 1/9; $p > 0.50$).

Analysis of final performance on Mix FR DRO schedules. In order to summarize the final performance level, the mean total number of responses per DRO period, mean number of runs per DRO period, and mean number of responses emitted per initial run in each DRO period were each averaged over the last ten sessions at each level of the ratio requirement for each animal. A run was defined as a sequence of responses separated by interresponse times (IRTs) of less than three seconds. The group means are shown as functions of the ratio in Figures 8, 9, and 10. Individual means and standard deviations over the last ten sessions at each level of the ratio are shown in Tables C, D, and E in Appendix I.

To further analyze steady state performance, median latencies of the first response in reinforcement periods following reinforcement and following DRO periods were averaged for each animal over the last ten sessions at each level of the ratio requirement. These means of the median latencies are shown in Table 2. Latencies for the FR 4 level were not available as programming did not permit recording of latencies while FR 4 data were being collected.

With respect to the lesion variable, analyses of variance showed no significant effect on mean total responses per DRO ($F = 1.39$; d.f. = 2/11; $p > 0.10$), mean number of runs per DRO ($F = 1.70$; d.f. =

FIG. 8. Mean total responses per DRO period as a function of the fixed ratio for each group. Each data point represents the group mean of the mean number of responses emitted per DRO period by each animal over the last ten sessions (sessions 21 through 30) at each level of the schedule. The means for the second determination at Mix FR 1 DRO 30 are shown separately to the right.

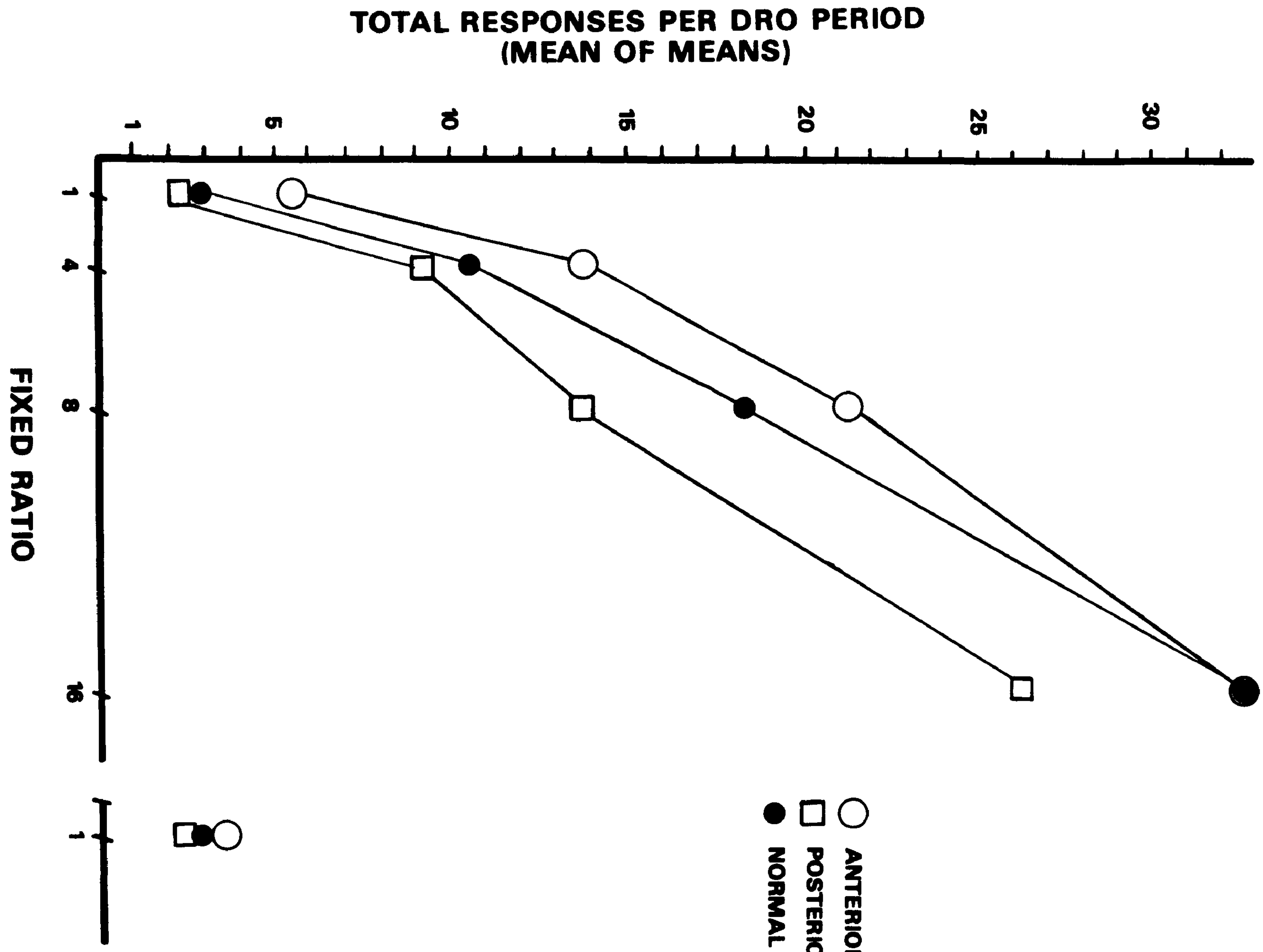

TOTAL RESPONSES PER DRO PERIOD
(MEAN OF MEANS)
1
5
10
15
20
25
30
FIXED RATIO
1
4
8
16
1
○ ANTERIOR
□ POSTERIOR
● NORMAL

FIG. 9. Mean number of runs per DRO period as a function of the fixed ratio for each group. Each data point represents the group mean of the mean number of runs emitted per DRO period by each animal over the last ten sessions (sessions 21 through 30) at each level of the schedule. The means for the second determination at Mix FR 1 DRO 30 are shown separately to the right.

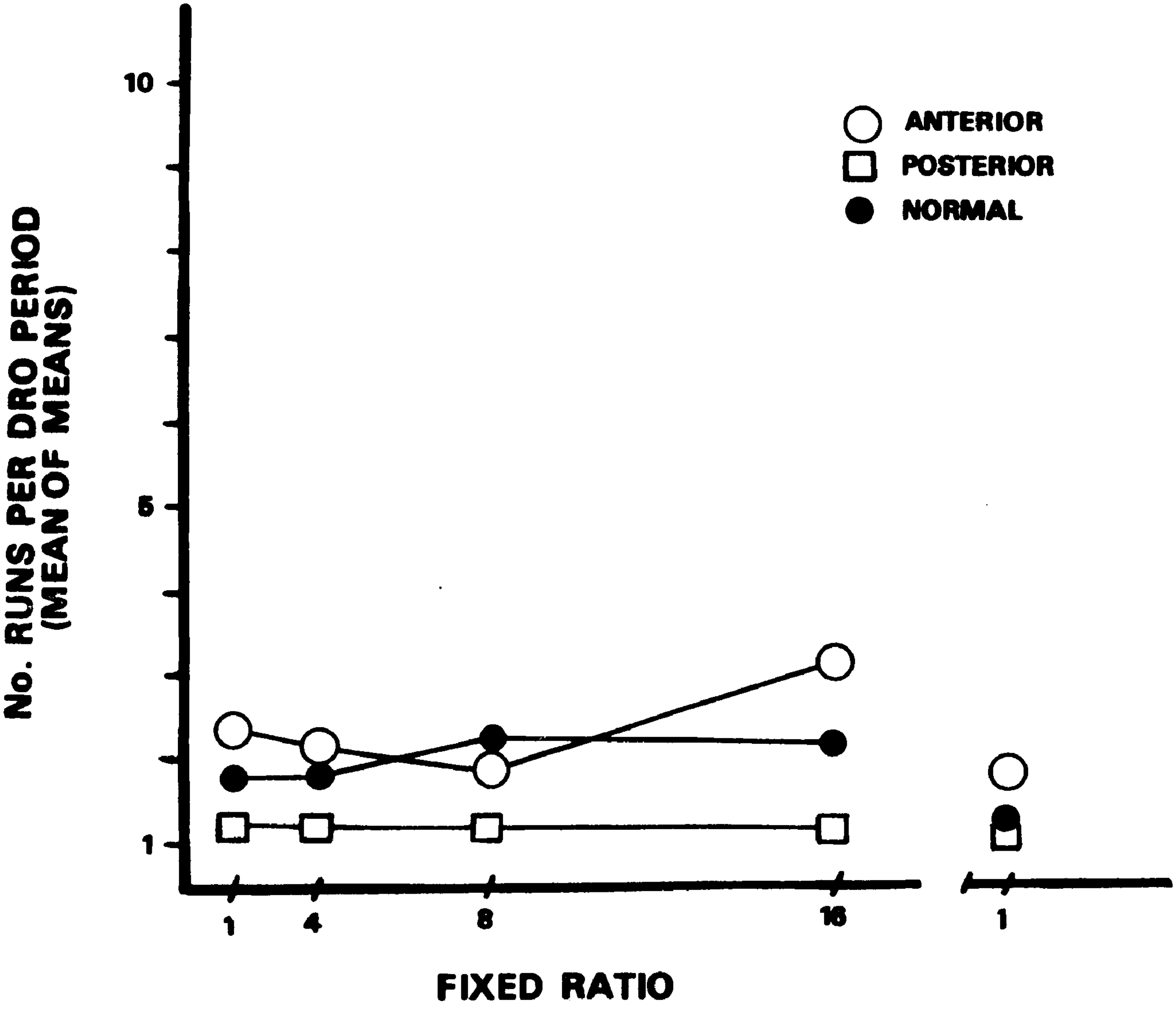
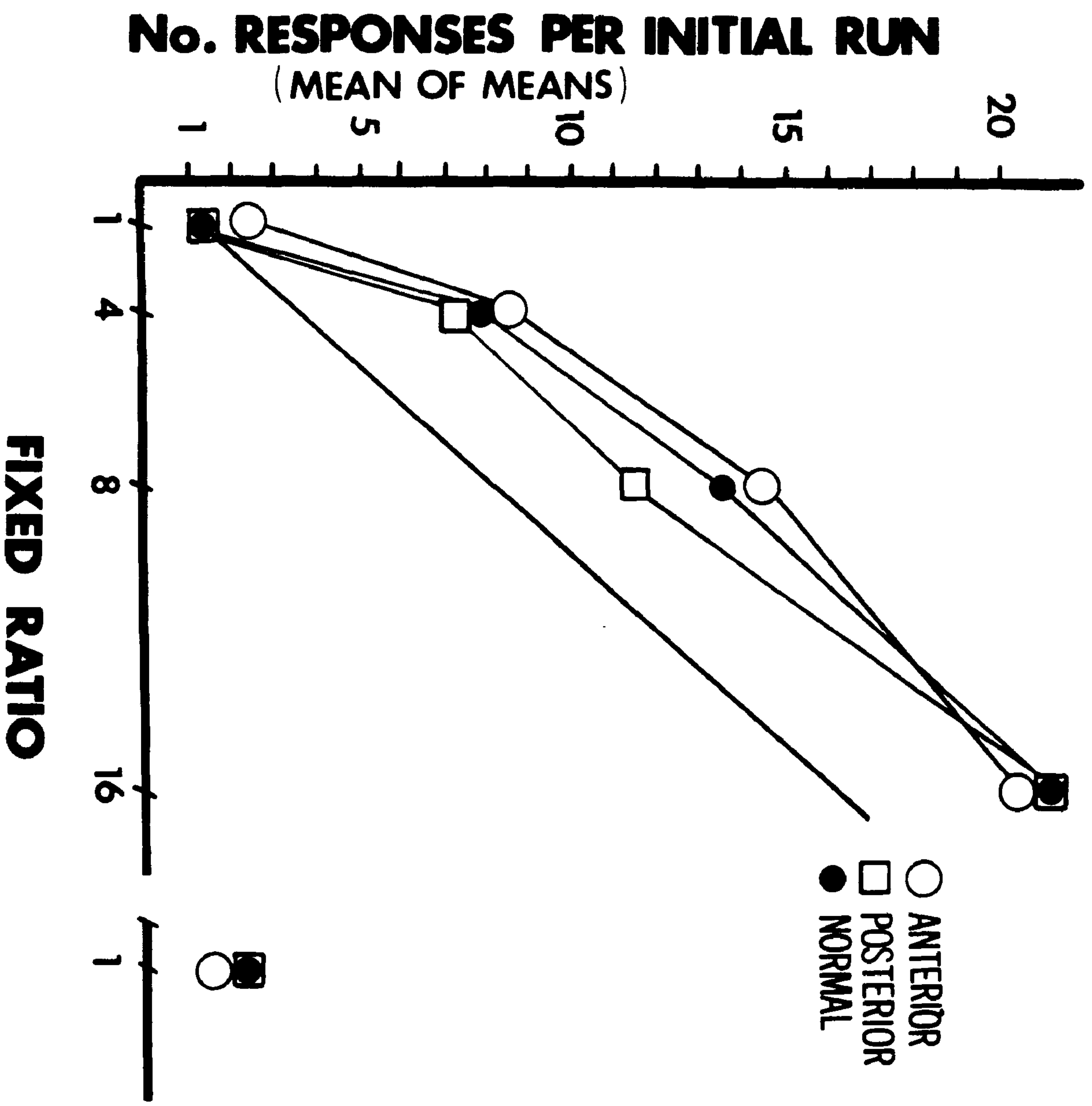

10
5
1
No. RUNS PER DRO PERIOD
(MEAN OF MEANS)
○ ANTERIOR
□ POSTERIOR
● NORMAL
1
4
8
16
1
FIXED RATIO

FIG. 10. Mean number of responses per initial run in DRO as a function of the fixed ratio for each group. Each data point represents the group mean of the mean number of responses emitted in the initial run in each DRO period by each animal over the last ten sessions (sessions 21 through 30) at each level of the schedule. The means for the second determination at Mix FR 1 DRO 30 are shown separately to the right. The solid line which does not connect data points is drawn through the values of the FR requirement measured on the ordinate to show parity.


No. RESPONSES PER INITIAL RUN
(MEAN OF MEANS)
1
5
10
15
20
FIXED RATIO
1
4
8
16
1
○ ANTERIOR
□ POSTERIOR
● NORMAL

2/11; $p > 0.10$), mean number of responses per initial run in DRO (F = 0.14; d.f. = 2/11; $p > 0.250$), or mean of median latencies of first response in reinforcement periods (F = 0.28; d.f. = 2/11; $p > 0.250$).

It can be seen from Figures 8 and 10 that for all three groups mean total responses per DRO and mean number of responses per initial run in each DRO periods were increasing functions of ratio size (F = 71.29; d.f. = 3/33; $p < 0.005$; and F = 58.15; d.f. = 3/33; $p < 0.005$; respectively). The number of runs emitted per DRO period (see Figure 9) was not found to be significantly affected by changes in ratio size (F = 0.85; d.f. = 3/33; $p > 0.250$).

That number of runs did not increase with increases in ratio size indicated that increases in the total number of responses emitted per DRO period as a function of ratio size were to be accounted for in terms of increases in the number of responses per run and not number of runs per DRO period.

It can also be seen from Figure 10 that mean number of responses emitted per initial run in DRO exceeded the number of responses required by the ratio. Inspection of Table E in the appendix, reveals two exceptions at the FR 16 level, however; rat 405A had a mean of only 2.6 responses per initial run and rat 403N a mean of only 15.7.

The durations of the median latencies of the first response in reinforcement periods (see Table 2) were also found to increase with changes in ratio size (F = 5.77; d.f. = 2/22; $p < .025$). Further inspection of Table 2 reveals several cases (e.g., rats 414A, 402P, and 407N) in which median latency did not increase when the ratio increased. Furthermore, if linearity is assumed, the median latencies in some cases increased disproportionately at the FR 16 level compared to increases at the FR 4

TABLE 2

Median Latency (Seconds) of First Response in Each Reinforcement Period Over the Last Ten Sessions

Rat and Group	FR 1 (1st)		FR 1 (2nd)		FR 8		FR 16	
	Post Reinf.	Post DRO	Post Reinf.	Post DRO	Post Reinf.	Post DRO	Post Reinf.	Post DRO
Anterior								
395	5	0	4	2	6	1	26	6
405	8	3	3	1	5	2	74	17
414	19	6	6	5	5	2	4	2
416	3	1	-	-	8	8	-	-
Mean	8.8	2.5	4.3	2.6	6.0	3.0	34.7	8.3
Posterior								
396	5	1	4	6	7	15	9	20
400	2	1	4	0	4	1	45	1
402	1	1	1	0	4	0	2	0
410	2	1	3	3	5	23	11	43
415	2	1	1	0	1	1	3	2
Mean	2.4	1.0	2.6	1.8	4.2	8.0	14.0	13.4
Normal								
397	1	0	1	0	1	1	7	3
398	1	1	1	0	2	0	3	2
403	8	0	5	1	8	1	32	12
407	2	1	1	2	2	1	2	18
412	1	1	0	1	2	14	2	15
Mean	2.6	0.6	1.6	0.8	3.0	3.4	9.2	10.0

or FR 8 levels (e.g., rats 405A, 400P, and 403N). From inspection of cumulative records and functions showing mean number of responses per initial run over sessions (see next section) it was apparent that those rats which showed the greatest increases in median latency at the FR 16 level also emitted a greater proportion of initial runs containing fewer responses than the FR requirement. In these cases it was assumed that the increased latencies were indicative of a breakdown in schedule control (i.e., ratio strain) rather than a simple consequence of increases in ratio size (Felton and Lyon, 1966).

It was also found that latencies following reinforcement were longer than those following DRO periods ($F = 5.93$; d.f. = 1/11; $p < 0.05$). Inspection of Table 2 showed this to be true for the anterior group at all levels of the ratio and for the posterior and normal groups at the FR 1 level only. None of the interactions, however, were found to be significant.

Planned *a priori* t tests were used to estimate the significance of differences in group performance between the first and second determinations at the FR 1 level. When the two tailed confidence level was corrected for the number of independent comparisons no significant differences were found on any of the dependent variables.

As the analyses of the last ten sessions at each ratio level failed to reveal any significant lesion effect it was decided to examine performances from early sessions. Mean total responses, number of runs, and mean number of responses per initial run per DRO period were each averaged over the first five sessions for each animal at each level of the schedule. These means are shown in Tables F, G, and H in Appendix I. Analyses of variance again found the lesion factor to be not significant.

Analysis of initial runs. Since performance on the Mix FR DRO schedule was presumed to involve the discrimination of number of responses as a stimulus, distributions of the number of responses emitted per initial run in DRO periods over the last ten sessions at the FR 4, 8, and 16 levels were analyzed to determine whether or not they could be used to provide discriminability functions. Relative frequency distributions of the number of responses emitted in each initial run are shown in Figures 11, 12, and 13. With the exception of those for rats 405A and 414A at the FR 1 level, these distributions appeared positively skewed. Distributions became flatter as the FR requirement was increased, reflecting increased variability.

Inspection of the distributions at the FR 4 level showed that the anterior rats emitted a higher proportion of runs containing only a single response than did either the posterior or normal animals. For the rats in the anterior group the proportion of runs containing a single response at the FR 4 level ranged from 3 to 16 percent. The proportion of runs containing a single response failed to exceed 3 percent for any animal in either the posterior or normal group. This difference did not appear to extend to short runs (i.e., runs containing fewer responses than the ratio requirement) generally, and held only at the FR 4 level.

These distributions may be regarded as psychophysical functions in which case the standard deviations (see Table E, Appendix I) may be considered estimates of the difference threshold for number of responses as a stimulus (Platt and Senkowski, 1970). In this regard it was noted that the increases in standard deviations from FR 8 to FR 16 were proportionately greater than the increases from FR 4 to FR 8. Percent

FIG. 11. Relative frequency (percent) of number of responses in initial runs in DRO periods on Mix FR 4 DRO 30 seconds. Numerals on the abscissa represent upper limit of class interval the width of which is 1/4 of the FR requirement (one response). The shaded bar represents the interval containing the median.

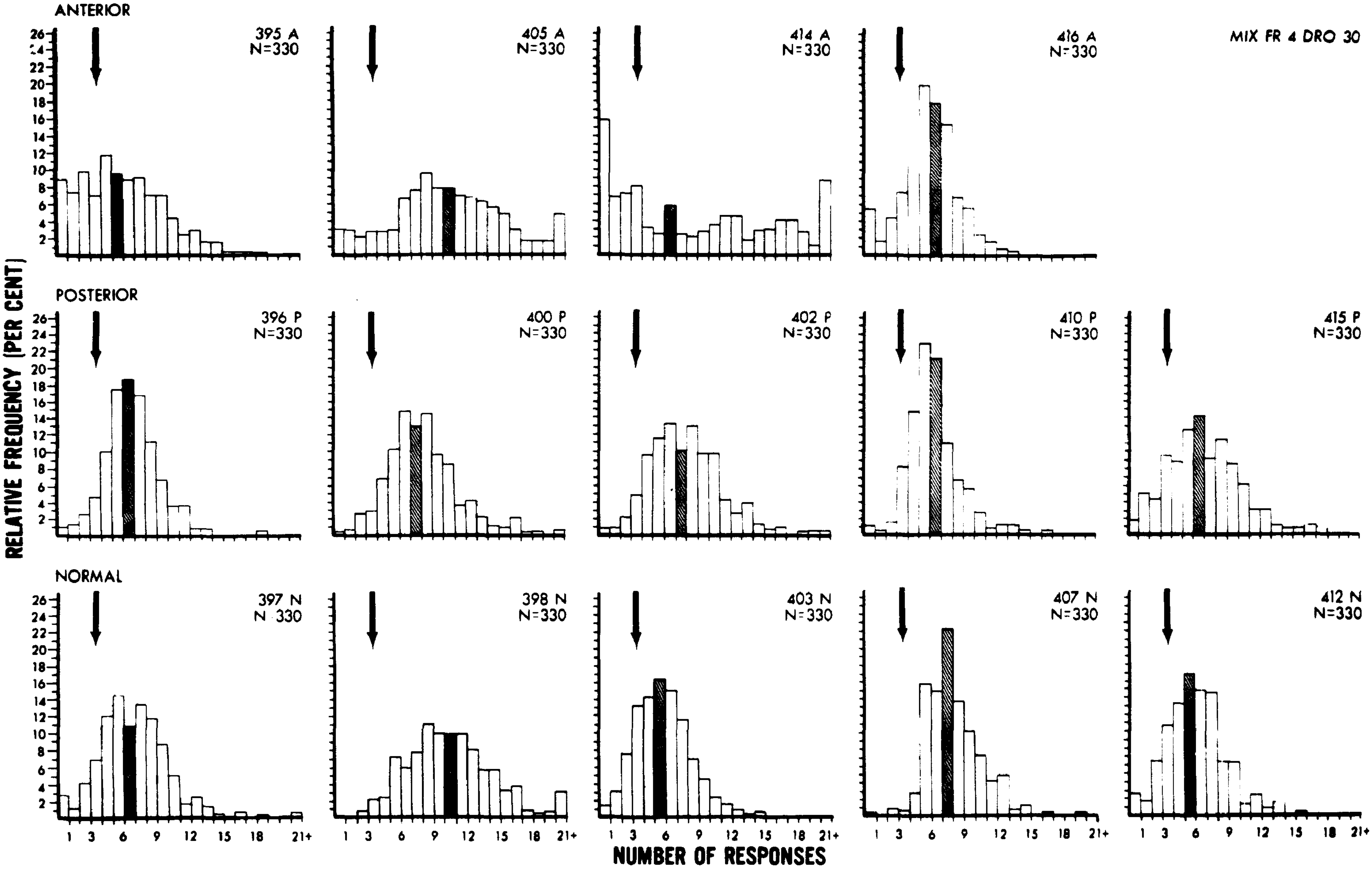

ANTERIOR
395 A N=330
405 A N=330
414 A N=330
416 A N=330
MIX FR 4 DRO 30
POSTERIOR
396 P N=330
400 P N=330
402 P N=330
410 P N=330
415 P N=330
NORMAL
397 N N 330
398 N N=330
403 N N=330
407 N N=330
412 N N=330
RELATIVE FREQUENCY (PER CENT)
2 4 6 8 10 12 14 16 18 20 22 24 26
1 3 6 9 12 15 18 21+
NUMBER OF RESPONSES

FIG. 12. Relative frequency (percent) of number of responses in initial runs in DRO periods on Mix FR 8 DRO 30 seconds. Numerals on the abscissa represent upper limit of class interval the width of which is 1/4 of the FR requirement (two responses). The shaded bar represents the interval containing the median.

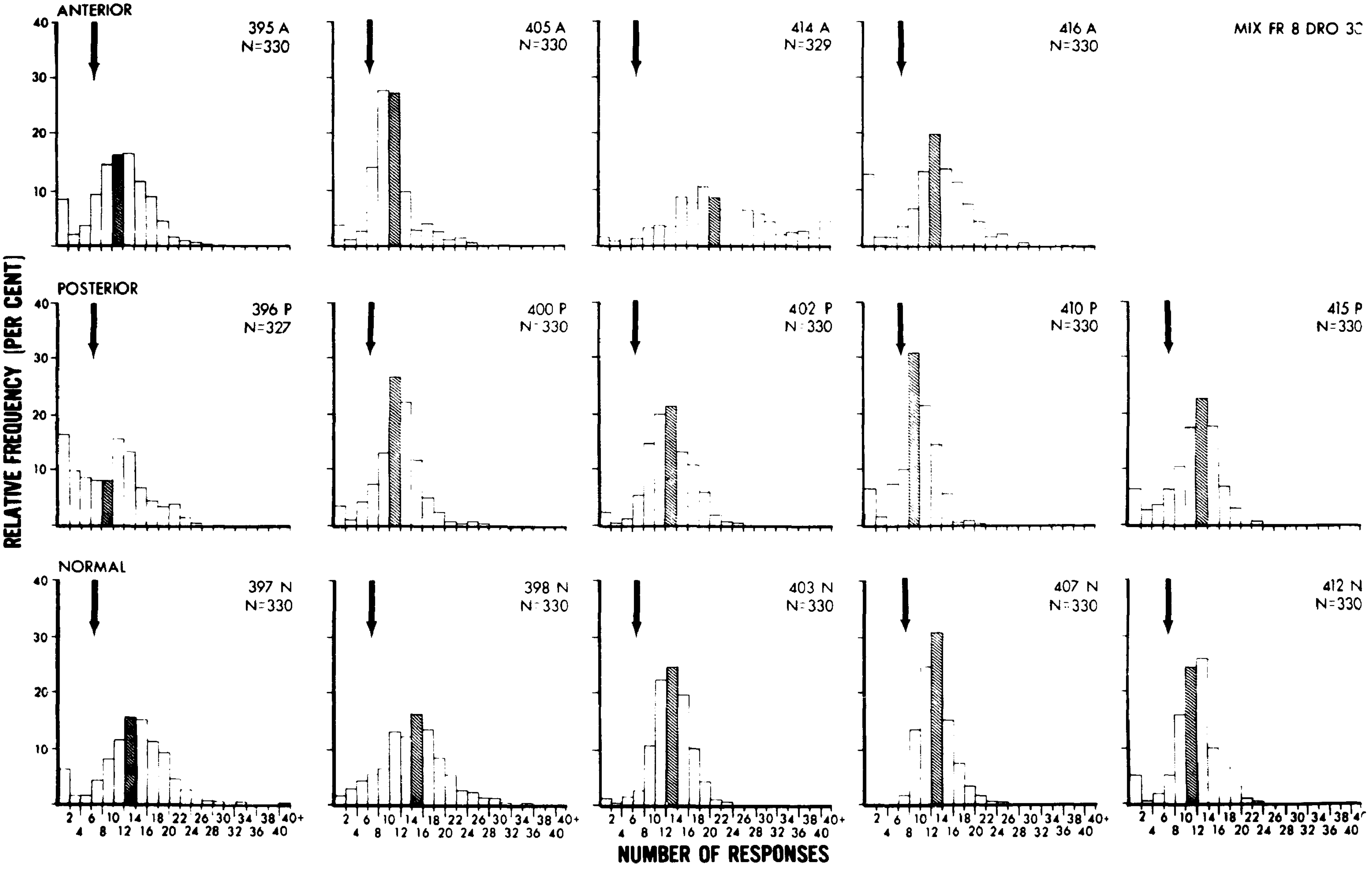
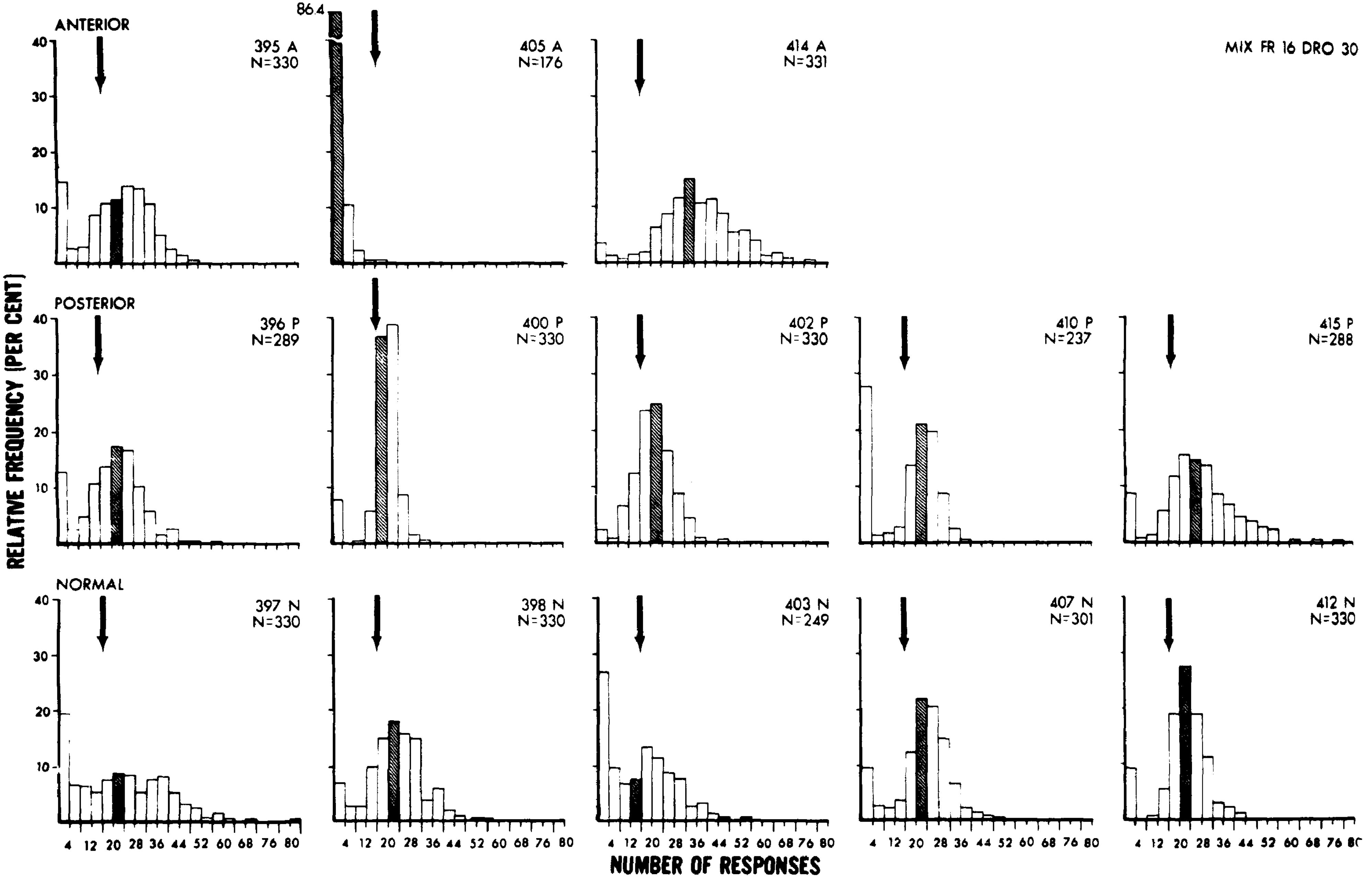

ANTERIOR
395 A
N=330
405 A
N=330
414 A
N=329
416 A
N=330
MIX FR 8 DRO 3C
POSTERIOR
396 P
N=327
400 P
N=330
402 P
N=330
410 P
N=330
415 P
N=330
NORMAL
397 N
N=330
398 N
N=330
403 N
N=330
407 N
N=330
412 N
N=330
RELATIVE FREQUENCY (PER CENT)
40
30
20
10
2
4
6
8
10
12
14
16
18
20
22
24
26
28
30
32
34
36
38
40
40+
NUMBER OF RESPONSES

FIG. 13. Relative frequency (percent) of number of responses in initial runs in DRO periods on Mix FR 16 DRO 30 seconds. Numerals on the abscissa represent upper limit of class intervals the width of which is 1/4 of the FR requirement (four responses). The shaded bar represents the interval containing the median.

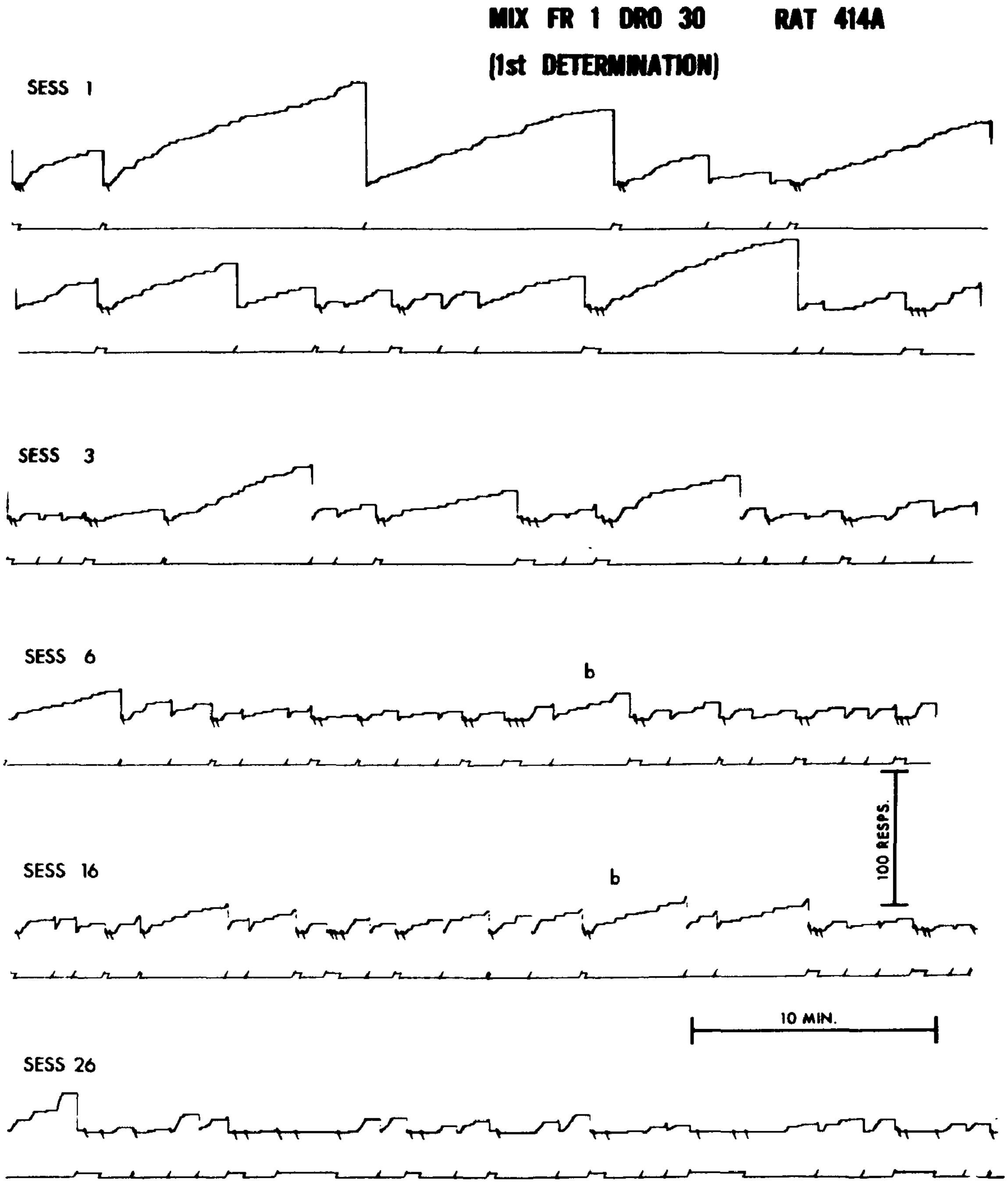

ANTERIOR
POSTERIOR
NORMAL
MIX FR 16 DRO 30
86.4
395 A N=330
405 A N=176
414 A N=331
396 P N=289
400 P N=330
402 P N=330
410 P N=237
415 P N=288
397 N N=330
398 N N=330
403 N N=249
407 N N=301
412 N N=330
RELATIVE FREQUENCY (PER CENT)
10
20
30
40
NUMBER OF RESPONSES
4 12 20 28 36 44 52 60 68 76 80

increases in size of mean standard deviation from FR 4 to FR 8 and from FR 8 to FR 16 were 30 and 50 percent respectively for the anterior group, 60 and 100 percent respectively for the posterior group, and 48 and 148 percent respectively for the normal animals. The standard deviations obtained in the present experiment were thus not linearly related to the size of the fixed ratio, and Weber's Law or its linear generalization does not describe the present data (Platt and Senkowski, 1970).

Acquisition of Mixed Fixed-Ratio DRO schedules. Representative cumulative records of responding at the FR 1 level of the mixed schedule are shown for rats 414A and 410P in Figures 14 and 15 respectively. Additional records are shown in Figures C through Q in Appendix I. It can be seen that early sessions were marked by DRO periods in which responding continued for several minutes and produced negatively accelerated extinction curves. With continued exposure to the schedule responding during DRO periods diminished until, during the final ten sessions, records were characterized by the emission of a single response followed by a pause of no responding until the end of the DRO period was signalled. Instances of continued responding during DRO did not disappear entirely, however, as can be seen from the points marked "a" on the records (see Figure 15). During session 26 for example, rat 410P emitted two responses in each of six DRO periods and three responses in each of three DRO periods. The record for rat 410P during session 26 is fairly typical of the performance of both posterior and normal animals.

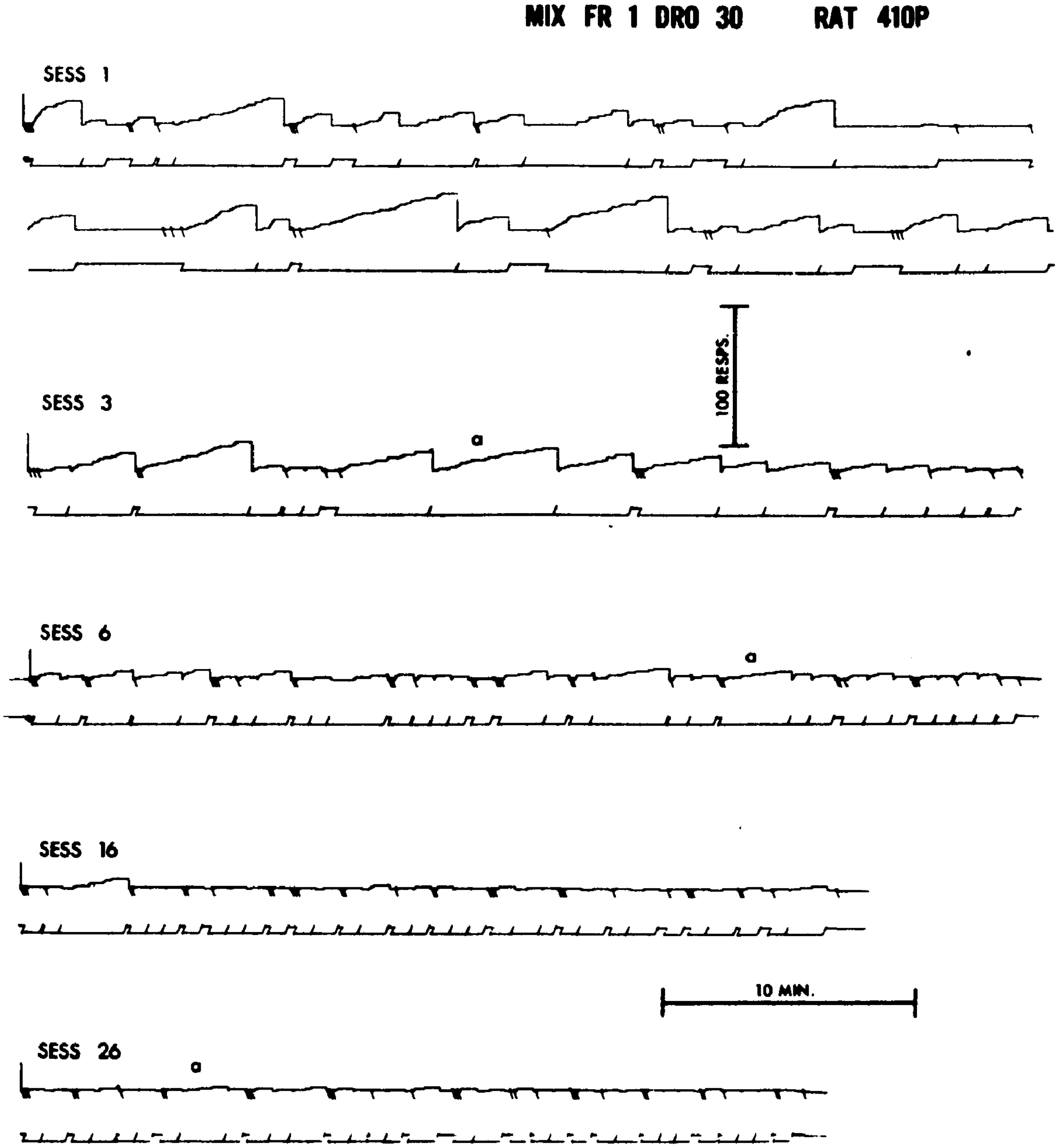
In the case of the anterior animals DRO responding was more often characterized by bursts of responses which were relatively closely

FIG. 14. Portions of representative cumulative records of rat 414A from sessions 1, 3, 6, 16, and 26 of the first steady state determination of Mix FR 1 DRO 30 seconds. The response pen resets at the end of each reinforcement and DRO period. Hatch marks indicate reinforcement. The event marker is up during reinforcement and down during DRO periods.



MIX FR 1 DRO 30 RAT 414A
(1st DETERMINATION)

FIG. 15. Portions of representative cumulative records of rat 410P from sessions 1, 3, 6, 16, and 26 of the first steady state determination at Mix FR 1 DRO 30 seconds. The response pen resets at the end of each reinforcement and DRO period. Hatch marks indicate reinforcement. The event marker is up during reinforcement and down during DRO periods.


MIX FR 1 DRO 30
RAT 410P
SESS 1
SESS 3
a
100 RESPS.
SESS 6
a
SESS 16
10 MIN.
SESS 26
a

spaced in time. Examples can be seen from the points marked "b" in record of rat 414A (see Figure 14). This pattern of responding was also typical of rat 405A and to a lesser extent of rats 395A and 416A.

When the mixed schedule involved ratios greater than one, DRO responding during early sessions at a given level was again marked by bouts of continued responding and negatively accelerated extinction curves. Over sessions these negatively accelerated curves gave way to curves characterized by runs of closely spaced responses followed by pauses of no responding. In cases where only one such run was emitted the record resembled a plateau. In DRO periods in which additional runs were emitted a stepwise configuration emerged (see points marked "c" in Figure 16). Comparison of DRO responding (Figure 16, event marker down) with reinforced periods (Figure 16, event marker up) revealed that initial runs emitted in DRO periods generally contained more responses than required by the ratio. This tendency to overshoot the ratio in DRO periods was also evident in the mean number of response per initial run (see Table E, Appendix I).

At the higher ratios (e.g., FR 16) initial runs containing fewer responses than the ratio occurred. The points marked "d" in the record of rat 405A at FR 16 (see Figure 17) are typical. In the case of rat 405A these short runs occurred in about 90% of DRO periods and with approximately the same frequency in reinforced periods of the schedule. Two other animals, 410P and 403N revealed similar frequencies of short runs.

These short runs suggested ratio "strain," i.e., increases in frequency and duration of within run pausing associated with increases in ratio size (Ferster and Skinner, 1957; Powell, 1970).

FIG. 16. Portions of representative cumulative records of rat 405A from sessions 1, 3, 6, 16, and 26 of Mix FR 4 DRO 30 seconds. The response pen resets at the end of each reinforcement and DRO period. Hatch marks indicate responses during the three second time out following DRO. The event marker is up during reinforcement and down during DRO periods.

MIX FR 4 DRO 30 RAT 405A

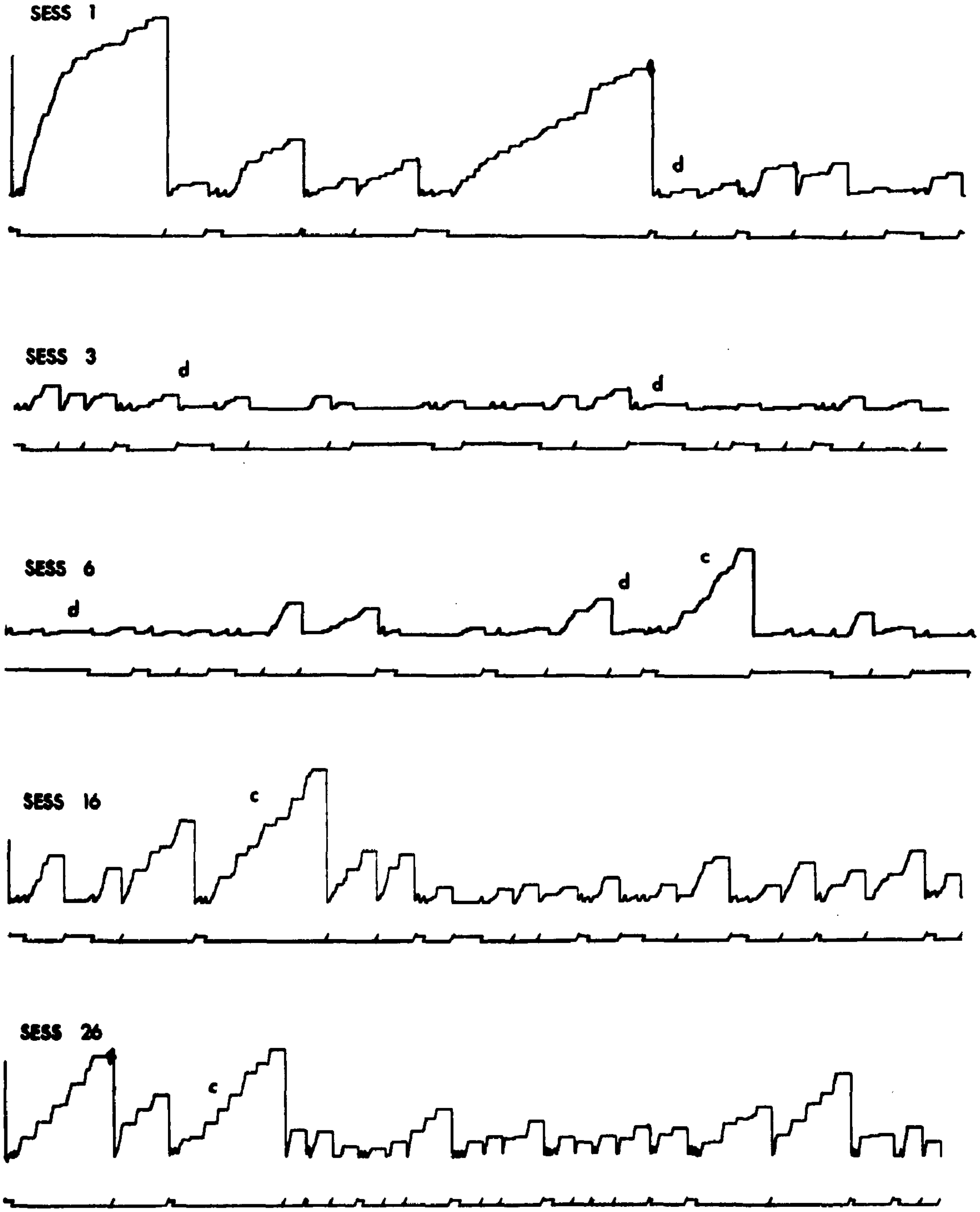
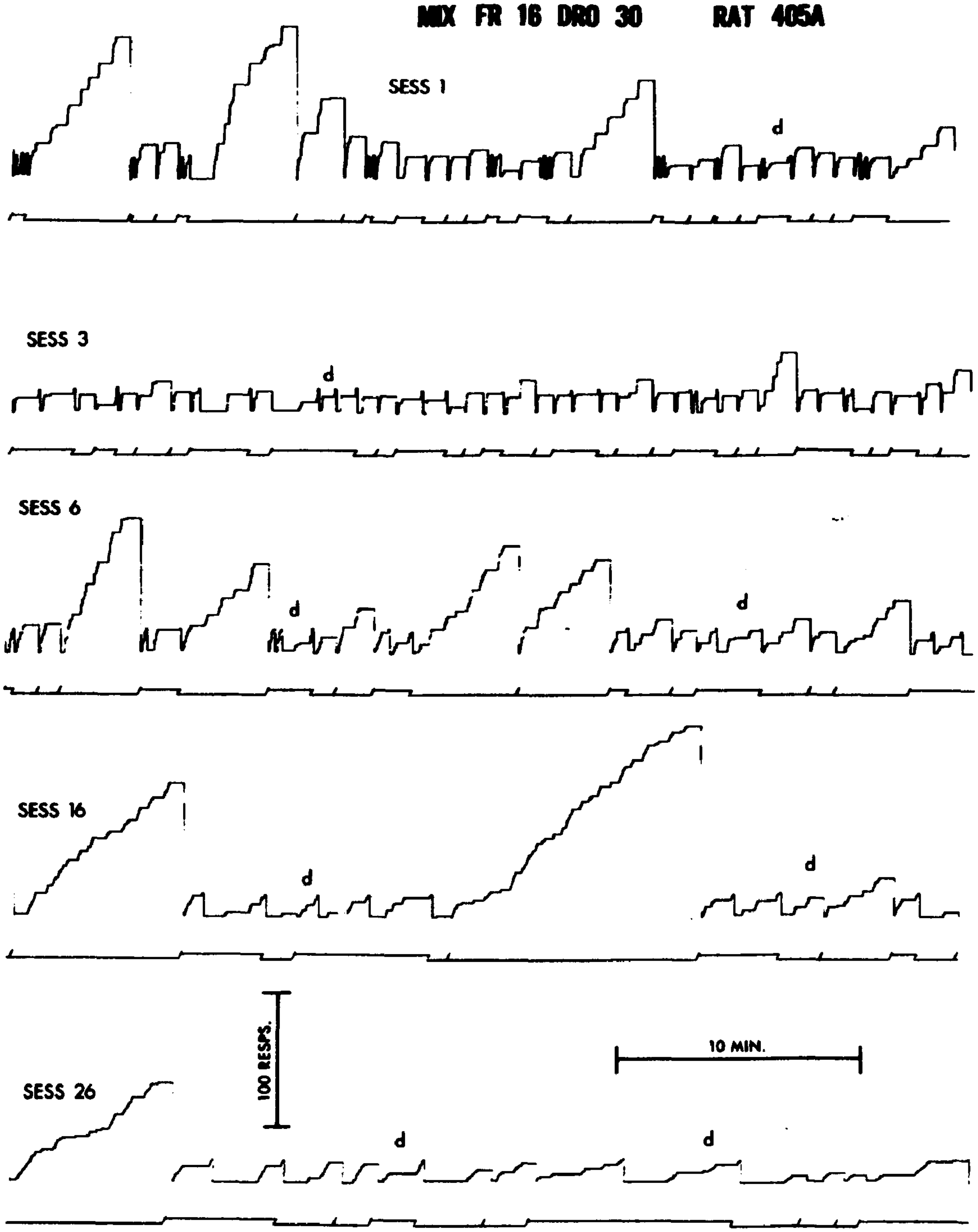


FIG. 17. Portions of representative cumulative records of rat 405A from sessions 1, 3, 6, 16, and 26 of Mix FR 16 DRO 30 seconds. The response pen resets at the end of each reinforcement and DRO period. Hatch marks indicate responses during the three second time out following DRO. The event marker is up during reinforcement and down during DRO periods.


MIX FR 16 DRO 30
RAT 405A
SESS 1
d
SESS 3
d
SESS 6
d
d
SESS 16
d
d
100 RESPS.
10 MIN.
SESS 26
d
d

When such pausing occurred during DRO periods it tended to perturb the typical plateau and stepwise configurations described earlier. In some records (e.g., Figure 17) this breakdown in the usual pattern of responding was also accompanied by increases in the latency of the first response in both DRO and reinforcement periods. By way of contrast, Figure 18 shows cumulative records of rat 414A in which the appearance of short runs did not disrupt the usual pattern of responding.

A more quantitative description of acquisition of the mixed schedules was obtained by plotting mean total response per DRO period, mean number of runs per DRO period, and mean number of responses emitted per initial run in each DRO period for every third session at each level of the ratio. Figures 19, 20, and 21 show these curves for the first determination at the FR 1 level and for the FR 16 level.

Inspection of these figures shows that responding at the FR 1 level provided the greatest degree of uniformity. Curves showing mean total responses per DRO for every third session at the FR 1 level (Figure 19) were decreasing and negatively accelerated. Curves showing mean number of runs per DRO period at the FR 1 level (see Figure 20) were also decreasing and negatively accelerated. When the mean number of responses per initial run in DRO periods were plotted, however, the curves (see Figure 21) revealed relatively little increase or decrease over sessions. A notable exception was rat 405A.

Curves for the FR 16 level are shown in Figures 22, 23, and 24. At higher ratios curves showing mean total responses per DRO period (see Figure 22) and mean number of responses per initial run (see Figure 24) reveal inversions over sessions. These probably reflected a breakdown in schedule control over responding. Such breakdowns were

FIG. 18. Portions of representative cumulative records of rat 414A from sessions 1, 3, 6, 16, and 26 of Mix FR 16 DRO 30 seconds. The response pen resets at the end of each reinforcement and DRO period. Hatch marks indicate responses during the three second time out following DRO. The event marker is up during reinforcement and down during DRO periods.

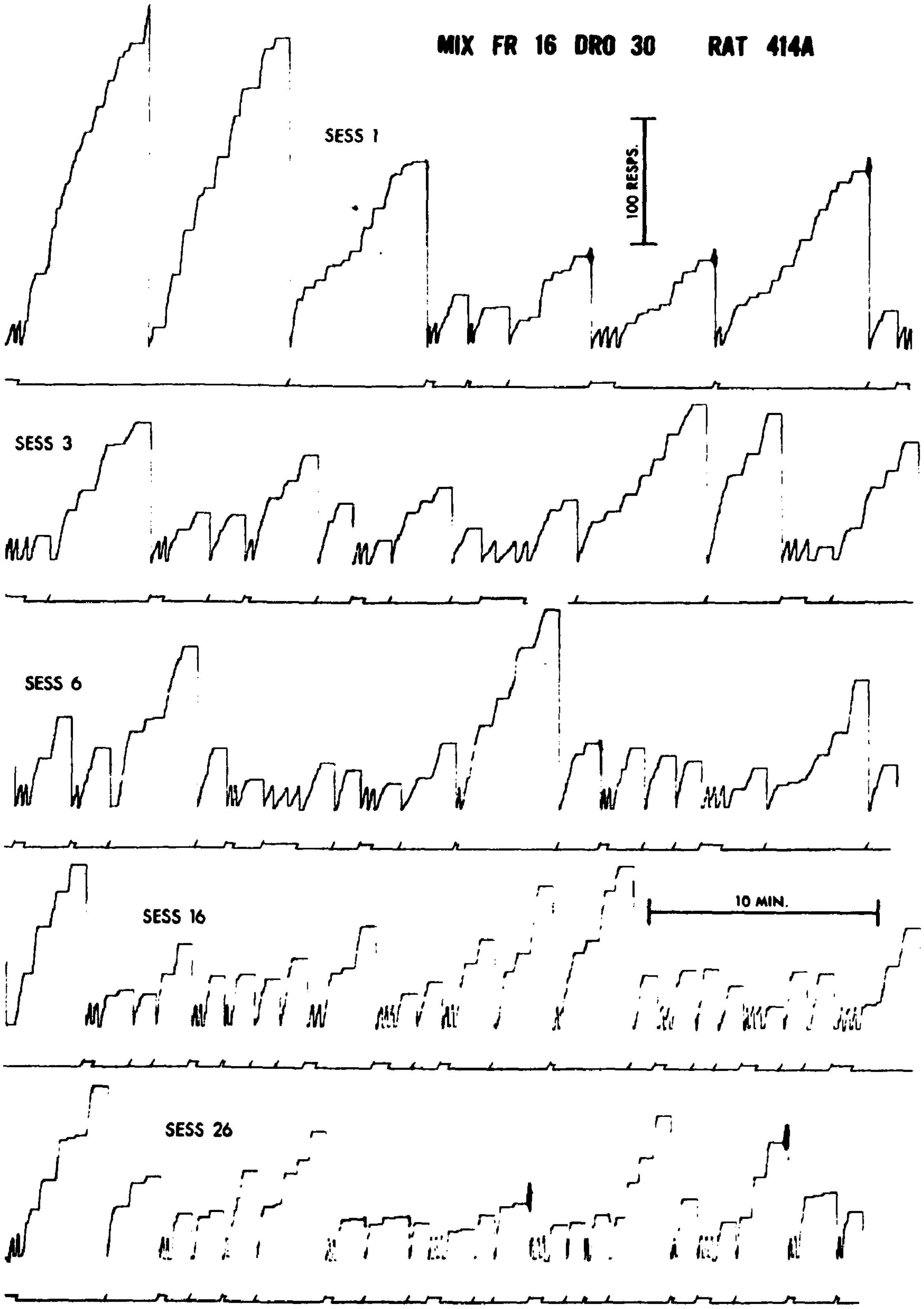

MIX FR 16 DRO 30 RAT 414A
SESS 1
100 RESPS.
SESS 3
SESS 6
SESS 16
10 MIN.
SESS 26

FIG. 19. Mean total responses per DRO period for every third session for each animal at the first determination of Mix FR 1 DRO 30 seconds. Each data point represents the session mean of the total number of responses emitted during each DRO period.

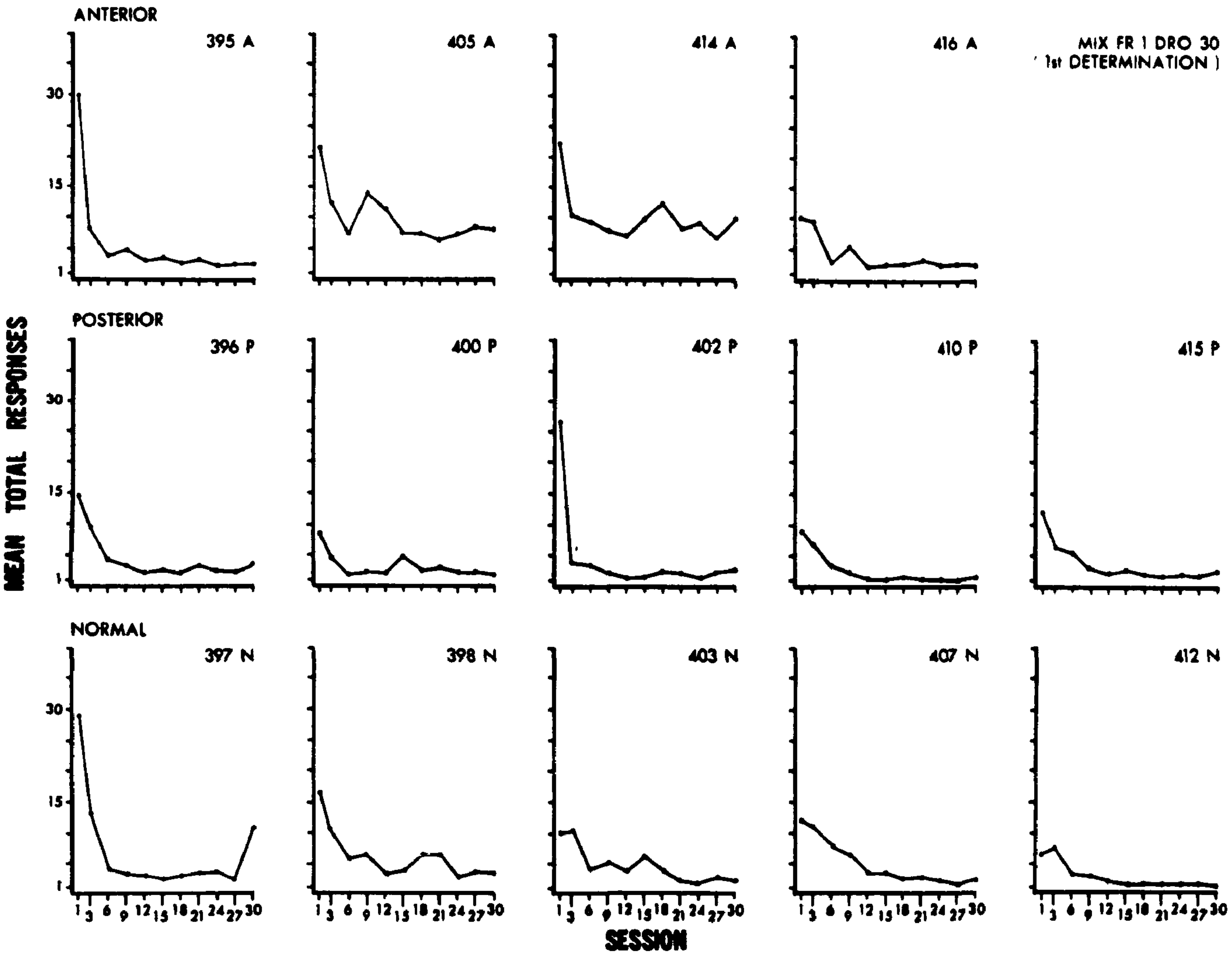

ANTERIOR
395 A
405 A
414 A
416 A
MIX FR 1 DRO 30
(1st DETERMINATION)
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
MEAN TOTAL RESPONSES
1
15
30
1 3 6 9 12 15 18 21 24 27 30
SESSION

FIG. 20. Mean number of runs per DRO period for every third session for each animal at the first determination of Mix FR 1 DRO 30 seconds. Each data point represents the session mean of the number of runs emitted in each DRO period.

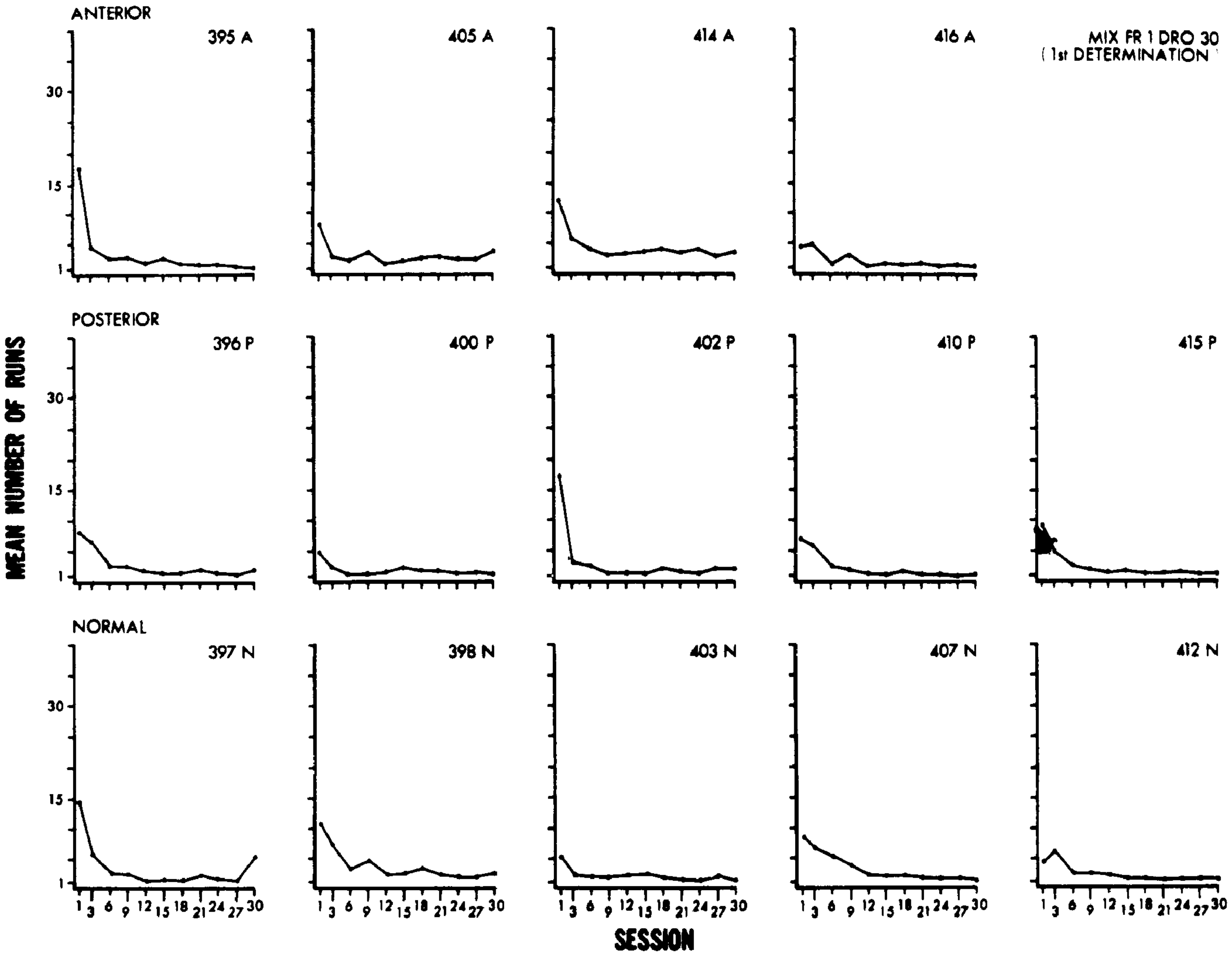

ANTERIOR
395 A
405 A
414 A
416 A
MIX FR 1 DRO 30
(1st DETERMINATION)
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
30
15
1
MEAN NUMBER OF RUNS
1 3 6 9 12 15 18 21 24 27 30
SESSION

FIG. 21. Mean number of responses in initial run in DRO for every third session for each animal at the first determination of Mix FR 1 DRO 30 seconds. Each data point represents the session mean of the number of responses emitted in the initial run in each DRO period before the first interresponse time greater than three seconds.

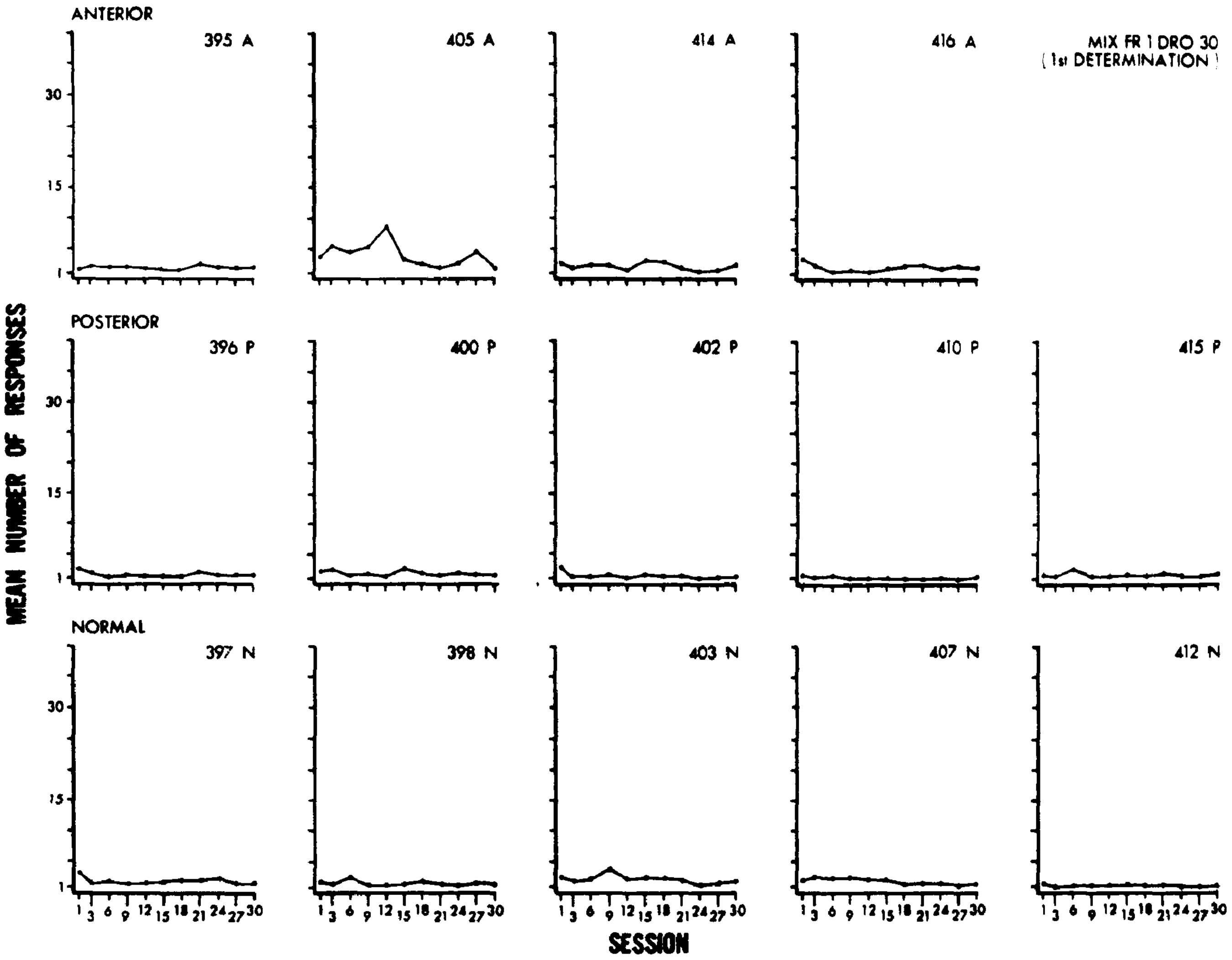

ANTERIOR
395 A
405 A
414 A
416 A
MIX FR 1 DRO 30
(1st DETERMINATION)
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
30
15
1
1 3 6 9 12 15 18 21 24 27 30
MEAN NUMBER OF RESPONSES
SESSION

FIG. 22. Mean total responses per DRO period for every third session for each animal on Mix FR 16 DRO 30 seconds. Each data point represents the session mean of the total number of responses emitted in each DRO period.

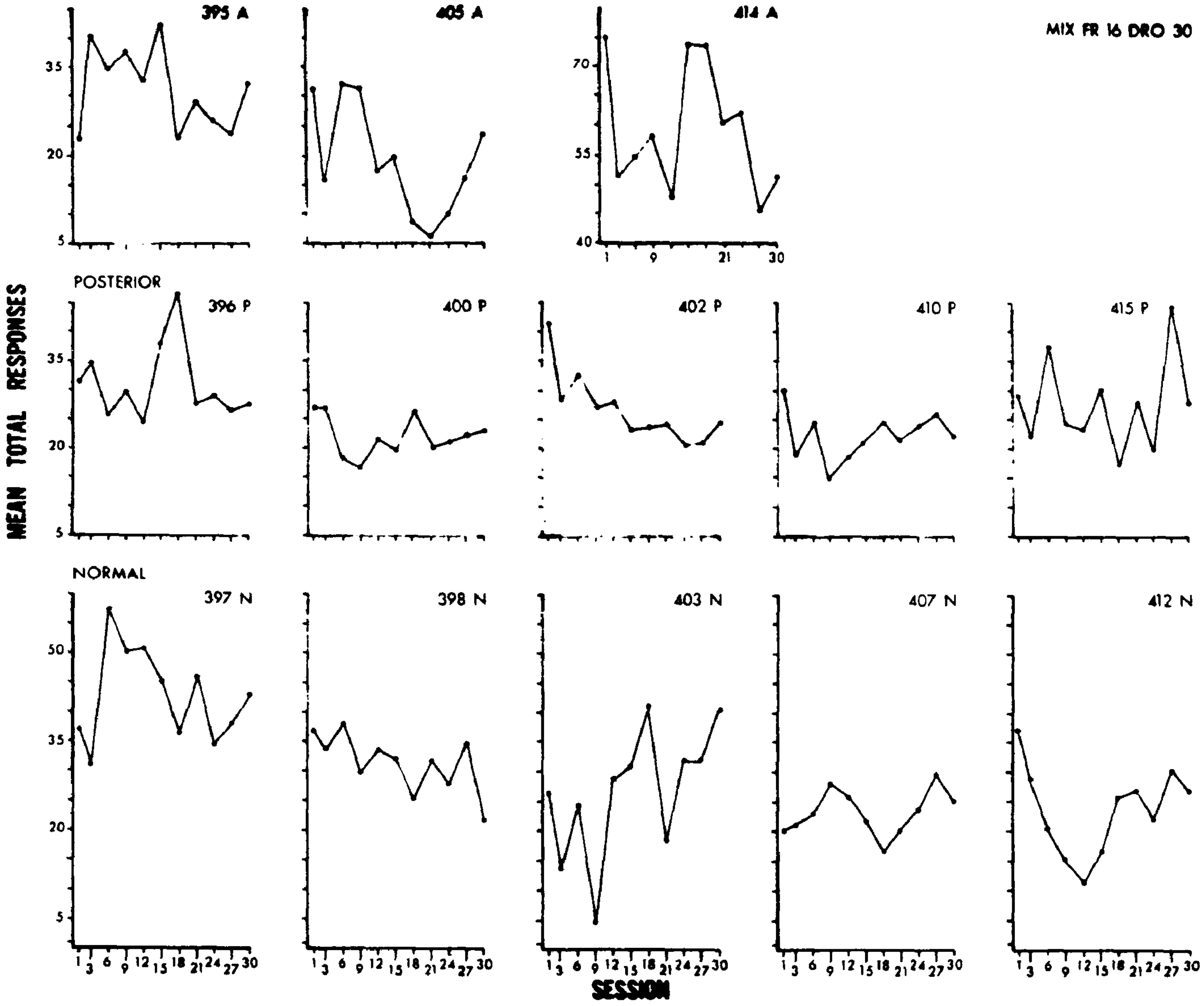

MIX FR 16 DRO 30
MEAN TOTAL RESPONSES
395 A
405 A
414 A
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
5
20
35
50
40
55
70
1
3
6
9
12
15
18
21
24
27
30
SESSION

FIG. 23. Mean number of runs per DRO period for every third session for each animal on Mix FR 16 DRO 30 seconds. Each data point represents the session mean of the number of runs emitted in each DRO period.

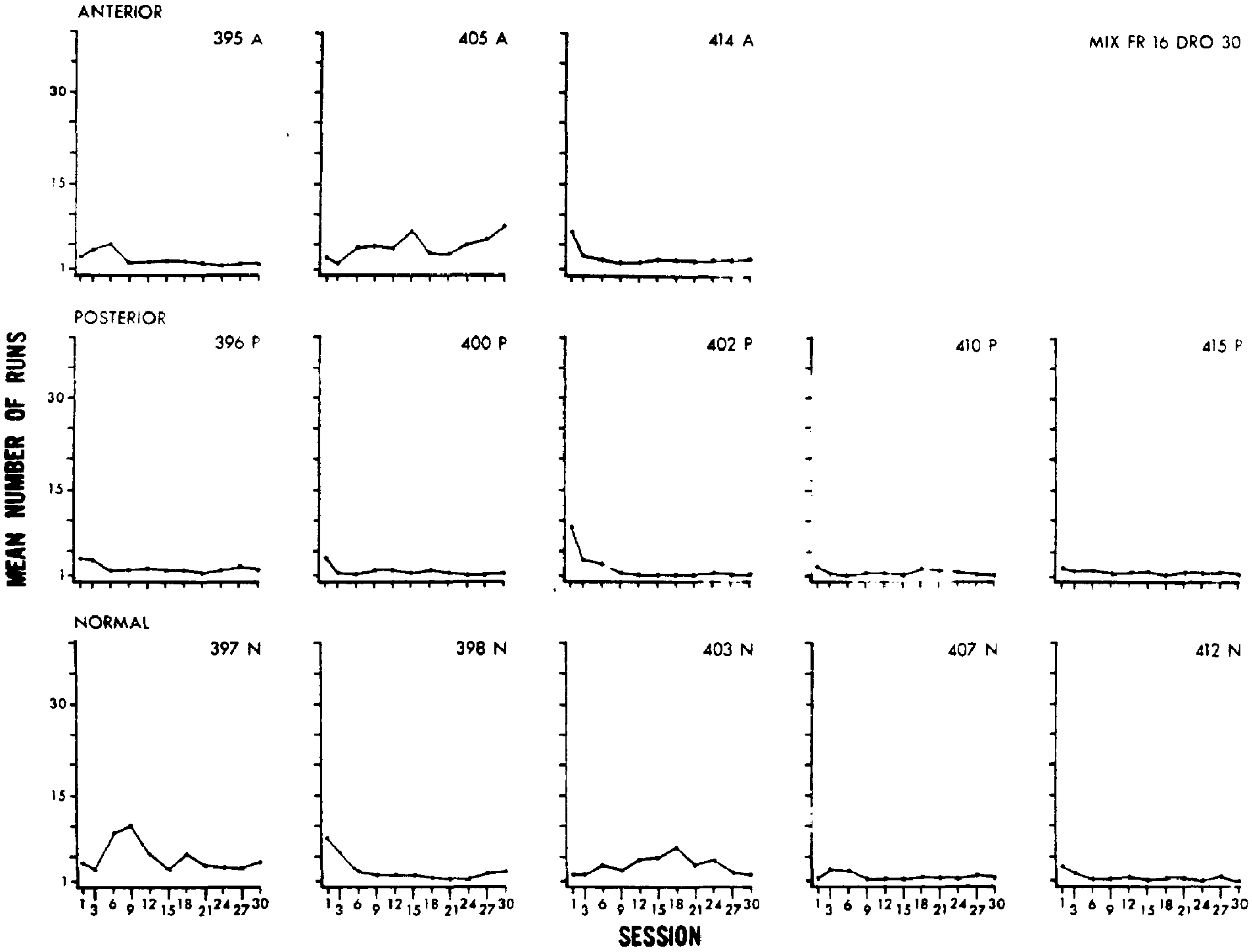
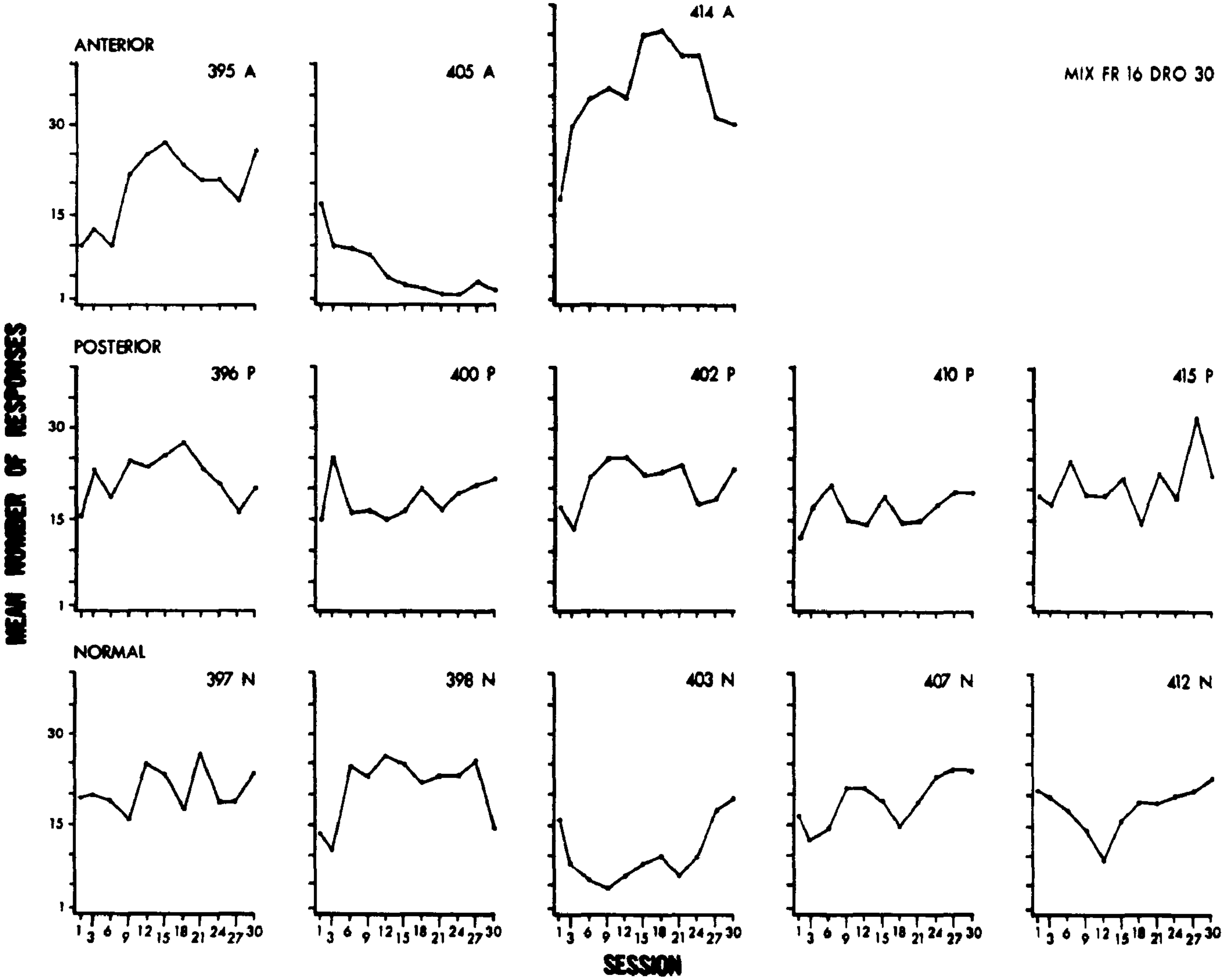

ANTERIOR
395 A
405 A
414 A
MIX FR 16 DRO 30
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
MEAN NUMBER OF RUNS
30
15
1
1 3 6 9 12 15 18 21 24 27 30
SESSION

FIG. 24. Mean number of responses in initial run in DRO for every third session for each animal on Mix FR 16 DRO 30 seconds. Each data point represents the session mean of the number of responses emitted in each DRO period before the first interresponse time greater than three seconds.


ANTERIOR
395 A
405 A
414 A
MIX FR 16 DRO 30
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
MEAN NUMBER OF RESPONSES
30
15
1
1 3 6 9 12 15 18 21 24 27 30
SESSION

further indicated by the increased incidence of runs containing fewer responses than required by the ratio.

Curves showing the mean number of runs per DRO period over sessions at the higher ratios (see Figure 23) revealed fewer perturbations. Generally these curves at each level of the ratio showed small decreases during the first three to nine sessions after which the number of runs stabilized.

Taken together, these curves showed that decreases in total number of responses emitted in DRO periods over sessions represented decreases in the number of runs emitted rather than in the number of responses per run.

Note should be made of the increases in the number of runs per DRO period shown in the curve for rat 397N at the FR 16 level (see Figure 23). A similar increase in number of runs was shown by 397N at FR 8 and by 398N at FR 4. Inspection of the cumulative records for the relevant sessions revealed continued bouts of unusually sustained responding during DRO periods in which the cumulative response curve more closely resembled the kind of sustained responding seen during initial exposures at the FR 1 level (e.g., Figure 14, session 1) rather than the more characteristic stepwise configuration. This indicated a disruption of the type of control over responding which is normally associated with FR schedules (Ferster and Skinner, 1957).

Additional acquisition curves are shown in Figures R through Z in Appendix I.

EXPERIMENT II

While Experiment I had been designed to study post-operative acquisition and steady behavior maintained at several levels of the Mix FR DRO schedule, the purpose of Experiment II was to investigate post-operative retention following pre-operative acquisition at only one level of the FR requirement. FR 1 was selected as the level of choice as it was only at the FR 1 level in Experiment I that any differences between lesion groups may have existed. Further, it was felt that between subject variability arising from responding at ratios greater than one might obscure behavioral differences arising from the lesion variable.

Method

Subjects. Thirteen experimentally naive, male, Long Evans rats from Rockland Farms, Inc., Gilbertsville, Pennsylvania were used in the experiment. These animals arrived in the vivarium at approximately 60 days of age and were housed under the same conditions described in Experiment I. Rats were approximately 220 days old when Experiment II began. Five rats were assigned to each of the two lesion groups: anterior caudate and posterior caudate. Three animals were designated as normal, unoperated controls. Of the five animals assigned to the posterior group, two failed to survive surgery and one died from respiratory infection during post-operative testing. One of the three rats assigned to the unoperated group also died of respiratory infection. Upon completion of testing of the surviving animals it was decided to run five additional unoperated controls in order to provide sufficient comparison with the anterior lesion group. These additional animals were also obtained from Rockland Farms, Inc. and arrived in the vivarium at approximately 90 days of age. These rats began the experiment at approximately 112 days of age. Feeding and deprivation procedures were carried out as described in Experiment I with the exception that ad libitum weights were determined only once.

Apparatus. The apparatus was the same operant conditioning apparatus used in Experiment I.

Procedure. Rats were adapted to the experimental chambers, shaped and magazine trained in the manner described in Experiment I. Following shaping, each animal received six daily sessions of FR 1 of 66 dipper presentations per session. Following these FR 1 sessions all animals were switched to Mix FR 1 DRO 30 schedule for 15 daily sessions of 34 reinforcements each. The mixed schedule was identical to that described in Experiment I.

After completion of the 15 sessions on the mixed schedule, the rats were placed back on free feeding for one week prior to surgery. Subjects were ranked according to the mean number of responses emitted per extinction period over the last five sessions and assigned to one of three groups such that the mean ranks for each group were approximately the same. The loss of subjects from the original 13 animals negated this attempt at matching.

Following surgery all operated animals were permitted one month to recover before being returned to the deprivation regimen. After return to 80% of ad libitum weight, all animals were again run for six daily sessions of FR 1 of 66 reinforcements each followed by 15 daily sessions on Mix FR 1 DRO 30 seconds of 34 reinforcements each.

The pre and post-operative procedural pattern followed for the five additional normal animals was the same as that for the original subjects. The procedure was not begun, however, until the original animals had completed all testing.

Surgery and Histology. Surgical and histological procedures were the same as those described in Experiment I.

Results

Figures 25 and 26 show composite reconstructions of caudate lesions in anterior and posterior rats respectively. Figures 27 and 28 show photographs of representative sections through the lesion of each animal. Estimated percent of caudate tissue destroyed in each rat, as well as the maximal anterior and posterior extents of each lesion bilaterally, are shown in Table I in Appendix II.

Anterior and posterior lesions were somewhat larger in Experiment II than in Experiment I, but were still small compared with lesions reported in other studies. It can be seen from Figures 25 through 28 that some damage to tissue adjacent to the caudate was sustained. For example, with the exception of rat 191A all anterior lesions extended posteriorally to intrude upon the globus pallidus. Lesions in rats 189A, 195A, and 191A produced damage to the nucleus accumbens, and damage to the anterior commissure can be seen in the lesions of rats 191A and 197A. Adjacent piriform cortex was damaged in rats 189A, 191A, and 197A. As in Experiment I, posterior lesions were placed more dorsally than intended and in both cases damage to the corpus callosum and overlying cortex was noted.

Retention of Mix FR 1 DRO 30 Seconds. Since only two of the rats in the original posterior group survived surgery, the statistical analysis treated only data from the anterior and normal animals. The data from the two posterior rats are shown, however.

In order to compare performances of the anterior and normal animals data were averaged for each group over the last five pre-operative,

FIG. 25. Composite reconstructions of lesions in the Anterior Caudate group (N = 5) in Experiment Two. Drawings were made on plates from the Pellegrino and Cushman (1967) atlas. The shaded areas represent individual lesions. The blackened areas represent the overlapping of two or more lesions. The number to the upper left of each drawing indicates millimeters anterior to bregma.

ANTERIOR

(EXPERIMENT II)

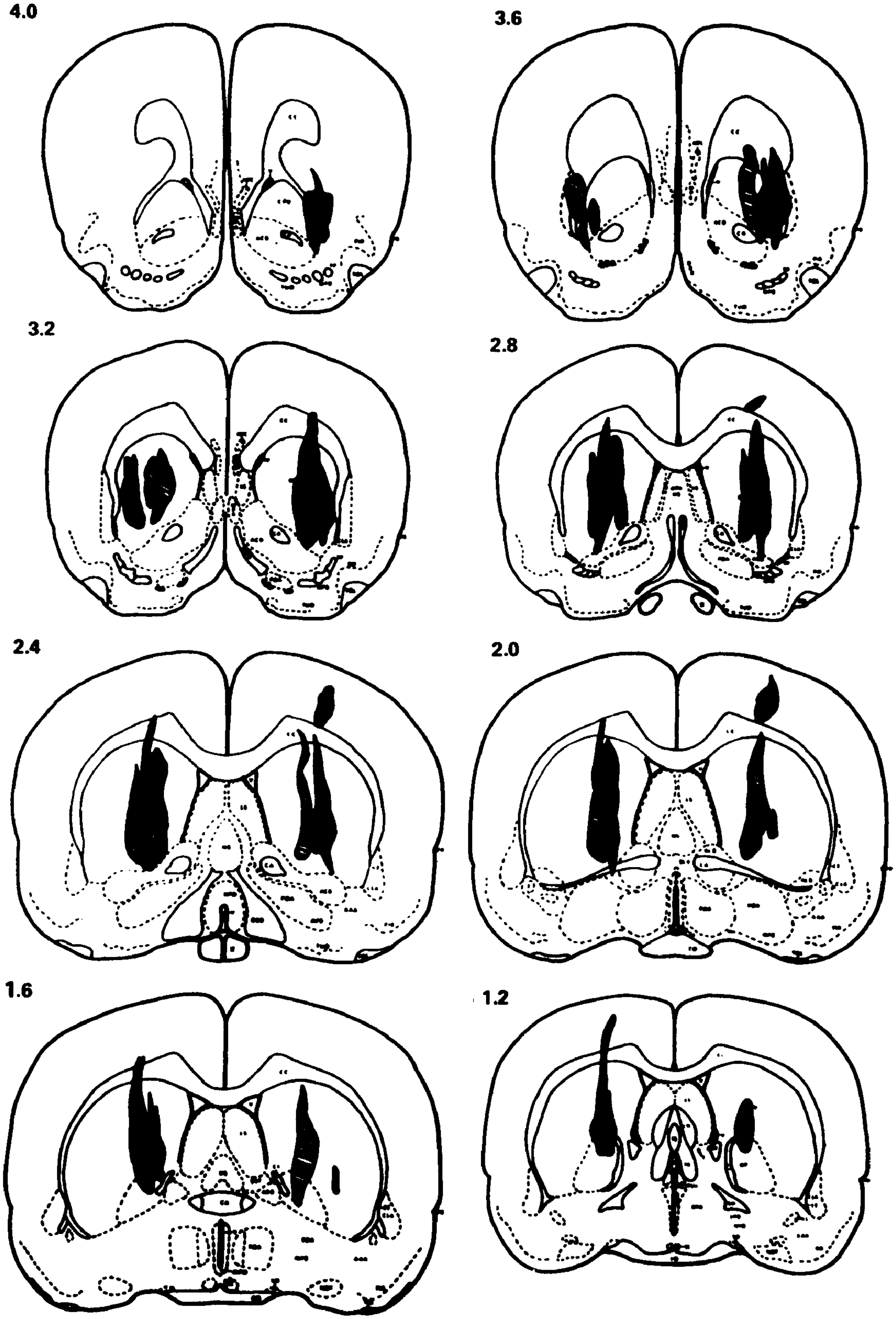


FIG. 26. Composite reconstructions of the lesions in the two rats with lesions aimed at the posterior caudate in Experiment Two. Drawings were made on plates from the Pellegrino and Cushman (1967) atlas. The shaded areas represent individual lesions. The blackened areas represent the overlapping of lesions. The number to the upper left of each drawing indicates millimeters anterior to bregma.

POSTERIOR

(EXPERIMENT II)

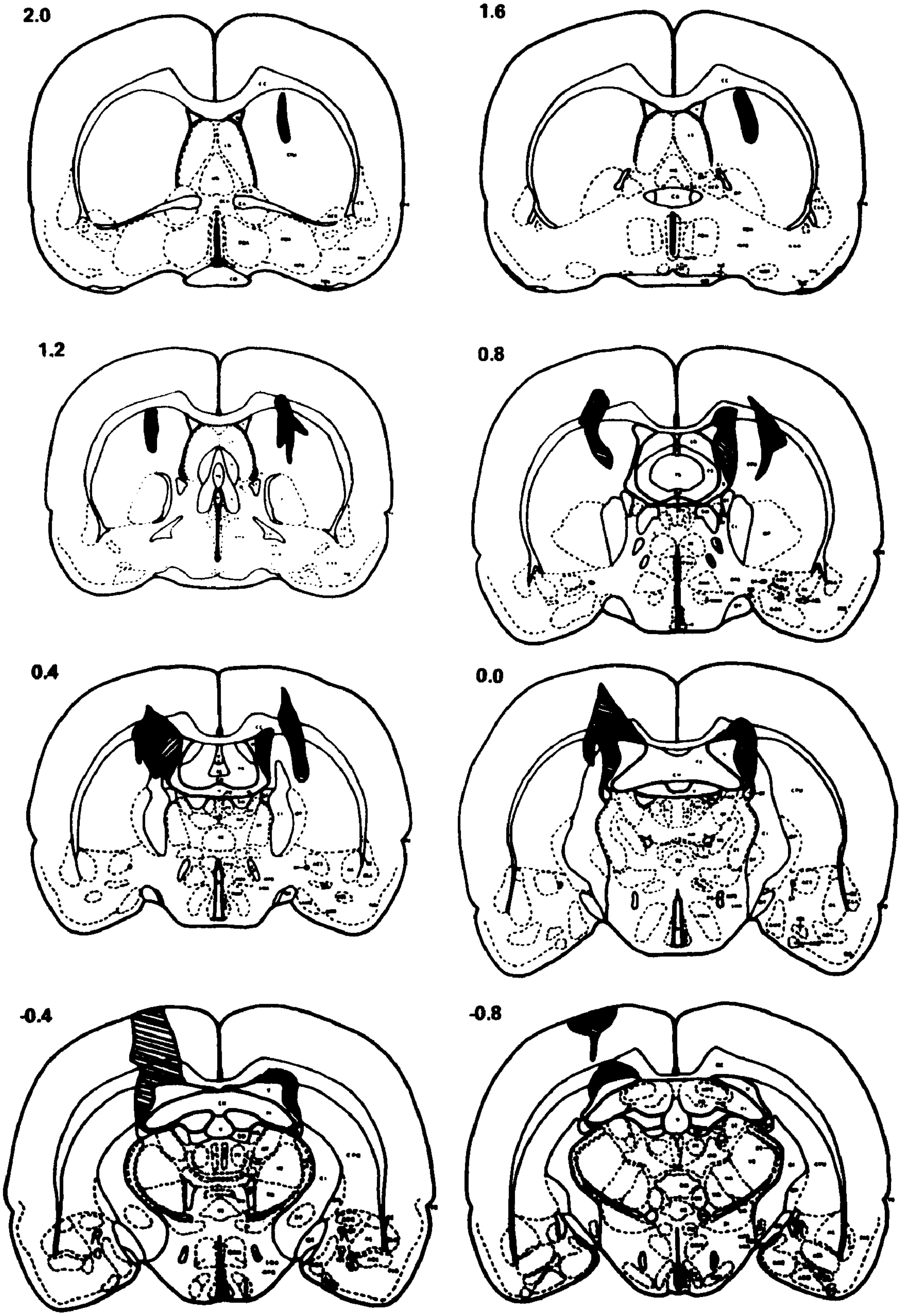
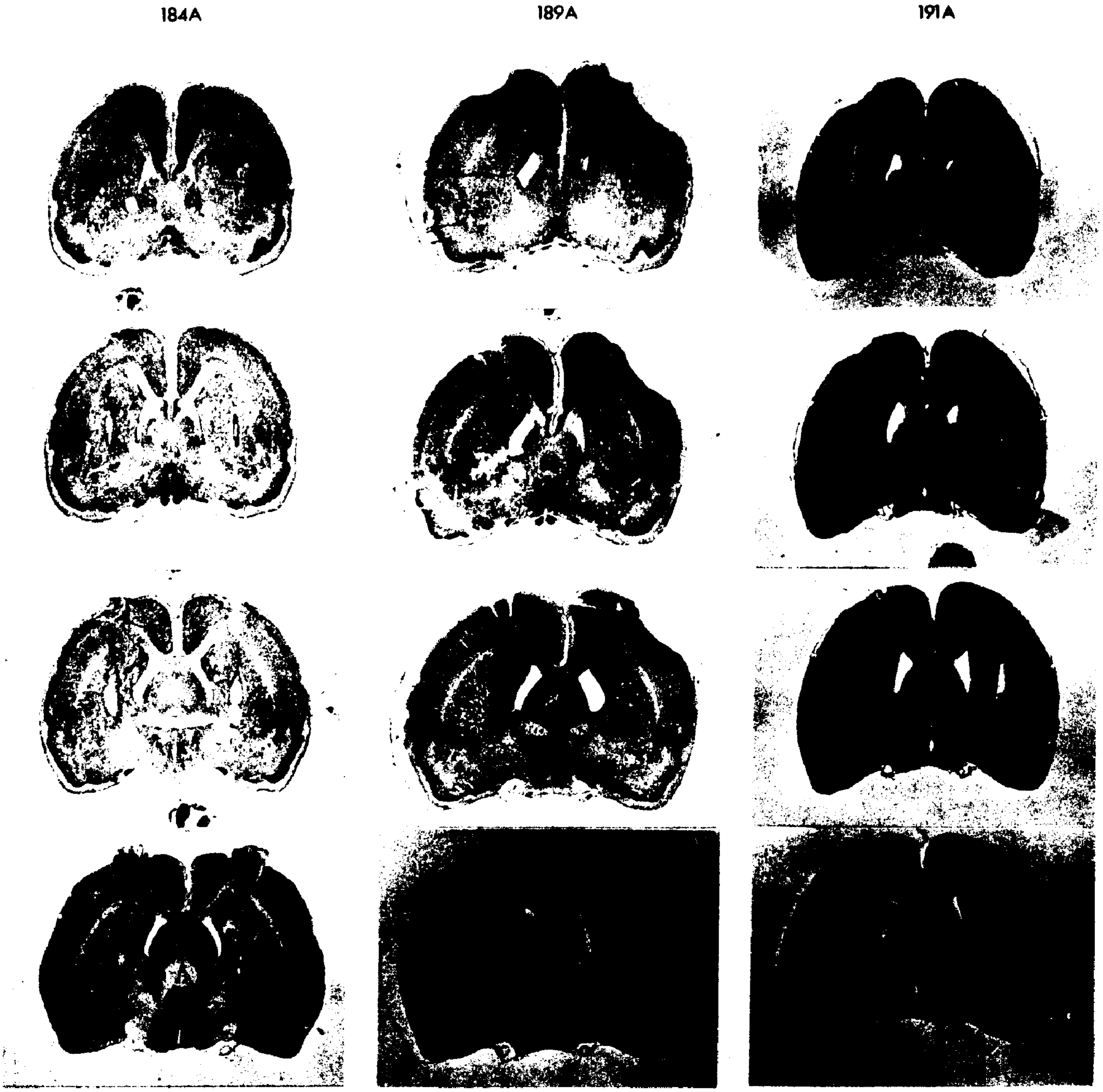


FIG. 27. Photographs of representative sections of the lesions in the anterior caudate group (Experiment II).

ANTERIOR
(EXPERIMENT II)



ANTERIOR
(EXPERIMENT II)

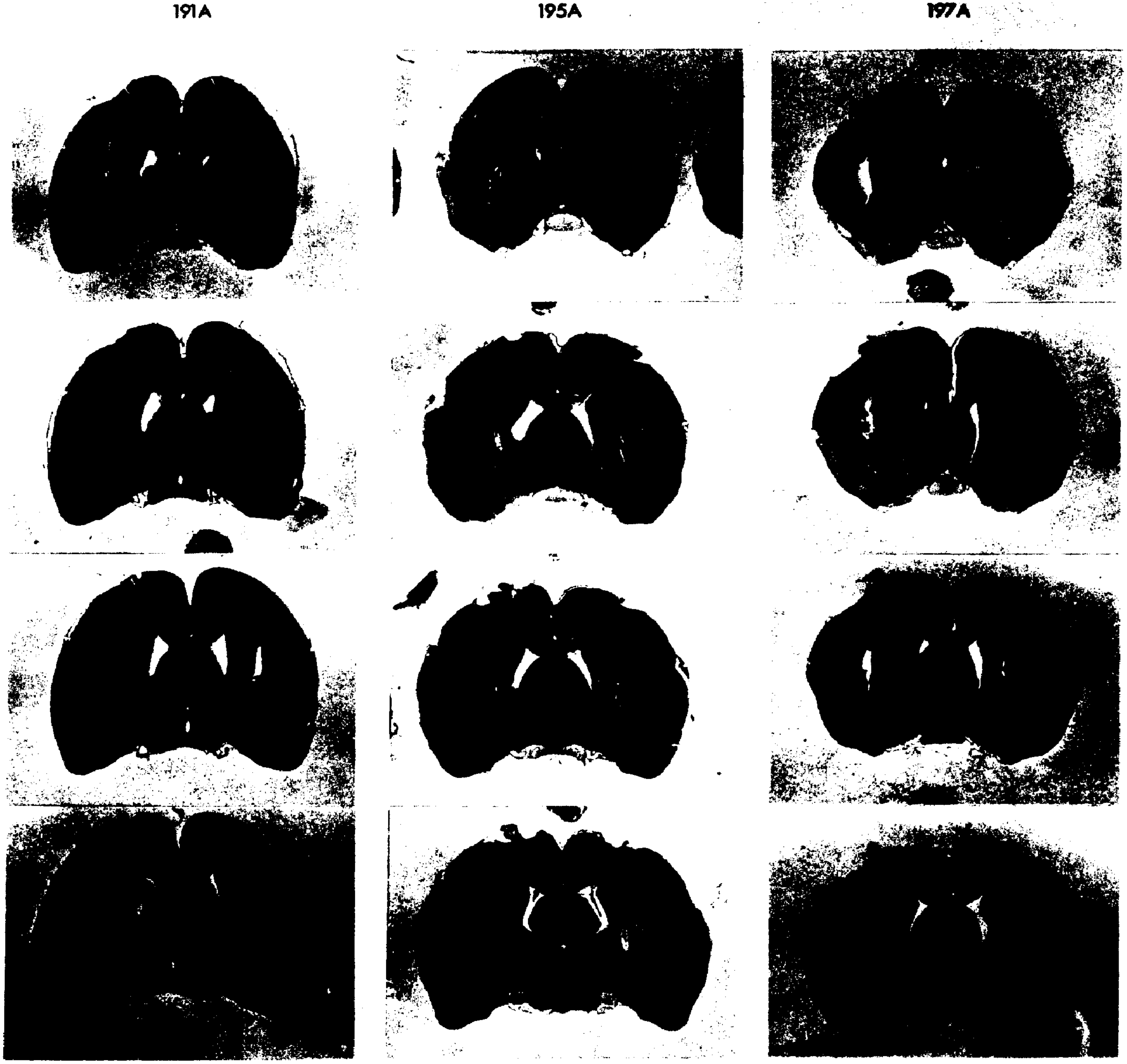
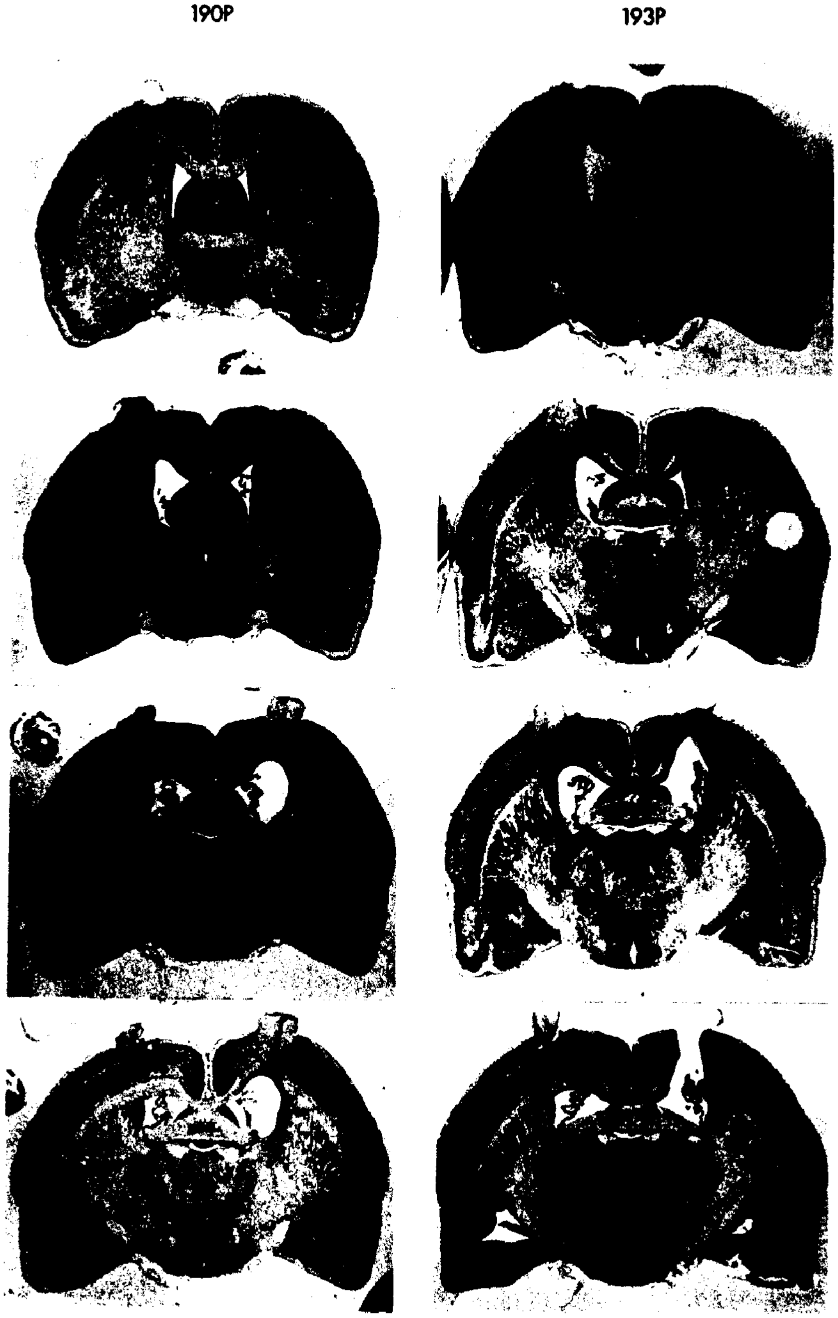


FIG. 28. Photographs of representative sections of the lesions in the posterior caudate group (Experiment II).

POSTERIOR
(EXPERIMENT II)



first five post-operative, and last five post-operative sessions. These data are presented in Figures 29 through 31. Means for individual rats are given in Appendix II (see Tables J through M). A series of a priori t tests were used to determine significance of difference at the .05 level, two tailed.

Figure 29 shows the means and ranges of the total number of responses emitted during DRO periods for anterior, posterior, and normal animals. While the pre-operative mean of 3.4 for the anterior group was larger than that of 2.3 for the normal group this difference was not significant (t = 1.02, d.f. = 10) and was due to the performance of a single anterior rat, 191A. When the performance of rat 191A is excluded the mean of the anterior group became 2.2 responses per DRO period. When post-operative means were compared it was clear that there was no longer any overlap between the two groups. The mean of the anterior group increased to 5.8 responses during the first five post-operative sessions compared to 2.2 responses for the normal group (t = 5.67, d.f. = 10). Although the mean of the anterior group decreased to 4.2 responses per DRO period during the last five post-operative sessions the mean of the normal group decreased to 1.7 such that the anterior group mean was still significantly greater (t = 5.98, d.f. = 10).

The mean number of runs emitted per DRO period are shown in Figure 30. It can be seen that the result was essentially the same as that for the mean total number of responses in DRO. Pre-operative means of anterior and normal groups did not differ significantly before surgery (t = 0.83, d.f. = 10), but post-operative means did differ significantly: For the anterior group during the first five post-operative sessions the mean number of runs emitted increased to 4.1 compared to 1.7 for the

FIG. 29. Means and ranges of the mean total number of responses emitted during DRO periods by animals in each group over the last five pre-operative (sessions 11-15), first five post-operative (sessions 1-5), and last five post-operative (sessions 11-15) sessions. Each mean is an average of the individual session means for each animal over the indicated sessions. The range shown is that of the individual means for each animal over the indicated sessions.

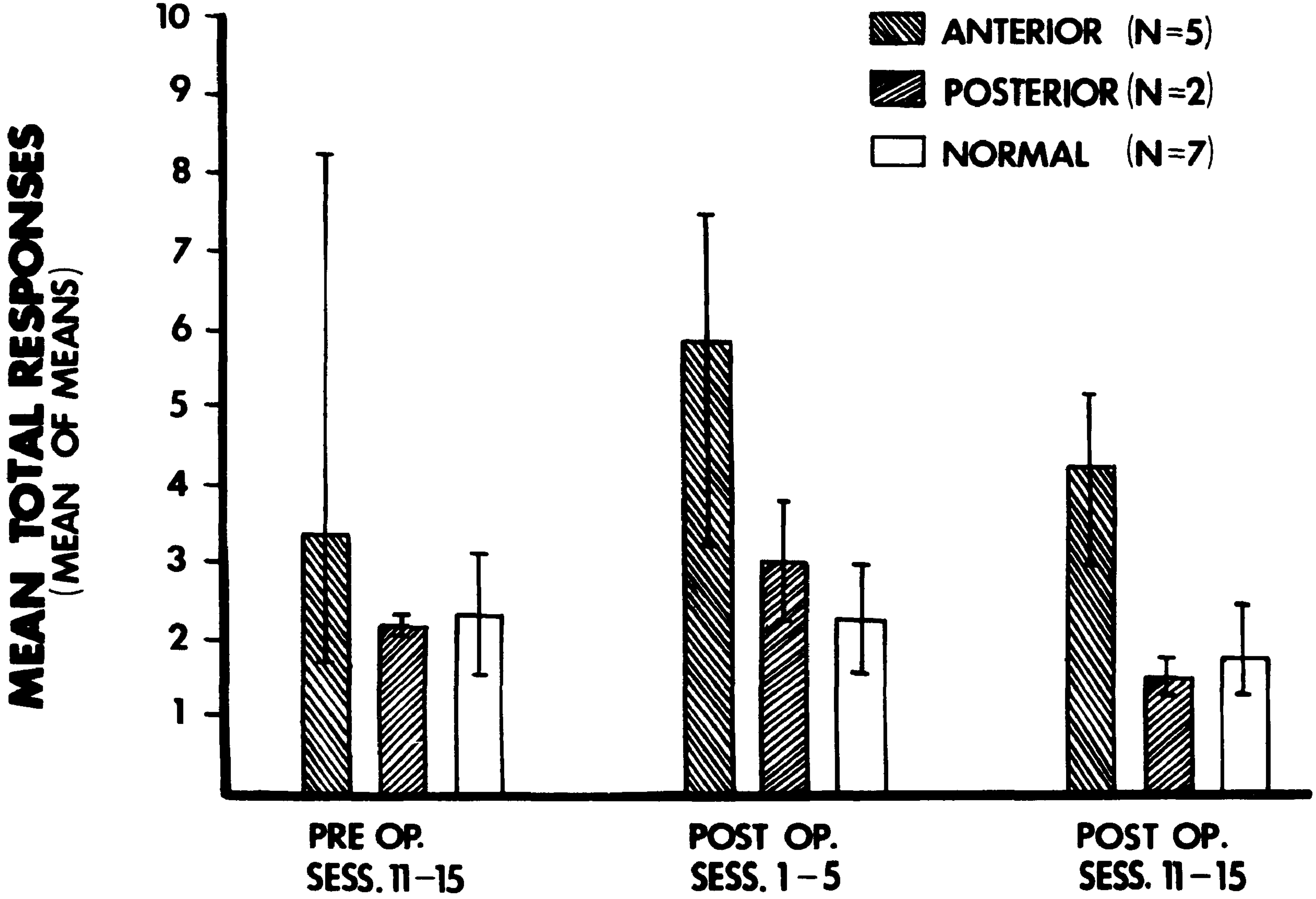

MEAN TOTAL RESPONSES
(MEAN OF MEANS)
10
9
8
7
6
5
4
3
2
1
ANTERIOR (N=5)
POSTERIOR (N=2)
NORMAL (N=7)
PRE OP. SESS. 11–15
POST OP. SESS. 1–5
POST OP. SESS. 11–15

FIG. 30. Means and ranges of the mean number of runs emitted per DRO period by the animals in each group over the last five pre-operative sessions, and first five and last five post-operative sessions. Each mean is an average of the mean number of runs per DRO for each of the sessions indicated by each animal in the group. The range shown is that of the individual means for each animal over the sessions indicated.

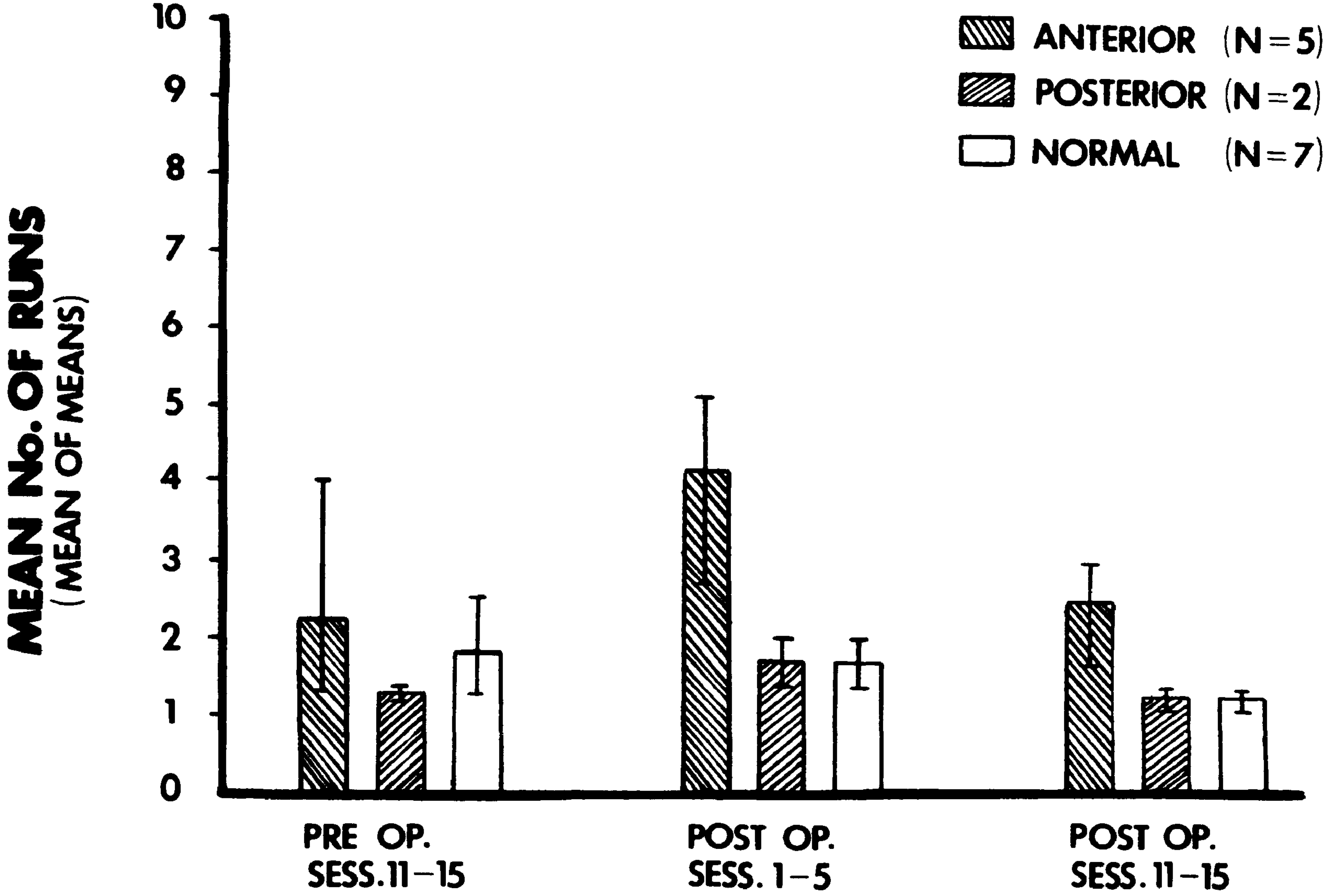
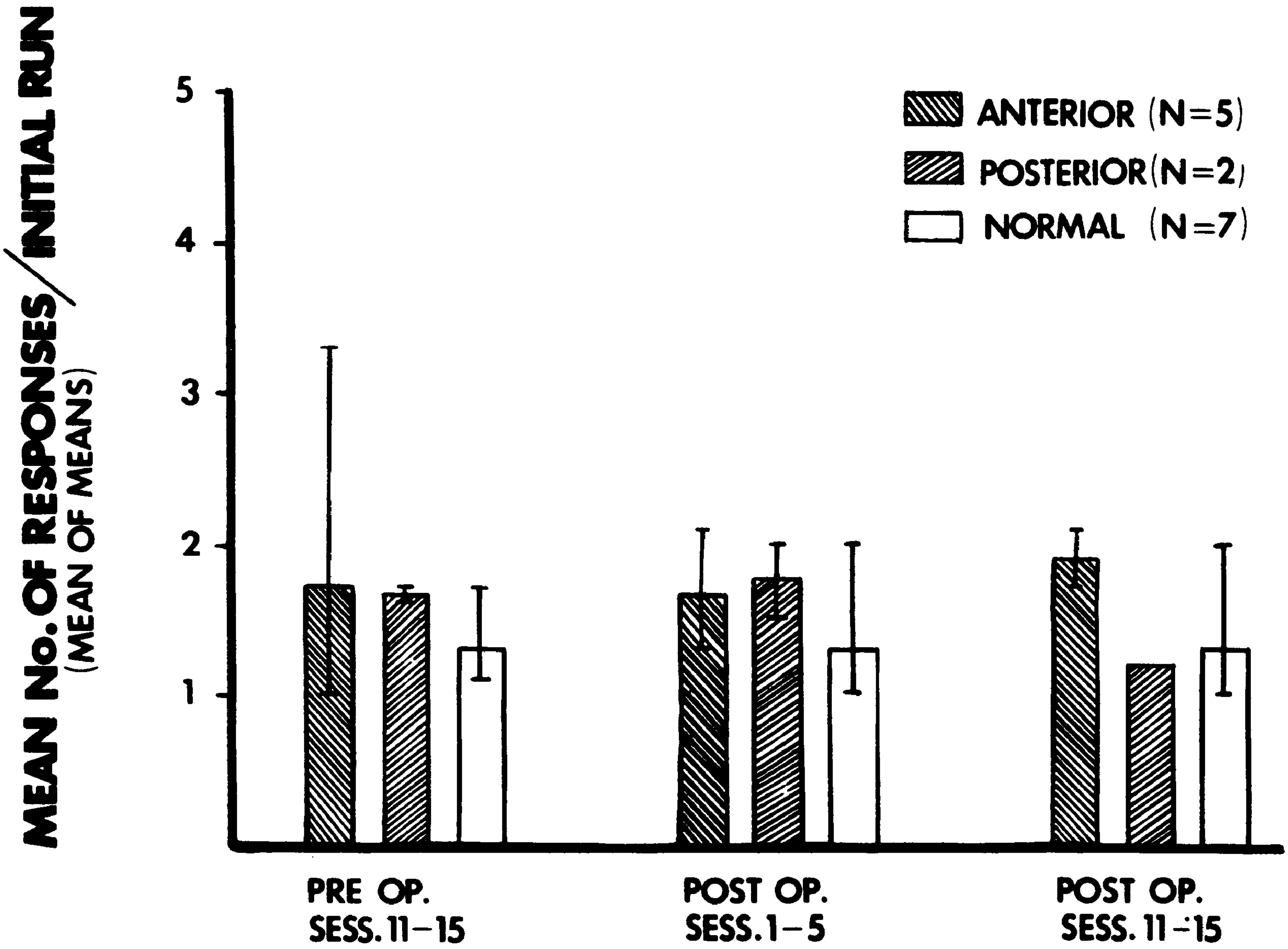

MEAN No. OF RUNS
(MEAN OF MEANS)
10
9
8
7
6
5
4
3
2
1
0
ANTERIOR (N = 5)
POSTERIOR (N = 2)
NORMAL (N = 7)
PRE OP.
SESS. 11–15
POST OP.
SESS. 1–5
POST OP.
SESS. 11–15

FIG. 31. Means and ranges of the mean number of responses emitted per initial run in each DRO period by the animals in each group over the last five pre-operative sessions, and first five and last five post-operative sessions. Each mean is an average of the mean number of responses per initial run for each of the sessions indicated for each animal in the group. The indicated range is that of the individual means for each animal over the sessions indicated.

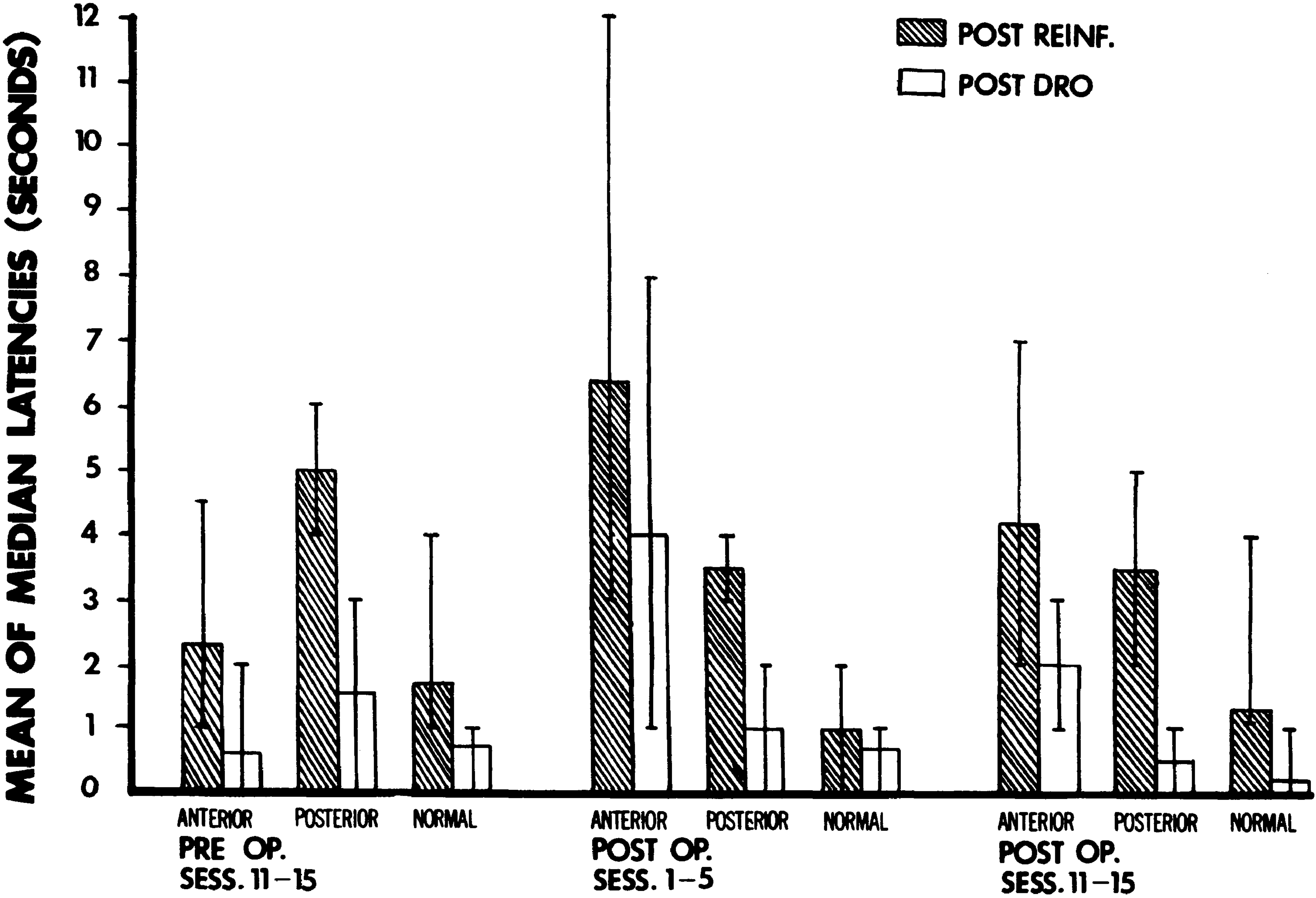

MEAN No. OF RESPONSES/INITIAL RUN
(MEAN OF MEANS)
5
4
3
2
1
▧ ANTERIOR (N=5)
▨ POSTERIOR (N=2)
☐ NORMAL (N=7)
PRE OP.
SESS. 11–15
POST OP.
SESS. 1–5
POST OP.
SESS. 11–15

normals (t = 7.10, d.f. = 10). Over the last five post-operative sessions the mean number of runs emitted by the anterior animals dropped to 2.5, but this was still significantly larger than the normal group's mean of 1.3 (t = 5.36, d.f. = 10).

Figure 31 compares the mean number of responses emitted per initial run in each DRO period by anterior, posterior and normal rats. Attention must again be called to the atypical performance of rat 191A which emitted a mean of 3.3 responses per initial run pre-operatively and was responsible for the wide range shown for the anterior group (see Figure 30). No other animal in either group exceeded a mean of 1.7 responses per initial run such that the difference between the anterior and normal pre-operative means was not significant (t = 1.18, d.f. = 10). Contrary to the post-operative increases shown by anterior rats with regard to both mean total DRO responses and mean number of runs in DRO, there was no corresponding increase in mean number of responses in initial runs over the first five post-operative sessions. The mean number of responses per initial run emitted by anterior animals dropped to 1.6 which was not significantly different from the mean of 1.3 (t = 1.63, d.f. = 10) for the normals over the first five post-operative sessions. By the last five post-operative sessions, the mean number of responses emitted per initial run by the anterior rats increased to 1.9 which was significantly higher than the corresponding mean of 1.3 the normal group (t = 3.33, d.f. = 10).

Figure 32 shows the means of median latencies of the first response in each reinforcement period for all three groups. It was apparent that the mean post reinforcement medians were longer than the mean post DRO medians. This difference was statistically significant (F = 12.50, d.f. = 1/10, $p < .01$).

FIG. 32. Means and ranges of median latencies of first response in reinforced periods for each group over the last five pre-operative, first five post-operative, and last five post-operative sessions. The shaded bars shown mean of median latencies following reinforcement periods. The unshaded bars show mean of median latencies following DRO periods. The indicated range is that of the individual means for each animal over the sessions indicated.


MEAN OF MEDIAN LATENCIES (SECONDS)
0
1
2
3
4
5
6
7
8
9
10
11
12
▨ POST REINF.
☐ POST DRO
ANTERIOR
POSTERIOR
NORMAL
PRE OP.
SESS. 11–15
ANTERIOR
POSTERIOR
NORMAL
POST OP.
SESS. 1–5
ANTERIOR
POSTERIOR
NORMAL
POST OP.
SESS. 11–15

As with both mean total responses per DRO and mean number of runs per DRO, the mean of the median latencies of the anterior group showed an increase over pre-operative levels while that of the normal and of the posterior animals did not. Pre-operative differences between anterior and normal means were not statistically significant. This was true for both post-reinforcement latencies (t = 0.12, d.f. = 10) and post DRO latencies (t = 0.26, d.f. = 10). Over the first five post-operative sessions the mean of the post-reinforcement median latencies of the anterior group increased from 2.3 to 6.4 seconds which was significantly greater than the corresponding mean of 1.0 seconds for the normal group (t = 2.93, d.f. = 10). The post DRO mean of the median of the anterior animals was 4.0 over the first five post-operative sessions which was also significantly greater than 0.4 for the normals (t = 3.76, d.f. = 10). By the last five post-operative sessions both the mean post-reinforcement and mean post DRO median latencies of the anterior group were still greater than those of the normal group (t = 3.99, d.f. = 10; t = 3.84, d.f. = 10 respectively).

DISCUSSION

Initial Comments

Animals with caudate nucleus lesions have been characterized as unable to inhibit previously reinforced responses or to shift from one mode of responding to another when reinforcement contingencies are altered. Following Dews (1958) and Thompson (1963a), the present study attempted to translate this characterization into terms consistent with current experimental analyses of behavior (e.g., Schoenfeld and Farmer, 1970; Skinner, 1970). Accordingly, caudate animals were described as exhibiting increased frequencies of response during non-reinforced components of schedules which alternate non-reinforcement with reinforcement, or which require the emission of "other" behavior in order to satisfy a contingency (e.g., delayed response, DRL, DRO). It was further argued that increased frequencies of response under conditions of non-reinforcement were augmented when the changes in contingency were not correlated with exteroceptive stimuli. It was assumed that in the absence of such exteroceptive cues animals came to discriminate schedule components on the basis of their own behavior (i.e., response produced stimuli) and that caudate lesions interfered in some way with the control of responding by such stimuli.

The present study used Mix FR DRO schedules as these schedules typified the type of task on which caudate lesioned animals are normally impaired. Experiment I was designed to study post-operative acquisition and steady state behavior maintained at several levels of the FR requirement in rats with anterior caudate lesions, rats with posterior caudate lesions, and in unoperated animals. Separate anterior and posterior lesion groups were employed with the intention of further exploring the functional heterogeneity of the rat caudate. A further purpose of Experiment I was to provide normative and functional data descriptive of behavior maintained on Mix FR DRO schedules, and to extend to mixed schedules Boren's (1961) study of resistance to extinction as a function of ratio size.

Experiment II was undertaken in order to examine the effects of caudate lesions on behavior acquired pre-operatively at one level of the Mix FR DRO schedule.

Acquisition and Steady State Behavior on Mix FR DRO

Cumulative records of both lesioned and unoperated rats in Experiment I were found to share certain topographic features with records generated by responding on Mix FR Ext schedules (Ferster and Skinner, 1957; Weissman, 1960; Thompson, 1963b). Steady state responding during unreinforced components of Mix FR DRO was marked by initial runs of responses followed by pauses of little or no responding which either continued until the end of the unreinforced period or were followed by additional runs. The typical "plateau" or "stair step" configurations which resulted from this behavior indicated that animals on these schedules discriminated the condition of extinction. That both mean total responses per DRO period and mean number of runs per DRO period decreased over sessions provided further evidence for the development of such a discrimination and was reminiscent of the effects of repeated conditioning and extinction reported by Bullock and Smith (1953) on a prototypical mixed schedule.

Of course, animals do not discriminate extinction as such, but can be said to respond differentially under conditions of reinforcement and non-reinforcement. On mixed schedules these conditions presumably cannot be detected until the animal has emitted the number of responses required by the ratio. The question of interest, therefore, is what stimulus or stimuli actually controlled differential responding?

For the purpose of an experimental analysis of behavior it has been assumed that whatever the stimulus, it is correlated with the number of responses emitted in the ratio. This assumption has been investigated in several studies by Mechner (1958a, 1958b) and Platt and Senkowski (1970). In Mechner's procedure rats were required to complete an FR on one lever before being reinforced for a single "reporting" response on a second lever. Switching to the reinforced lever before completion of the FR on the "counting" lever was never reinforced, and on such occasions of premature switching the rat had to repeat the entire sequence. In Platt and Senkowski's procedure the "reporting" response on the second lever was replaced by the interruption of a photo beam across the food hopper. The datum of interest on such schedules has been the number of responses emitted on the FR lever before switching to the second lever or before interruption of the photo beam. For the purposes of the present study number of responses on the FR lever was assumed to correspond to the number of responses emitted in an initial DRO run (i.e., number of responses before the first IRT greater than 3 seconds) on the Mix FR DRO schedule.

Table 3 compares measures of central tendency of distributions of the number of responses in runs on the FR "counting" lever from Mechner (1958a) and from Platt and Senkowski (1970). In the present study Mix FR DRO schedules tended to produce longer runs, especially at the FR 8 levels. This can be accounted for, however, by procedural differences. In both Mechner's and Platt and Senkowski's studies the sequences by which rats were exposed to FR requirements were counterbalanced whereas

TABLE 3

Comparison of Run Length Data with Other Studies

Study	Fixed Ratio 4	 8	 12	 16
Platt and Senkowski (1970)				
Mean chain length emitted* (N = 8 rats)	6.0	10.0	14.0	18.0
Mechner (1958a)				
Median length of runs*				
Rat N 1	6.0	10.0	15.0	19.5
Rat N 2	7.5	11.5	15.5	21.0
Rat N 3	5.5	9.5	15.0	19.0
Rat N 4	9.0	12.5	16.5	21.0
Rat N 5	5.5	11.0	14.5	19.0
Rat N 6	7.0	12.5	17.0	24.0
Mean	6.8	11.2	15.6	20.6
The present study (unoperated animals)				
Mean no. responses per initial run in DRO periods				
Rat 397	7.5	14.4	——	22.6
Rat 398	11.1	14.6	——	22.8
Rat 403	6.3	13.4	——	15.7
Rat 407	8.5	13.3	——	22.7
Rat 412	6.6	12.0	——	21.8
Mean	8.0	13.5	——	21.1

*Data are estimates on figures presented in the study.

in the present study animals were exposed in ascending order, but were given more sessions in which to stabilize at each level. Also, the methods by which run terminations were defined differed. In the present study completion of a run was "reported" by the emission of a 3 second pause rather than by a response on a second lever or by interruption of a photo beam. In view of these procedural differences the correspondence between central tendency of run length distributions is noteworthy. It should also be pointed out that Mechner used the median in reporting his run lengths, a measure not influenced by occasional long runs.

These correspondences in central tendency did not extend to measures of variability, however. Platt and Senkowski (1970) found that both the means and standard deviations of the run length distributions were linearly increasing functions of the fixed ratio. This enabled them to use the linear generalization of Weber's Law (i.e., $\Delta I = KI + C$, where C is one JND above absolute threshold) to provide discriminability functions for number of responses as a stimulus. In the present study results showed the standard deviation not to be a linear function of the ratio.

This raises the important question of whether or not, in the present study, the nonlinearity of increases in standard deviation of run length distributions as a function of the ratio reflected a fundamental difference in the nature of the behavior generated by Mix FR DRO schedules as opposed to Mechner's (1958a) procedure, or simply the failure to sustain responding at higher ratios due to inadequate control (e.g., inadequate magnitude of reinforcement). In this regard differences in procedure must again be called into account. One source of increased variation in run length produced on Mix FR DRO schedules may have been the method by which run termination was determined in the present study. Under the three second pause criterion it would be possible for an animal to emit n responses, visit the dipper (i.e., terminate its run), and then return to the lever to make additional responses before the three second period had elapsed. This being the case, the number of responses per run could have been confounded with the speed with which an animal approached the dipper and then returned to the lever. This situation could have been remedied by arranging that any approach to the dipper be recorded by interruption of a photo beam as in Platt and Senkowski's (1970) procedure.

A more critical difference between Mechner's (1958a) procedure and the Mix FR DRO schedule concerns the lack of immediate consequences for either short or long runs in DRO. In Mechner's procedure short runs (i.e., switching to the reinforced lever before completing the FR) were not reinforced and required repetition of the entire sequence. In DRO, as long as at least one response was emitted, runs shorter than the FR had no immediate differential consequence. Furthermore, in the Mechner procedure, completed runs on the FR lever were quickly reinforced when the appropriate "reporting" response was made, while initial runs emitted in DRO in the present study did not have any immediate consequence. In other words, the contingency in effect in the DRO component of the mixed schedule did not involve the immediate differential reinforcement of runs containing the number of responses required by the FR. The primary source of control of DRO run lengths most probably resided, therefore, in induction from ratios emitted in reinforced components of the schedule. Since completion of the ratio in reinforced components was always exteroceptively cued by the operation of the dipper mechanism, "counting" (i.e., run length differentiation controlled by interoceptive stimuli) was not actually required by the Mix FR DRO schedule.

Another factor which must be considered is that on Mix FR DRO schedules the interpolation of the DRO contingency itself may have affected the extent to which number of responses functioned as a stimulus. While the contingency inherent in FR schedules is one in which reinforcement density is directly proportional to response density (i.e., rate of response), the contingency inherent in DRO is the opposite. The interpolation of the DRO contingency could therefore create a situation which would maximize the tendency of ratio behavior to break down as ratio requirements are increased. Rats maintained on simple FR schedules have shown declines in response rate at about FR 30 or 40 (Barofsky and Hurwitz, 1968). These ratios were much higher than the ratios at which responding in the present study appeared to break down. According to Powell (1970), when the duration of the post reinforcement pause is excluded from the calculation of response rate, decreases at higher ratios are seen to result from increases in the frequency and duration of within run pauses rather than from an increase in interresponse times generally. In Experiment I of the present study evidence of such increases in frequency and duration of within run pausing was seen in the cumulative records at the FR 8 and 16 levels. This resulted in situations in which, during a reinforcement period on FR 16 for example, an animal might emit 10 responses, pause for some seconds, and then emit the remaining six responses and be reinforced. Such a series of events could have destroyed the correlation between the number of responses and delivery of reinforcement, by which number of responses is assumed to acquire its stimulus function.

In spite of the foregoing discussion responding on Mix FR DRO schedules was found to reflect properties of fixed ratio responding generally. Both the mean total responses emitted per DRO and the mean number of responses emitted per initial run in DRO were found to be increasing functions of ratio size. Both findings support and extend Boren's (1961) findings concerning resistance to extinction as a function of ratio size. Furthermore,the fact that the means of the median latencies of the first response in reinforcement periods were found to increase with increases in ratio size agrees with reports that post reinforcement pause duration increases as the ratio is increased (Felton and Lyon, 1966).

Effects of Caudate Lesions

Since data from Experiment I both failed to provide discriminability functions for number of responses as a stimulus and reveal differences in performance between lesioned and unoperated rats, no statement can be made with regard to the hypothesis that caudate lesioned animals are somehow impaired on tasks which require discrimination of response produced stimuli. What remains is to account for the absence of a lesion effect in Experiment I in view of its presence in Experiment II, and to attempt a characterization of the Experiment II effect in the light of the present data.

It could be argued that lesion effects were obscured by increased variability both within animals and groups due to difficulties in maintaining performances at the higher FR levels. This would not, however, account for the lack of any effect at the FR 1 level where behavior is presumably unaffected by processes associated with ratio responding or intermittent reinforcement generally.

It has been argued that performance deficits shown by caudate lesioned animals may result from lesion induced hyperactivity (e.g., Whittier and Orr, 1962). It might be, therefore, that the failure of Experiment I to reveal a significant lesion effect resulted from the failure of the lesions to produce the necessary increases in activity levels. The failure of Experiment I to show differences in number of activity cage tilts between any of the groups cannot be taken in support of such an argument, however. Activity measures were not made until the completion of testing at all FR levels of the mixed schedule; about a year following surgery. Since caudate lesion induced hyperactivity is reported to diminish within one to six weeks following surgery, the measures from Experiment I do not really bear upon the hypothesis (Whittier and Orr, 1962; Green, _et_ _al_., 1967; Albert and Bignami, 1968). It is also possible that the cursive hyperkinesia reported by Whittier and Orr (i.e., uninterrupted bouts of running in wheels, etc.) is not revealed in tilt boxes or stabilimeter cages. Lynch (1970) has suggested that there are two separate forebrain systems in the rat which mediate different types of activity. One kind is better revealed in running wheels while the other is better revealed in stabilimeter cages. It is of interest that Lynch's caudate rats failed to show any post-operative hyperkinesia in stabilimeter cages which were similar in design to the tilt boxes used in the present study.

Another reason for the absence of a lesion effect in Experiment I may have been lesion size and/or extent. Table 4 gives estimated percent caudate tissue damaged in a number of recent studies. When compared with the percent damage in the present study (see Tables 3 and 1 in Appendices I and II respectively) it can be seen that the lesions in

TABLE 4

Estimates of Percent Caudate Tissue Destroyed in Recent Studies of the Behavioral Effects of Lesions of the Caudate Nucleus[1]

Study	Percent Tissue Destroyed
Albert & Bignami (1968)	10 - 20
Allen, _et al_. (1972)	5 - 25
Borst, _et al_. (1970)	10 - 30
Chorover & Gross (1963)	12 - 40 @
Divac (1971)	25 - 30
Green, _et al_. (1967)	20 @
Gross, _et al_. (1965)	no information
Hansing, _et al_. (1968)	20 - 30
Kirkby & Kimble (1968)	25 - 85 @
Meyer (1967)	7 - 40
Mikulas (1966)	10 - 15 @
Mikulas & Isaacson (1968)	15 @
Mitcham & Thomas (1972)	5 - 15
Neill & Grossman (1970)	"small" @
Potegal (1969)	10 - 50 @
Schmaltz & Isaacson (1968)	1.3 - 20.4 @
Schwartzbaum & Donovick (1968)	15 - 20 @
Whittier & Orr (1962)	no information
Winocur & Mills (1969)	12 - 25

---

[1] Except where indicated all estimates are based on inspection of photographs and/or reconstructions provided in the published report.

@Estimate reported by the investigator(s).

the present study were relatively small. Lesions in Experiment II, however, evidenced slightly greater percent tissue destruction as well as greater anterior to posterior extent. Since the probability of appearance of a caudate lesion effect has been shown to be related to lesion size, the somewhat larger lesions in Experiment II may be called into account for the effect shown in that experiment (Thompson, 1959; Thompson and Mettler, 1963).

The question of lesion size must, however, be considered in relation to lesion locus. A relatively large lesion may be effective, not because of mass action, but because the larger lesion more probably involved a critical area. The anterior lesions in Experiment II, in addition to being slightly larger, also extended further anteriorally. That a caudate lesion effect of the type revealed in Experiment II should depend upon the anterior extent of the lesions is suggested by the performance of rats 405A and 414A at the FR 1 level in Experiment I. Both rats showed greater mean number of responses per DRO period at that level than did any other animal, and both showed the greatest anterior extent of lesion. Of these two animals, rat 414A had the greater anterior extent and continued to maintain the highest response output of any animal in Experiment I. That the caudate lesion effect in the present study depended upon the anterior extent of the lesions is also supported by the apparent absence of any lesion effect in posterior animals in either Experiments I or II. Such evidence is far from conclusive, but does suggest an avenue for further study.

With regard to the performance of the posterior rats, it must be added that the posterior lesions were also placed more dorsally than were the anterior lesions. It may be, therefore, that the dorsal-ventral axis is the more critical as has been suggested by Neill and Grossman (1970). In this regard it should be recalled that Meyer (1967) failed to show a caudate lesion effect with rats on a DRL schedule while Schmaltz and Isaacson (1968) reported an effect using DRL. Meyer's lesions were placed more dorsally than those of Schmaltz and Isaacson.

It is also possible that variables such as lesion size and locus may interact with procedural variables. Thompson (1959) suggested that when caudate lesions are small their effects may be better revealed in post-operative retention of behavior acquired pre-operatively than in post-operative acquisition. The results of Experiment II in the present study support this contention as do several other studies involving the testing of caudate lesioned rats on schedules of reinforcement. The previously discussed discrepancy between the studies of Schmaltz and Isaacson (1968) and Meyer (1967) may be accounted for in this manner. Schmaltz and Isaacson reported increased rates of responding in caudate lesioned rats on DRL schedules when animals were tested for post-operative retention, while Meyer, whose caudate rats acquired the DRL behavior post-operatively, did not report an effect. Similarly, Unger (1966) failed to show a caudate lesion effect in rats which acquired FI responding post-operatively, while Hansing, *et al.*, (1968) did show increased responding on FI post-operatively following pre-operative acquisition.

Characterization of Caudate Lesion Effects

Data from Experiment II showed that rats with lesions of the anterior caudate nucleus emitted more responses in DRO periods than did either posterior caudate rats or unoperated controls. Furthermore, this increase in response output during DRO was related to increases in the number of individual response runs in DRO (i.e., single responses separated by IRTs of 3 seconds or greater) rather than increases in the number of responses emitted per run. These findings are in agreement with other reports of increased responsiveness on schedules of reinforcement in caudate lesioned rats as well as caudate lesioned monkeys (Thompson, et al., 1962; Thompson, 1963a, 1963b; Unger 1966; Hansing, et al., 1968; Schmaltz and Isaacson, 1968). Such increased rates of responding, however, do not lend themselves to characterization as failures of "inhibition". According to both Rescorla (1969), Hearst (1972), and Hearst, Besley, and Farthing (1970) the term inhibition should be applied only within the operational context of specific experimental criteria. These criteria appear to agree on the necessity of establishing a baseline against which to measure decreases in response frequency or other response decrements (e.g., decreased magnitude, increased latency, etc.). Given such a requirement it would appear that a demonstration of disinhibition, or failure of inhibition, would depend upon first having satisfied the criteria for the demonstration of inhibition. Neither the present study nor those just cited in agreement with the present study satisfy this requirement.

The failure of the caudate lesioned rats in Experiment II to regain pre-operative performance levels, especially the increased number of runs per DRO, could be construed as a "memory" deficit. Such an explanation is particularly tempting in view of recent investigations which suggest that stimulation of the caudate nucleus has effects similar to those of electroconvulsive shock (Wyers, Peeke, Williston, and Herz, 1968; Wyers and Deadwyler, 1971). The difficulty with a memory explanation of the lesion effect in the context of the present study, however, resides in the fact that from the point of view of an experimental analysis of behavior, such an "explanation" may be no less an appeal to a verbalism than an inhibition hypothesis. Ervin and Anders (1970) suggest that the term memory be restricted to those experiments in which behavioral changes occurring between successive observations can be functionally related to the duration of the interval between the two observations. The present experiment does not conform to such a design.

This is not to argue against a possible role for the caudate in whatever processes are encompassed by the term "memory". It is to state that in the interests of an experimental analysis of behavior, it does not appear operationally sound to introduce the concept of memory any more than the concept of inhibition in an attempt to characterize the present results.

In addition to the post-operative increases in DRO responding shown in Experiment II, an observation at the FR 4 level of Experiment I is of interest. At that level anterior rats emitted a higher proportion of initial runs in DRO which contained only a single response than did any of the animals in either of the two groups. This finding appears similar to Unger's (1966) observation that, using Mechner's (1958a) two lever procedure, caudate lesioned rats tended to shift from lever one (the FR "counting" lever) to lever two (the reinforced "reporting" lever) before completing the FR requirement on lever one. This, together with the finding that the lesioned rats tended to persist in responding on the reinforced lever, led Unger to argue that "perseveration" on the part of caudate lesioned rats was better characterized as the persistence of previously reinforced responses under conditions in which reinforcement is withheld. The results from Experiment II may be construed in a similar fashion. That is, increased responding during DRO periods, being characterized by the emission of single responses spaced by pauses of at least three seconds, could, applying Unger's reasoning, be taken as evidence that the lesioned rats persisted in the emission of that response sequence which had been most frequently reinforced, i.e., a bar press followed by an approach to the dipper. A much stronger case could be made for such a hypothesis, of course, if data reflecting the frequency and sequential nature of dipper approaches were available.

If such data were available, and did reveal the persistence of such sequences, it would support the argument that caudate rats tend to repeat those responses which are closer in time or space to the presentation of a terminal primary reinforcer. That is, the more contiguous a response with primary reinforcement the more probable that a caudate lesioned rat would persist in the emission of that response. This could be interpreted either in terms of increased effectiveness of the terminal reinforcer, or a failure to occasion "other" behavior due to a weakening of control exercised by conditioned reinforcers in proportion to their degree of discontiguity with primary reinforcement.

Both interpretations are admittedly speculative and problematic. Increases in the latency of the first response in reinforcement periods shown by anterior rats, for example, could reflect either an increase or a decrease in reinforcer "effectiveness." If increased latencies could be demonstrated to involve increased time spent in licking and sniffing about the dipper area this would support a reinforcer "effectiveness" hypothesis. On the other hand, increases in duration of post reinforcement pauses on FR schedules are traditionally associated with decreased reinforcer effectiveness resulting from satiation (Sidman and Stebbins, 1954). Also, Experiment I found that both anterior and posterior caudate rats showed decreases in water consumption following surgery, and such an observation is not consistent with the contention that caudate lesions increase reinforcer effectiveness.

It is worth digressing at this point to remark that although the absence of a sham operated control in Experiment I prevents attributing the decreased water consumption to caudate damage as opposed to surgical trauma, similar decreases in water consumption were reported by Whittier and Orr (1962). Informal observations made during Experiment I indicated that in the cases of two of the anterior rats decreased water consumption involved motor "awkwardness" in approaching the drinking devices. Whittier and Orr made similar observations and since their lesions corresponded more closely in placement to the anterior lesions in the present study it is conceivable that the anterior caudate, or fibers of passage, are involved in the motoric components of drinking and/or feeding behavior. This appears to be worthy of further investigation in view of the possible participation of the nigrostriatal pathway in the reproduction of the lateral hypothalamic syndrome (Fitzsimons, 1972).

In addition to possible changes in reinforcer effectiveness it was also postulated that the persistence of certain responses or response sequences by caudate rats might be described in terms of a weakening of control exercised by conditioned reinforcers temporally or spatially discontiguous with a terminal primary reinforcer. Such a contiguity hypothesis is attractive by virtue of its similarity to the position advanced by French (1964) concerning the role of "association" in frontal lobe function in monkeys. French argued that frontal lobe damage appeared to impair a monkey's ability to deal with discontiguity. If it could be demonstrated that a similar impairment resulted from damage to the rat caudate, this would be consistent with Divac's (1968b) hypothesis that the caudate nucleus of species such as the rat subserves functions similar to the cortex of "higher" species. If the contiguity hypothesis has merit, it is conceivable that stimulus contiguity rather than modality is more critical to a behavioral analysis of caudate function. Indeed, since the caudate nucleus receives fibers from virtually all sensory cortices and has a comparably limited output, it would seem anatomically well situated for such a modality non-specific function (Divac, 1968; Kemp and Powell, 1970).

While the contiguity hypothesis just outlined appears more attractive than the reinforcer effectiveness hypothesis, both, as previously stated, are speculative in the absence of direct empirical support from present data. However, both hypotheses lend themselves to experimental analysis. Reinforcer effectiveness could be further explored, for example, through the use of progressive ratio schedules as employed by Hodos (1961). The weakening of control over behavior by conditioned stimuli in proportion to degree of discontiguity from primary reinforcement might be studied through the use of chain schedules (Kelleher and Gollub, 1962; Kelleher, 1966). The latter type of investigation might be particularly fruitful if carried out in conjunction with tandem schedules in view of their relation to mixed schedules (i.e., the lack of exteroceptive discriminative stimuli).

Concluding Remarks

Due to the fact that Experiment I did not provide run length data which could be described by Weber's Law, and also failed to show a lesion effect, the positive results from Experiment II cannot be characterized in terms of an inability to discriminate or otherwise use response produced stimuli. However, since the contingencies inherent in the Mix FR DRO schedule did not maximize run length differentiation, the issue in general remains open.

The finding of Experiment II, that rats with anterior caudate lesions emit more responses during the DRO component of a Mix FR 1 DRO schedule, agrees with previous studies of schedule retention in both caudate lesioned rats and monkeys. This finding was not characterized as either a loss of response inhibition or of memory. In the interests of an experimental behavioral analysis of brain function, appeals to such processes should be restricted to those experimental procedures which give them operational credence. Furthermore, the facts that increased DRO responding was characterized by increases in the number of runs more than by increases in the number of responses per run, and that both post DRO and post FR latencies increased post operatively, argue against any interpretation in terms of simple perseveration of reinforced responses.

For the present, an interpretation of the lesion effect in Experiment II in terms of reduced control by conditioned reinforcers over the occasioning of "other" behavior, or an inability to deal with discontiguity appears to be a fruitful avenue for further exploration.

APPENDIX I

TABLE A

Sequences Used to Program Alternation of DRO and Reinforcement Periods on the Mix FR DRO Schedule*

	Top Bank of Stepping Switch											
Gellerman #2	0	0	0	1	1	0	1	1	0	1	0	(to series #6)
Gellerman #10	0	0	1	1	1	0	0	0	1	1	1	
Gellerman #7	0	0	1	1	0	0	1	1	0	1	1	
	Bottom Bank of Stepping Switch											
Gellerman #5	0	0	1	0	1	1	1	0	0	1	0	(to series #7)
Gellerman #4	0	0	1	0	1	1	0	0	1	1	1	
Gellerman #6	0	0	1	1	0	0	1	0	1	1	0	

---

*Each "0" represents a DRO period and each "1" an FR period. The first ten digits in each row are series taken from Gellerman (1933) as indicated. Series #10 was modified such that steps 8 and 9 are reversed from the original. This was done so that the probabilities of DRO following DRO, DRO following FR, FR following DRO, and FR following FR would be approximately equal. In all cases these probabilities were either 0.48 or 0.50. The 11th position in each row was wired to conform both with the design of the stepping switch (i.e., to permit switching of ground from top to bottom bank and back again), and to maintain the indicated probabilities. Each session started at the beginning of a series (leftmost position) with the starting row counterbalanced over sessions.

TABLE B

Estimates of Percent Caudate Tissue Destroyed and Anterior and Posterior Bilateral Extent of Lesions

Rat and Group	Percent Tissue Destroyed+	Extent of Lesion*			
		Left Side		Right Side	
		Anterior	Posterior	Anterior	Posterior
Anterior					
395A	44%	4.0	2.0	4.0	2.0
405A	14%	4.0	2.0	4.2	2.0
414A	15%	4.4	0.8	4.4	0.8
416A	9%	3.6	2.0	4.0	2.4
Posterior					
396P	7%	1.6	-0.4	0.8	-0.4
400P	14%	2.2	0.4	1.6	0.4
402P	6%	0.6	-1.0	0.6	-1.0
410P	17%	0.8	-1.2	0.8	-1.2
415P	12%	1.0	-1.0	1.2	0.8

---

*All stereotaxic coordinates are relative to bregma, atlas of Pellegrino and Cushman (1967).

+Refers to percent tissue destroyed within that area of caudate containing the lesion, not to percent of total caudate.

TABLE C

Total Number of Responses Per DRO Period: Means and Standard Deviations for the Last Ten Sessions at Each Level of the Fixed Ratio Requirement

Rat and Group	Fixed Ratio Requirement									
	FR 1	(1st)	FR 1	(2nd)	FR 4		FR 8		FR 16	
	M	SD	M	SD	M	SD	M	SD	M	SD
Anterior										
395A	2.6	1.6	1.8	1.3	11.7	8.6	21.1	14.3	29.3	15.6
405A	7.3	7.3	2.9	2.3	17.8	13.2	16.8	9.5	14.2	13.7
414A	10.0	7.6	5.0	3.3	18.4	10.3	30.9	16.9	55.0	38.3
416A	2.8	1.6	---	---	7.7	4.0	15.7	7.7	----	----
Mean	5.7	4.5	3.2	2.3	13.9	9.0	21.1	12.1	32.8	22.5
Posterior										
396P	2.8	2.3	3.3	2.5	8.5	4.1	15.0	9.3	28.7	13.6
400P	2.7	2.1	2.9	1.8	10.4	5.2	12.5	4.8	22.3	9.1
402P	2.0	1.8	1.9	1.9	9.1	3.7	14.8	6.6	24.2	9.3
410P	1.3	0.6	1.7	1.2	7.8	3.5	12.4	5.6	22.5	12.6
415P	2.2	1.1	2.5	1.5	9.9	6.5	14.7	9.0	33.1	18.0
Mean	2.2	1.6	2.5	1.8	9.1	4.6	13.9	7.0	26.2	12.5
Normal										
397N	4.8	3.7	4.8	4.1	12.4	9.7	28.0	35.5	48.0	29.8
398N	3.3	2.5	2.0	1.6	14.7	8.0	19.5	10.3	28.7	20.4
403N	2.5	1.7	3.1	1.9	7.9	4.6	15.6	6.1	35.1	20.1
407N	2.2	1.5	2.5	1.8	9.4	3.3	14.3	4.8	25.2	12.4
412N	1.4	0.8	1.9	1.7	7.8	3.8	14.4	7.5	25.6	13.1
Mean	2.8	2.0	2.9	2.2	10.4	5.9	18.4	12.8	32.5	19.2

TABLE D

Number of Runs Per DRO Period: Means and Standard Deviations for the Last Ten Sessions at Each Level of the Fixed Ratio Requirement

Rat and Group	Fixed Ratio Requirement									
	FR 1 (1st)		FR 1 (2nd)		FR 4		FR 8		FR 16	
	M	SD	M	SD	M	SD	M	SD	M	SD
Anterior										
395A	1.5	1.0	1.4	0.8	2.6	2.0	2.7	2.7	1.8	1.2
405A	2.8	2.5	1.6	1.1	2.0	1.5	2.0	1.2	5.7	5.1
414A	3.7	2.5	2.2	1.5	2.7	1.5	1.7	0.9	2.3	1.9
416A	1.5	0.9	---	---	1.4	0.9	1.4	0.8	---	---
Mean	2.4	1.7	1.7	1.1	2.2	1.5	2.0	1.4	3.3	2.7
Posterior										
396P	2.0	1.8	1.2	0.5	1.4	1.0	2.3	1.7	1.8	1.0
400P	1.6	1.3	1.2	0.6	1.4	0.9	1.1	0.4	1.3	1.0
402P	1.5	1.4	1.1	0.4	1.2	0.5	1.3	0.6	1.3	0.6
410P	1.6	0.5	1.2	0.6	1.3	0.6	1.5	0.9	1.6	1.1
415P	1.3	0.6	1.3	0.7	1.8	1.3	1.5	1.0	1.6	1.1
Mean	1.6	1.1	1.2	0.6	1.4	0.9	1.5	0.9	1.5	1.0
Normal										
397N	2.4	1.9	1.9	1.5	3.2	3.6	6.3	13.8	4.1	3.6
398N	2.2	1.6	1.2	0.5	2.2	1.9	2.2	1.4	1.7	1.2
403N	1.6	1.0	1.4	0.7	1.4	0.8	1.3	0.6	3.5	2.5
407N	1.6	1.0	1.4	0.7	1.4	0.7	1.2	0.5	1.6	0.9
412N	1.3	0.7	1.3	0.8	1.4	0.8	1.5	0.8	1.3	0.8
Mean	1.8	1.2	1.4	0.8	1.9	1.6	2.5	3.4	2.4	1.8

TABLE E

Initial Run Lengths in DRO Periods: Mean and Standard Deviations for the Last Ten Sessions of Each Level of the Fixed Ratio Requirement

Rat	Fixed Ratio Requirement									
and	FR 1	(1st)	FR 1	(2nd)	FR 4		FR 8		FR 16	
Group	M	SD	M	SD	M	SD	M	SD	M	SD
Anterior										
395A	2.0	1.1	1.4	0.8	6.5	3.7	11.7	5.0	21.9	11.7
405A	3.1	2.4	2.0	1.0	11.0	5.1	10.9	4.0	2.6	2.1
414A	2.4	2.5	2.5	1.8	9.4	7.1	22.5	8.7	36.8	13.2
416A	2.2	1.0	---	---	6.5	2.4	12.9	6.1	----	----
Mean	2.4	1.8	2.0	1.2	8.4	4.6	14.5	6.0	20.4	9.0
Posterior										
396P	1.7	0.8	2.7	1.7	7.3	2.4	10.0	6.0	20.9	10.5
400P	1.8	0.9	2.6	1.3	8.7	3.1	12.0	4.2	19.4	6.0
402P	1.4	0.6	1.7	1.7	8.4	3.1	13.3	4.6	21.3	6.5
410P	1.1	0.2	1.4	0.7	7.0	2.3	9.9	3.6	16.8	10.6
415P	1.9	0.8	2.2	0.9	7.3	3.1	11.9	4.2	26.6	11.6
Mean	1.6	0.7	2.1	1.3	7.7	2.8	11.4	4.5	21.0	9.0
Normal										
397N	2.4	1.2	3.0	1.9	7.5	3.1	14.4	5.9	22.7	15.7
398N	1.7	0.9	1.7	1.4	11.1	3.8	14.6	5.6	22.8	9.8
403N	1.8	0.8	2.4	1.1	6.3	2.5	13.4	3.4	15.7	10.6
407N	1.6	0.8	1.9	1.2	8.5	2.4	13.3	2.8	22.7	9.5
412N	1.0	0.2	1.5	1.0	6.6	2.6	12.0	3.8	21.8	7.9
Mean	1.7	0.8	2.1	1.3	8.0	2.9	13.5	4.3	21.1	10.7

TABLE F

Mean Total Number of Responses Emitted Per DRO Period for First Five Sessions at Each Level of the Fixed Ratio Requirement

Rat and Group	Ratio Requirement FR 1 (1st)	FR 1 (2nd)	FR 4	FR 8	FR 16
Anterior					
395A	13.1	8.6	17.0	17.8	29.3
405A	14.5	13.2	14.2	24.0	29.6
414A	12.5	23.6	34.8	50.2	15.3
416A	7.3	N.A.	14.7	16.0	15.4
Mean	11.9	15.3	20.2	27.0	22.4
Posterior					
396P	10.7	10.8	7.0	11.6	32.4
400P	5.0	7.6	11.9	17.0	27.1
402P	8.9	11.6	9.8	19.2	31.7
410P	7.1	7.7	13.1	12.4	24.2
415P	7.4	9.8	11.3	13.6	25.7
Mean	7.8	9.3	10.6	14.8	28.2
Normal					
397N	16.5	13.8	20.0	48.9	37.2
398N	11.6	8.0	18.6	22.7	42.3
403N	9.0	11.9	10.7	13.7	19.0
407N	9.2	9.0	12.9	14.8	21.6
412N	5.3	7.7	10.1	13.4	28.6
Mean	10.3	11.5	14.5	22.7	29.7

TABLE G

Mean Number of Runs Per DRO Period for First Five Sessions at Each Level of the Fixed Ratio Requirement

Rat and Group	Ratio Requirement FR 1 (1st)	FR 1 (2nd)	FR 4	FR 8	FR 16
Anterior					
395A	8.1	3.6	6.3	2.8	3.7
405A	4.3	4.6	3.8	1.9	3.4
414A	6.7	2.6	6.3	3.3	4.1
416A	3.7	N.A.	5.5	4.4	4.5
Mean	5.7	3.6	5.5	3.1	3.9
Posterior					
396P	6.9	3.5	3.3	2.1	3.0
400P	3.0	1.4	5.3	1.6	2.3
402P	6.3	2.0	2.2	5.9	6.9
410P	5.8	2.3	6.3	1.9	1.8
415P	5.1	1.7	3.5	1.9	1.9
Mean	5.4	2.2	4.1	2.7	3.2
Normal					
397N	8.3	2.5	5.8	6.2	9.3
398N	8.0	2.3	5.5	3.6	6.5
403N	5.3	2.4	2.8	2.1	2.5
407N	6.2	2.0	2.0	1.4	2.6
412N	4.6	1.8	3.0	1.4	2.2
Mean	6.5	2.2	3.8	2.9	4.6

TABLE H

Mean Number of Responses Emitted in Each Initial Run in DRO Period for the First Five Sessions at Each Level of the FR Requirement

Rat and Group	Ratio Requirement FR 1 (1st)	FR 1 (2nd)	FR 4	FR 8	FR 16
Anterior					
395A	1.9	3.9	5.1	10.5	10.7
405A	4.8	3.2	3.7	15.7	11.4
414A	2.6	9.1	8.0	15.1	24.4
416A	2.5	N.A.	4.9	8.2	13.2
Mean	3.0	5.4	5.4	12.4	14.9
Posterior					
396P	2.2	5.1	4.7	8.5	20.0
400P	2.2	6.1	3.9	13.8	21.3
402P	1.7	7.6	6.7	11.0	14.1
410P	1.3	3.8	2.8	9.9	16.9
415P	1.9	6.9	6.4	11.3	20.3
Mean	1.9	5.9	4.9	10.9	18.5
Normal					
397N	2.5	9.3	11.3	14.5	19.1
398N	1.8	5.5	10.3	14.9	14.6
403N	2.0	5.8	5.9	9.7	2.5
407N	2.2	5.4	9.9	13.7	12.5
412N	1.2	5.4	4.8	11.7	20.0
Mean	1.9	6.3	8.4	12.9	13.7

FIG. A. Milliliters of water consumed per kilogram of body weight for individual rats over five days before and eight days following surgery in Experiment I. Dotted line indicates day of surgery during which water consumption measures were not taken.

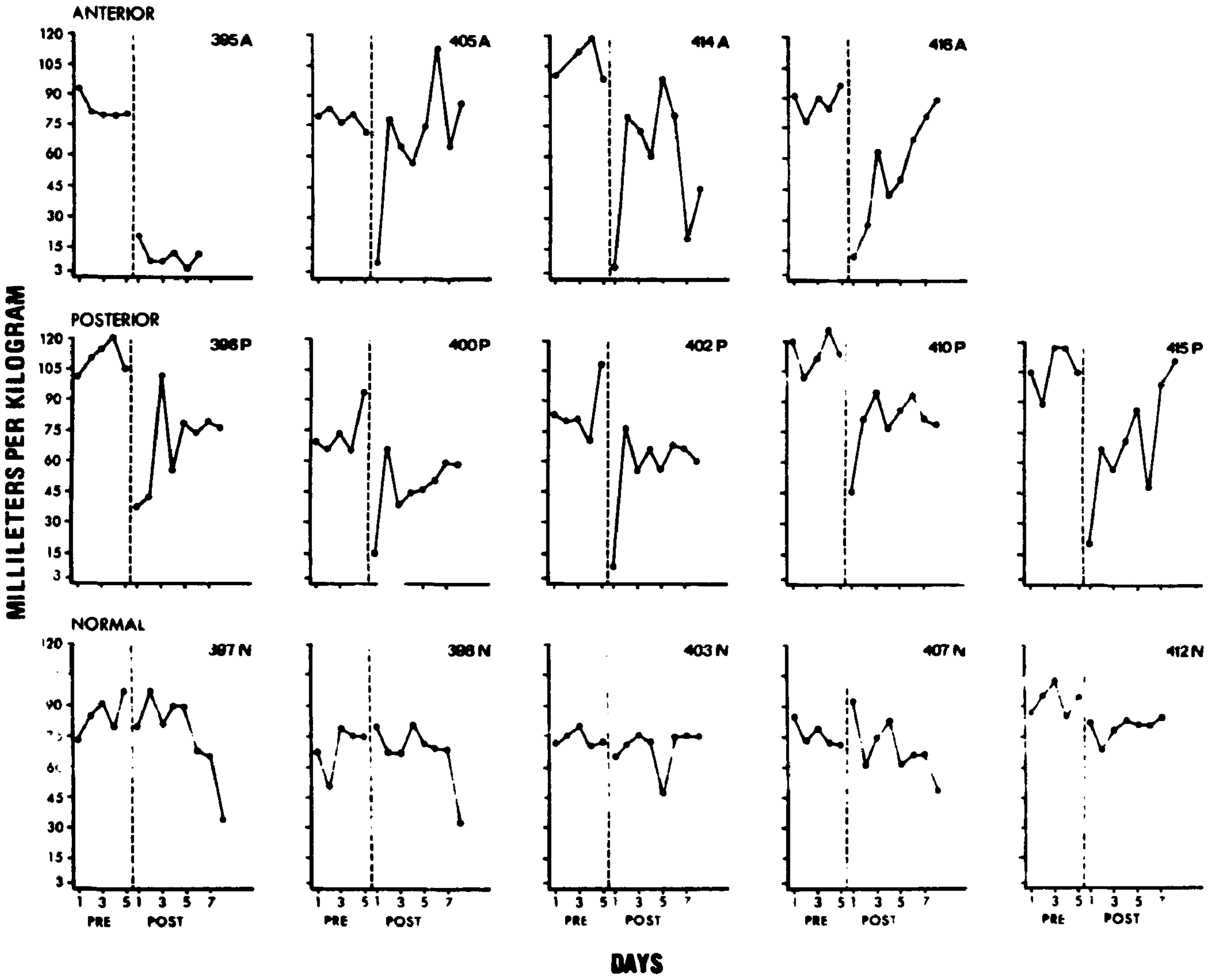
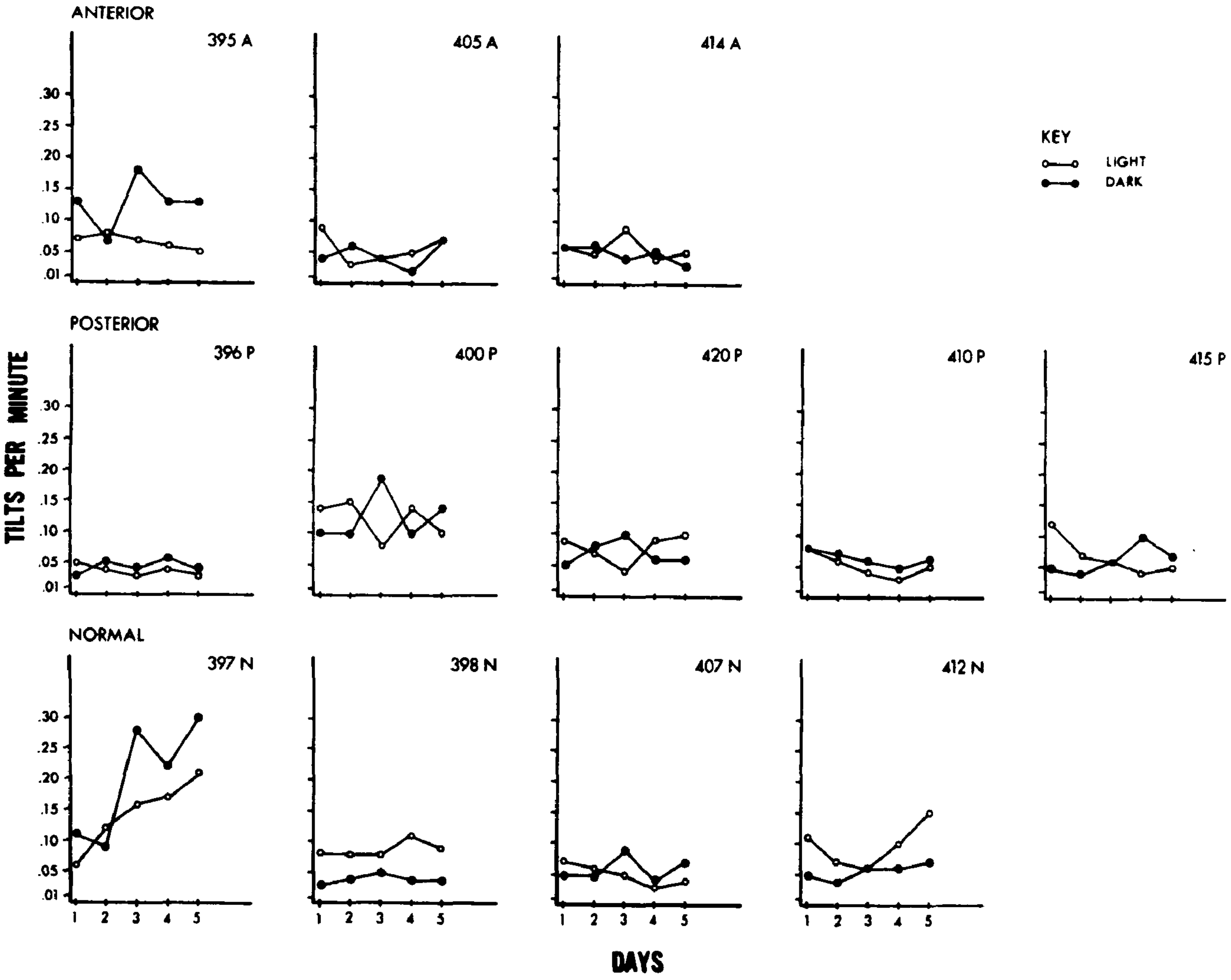
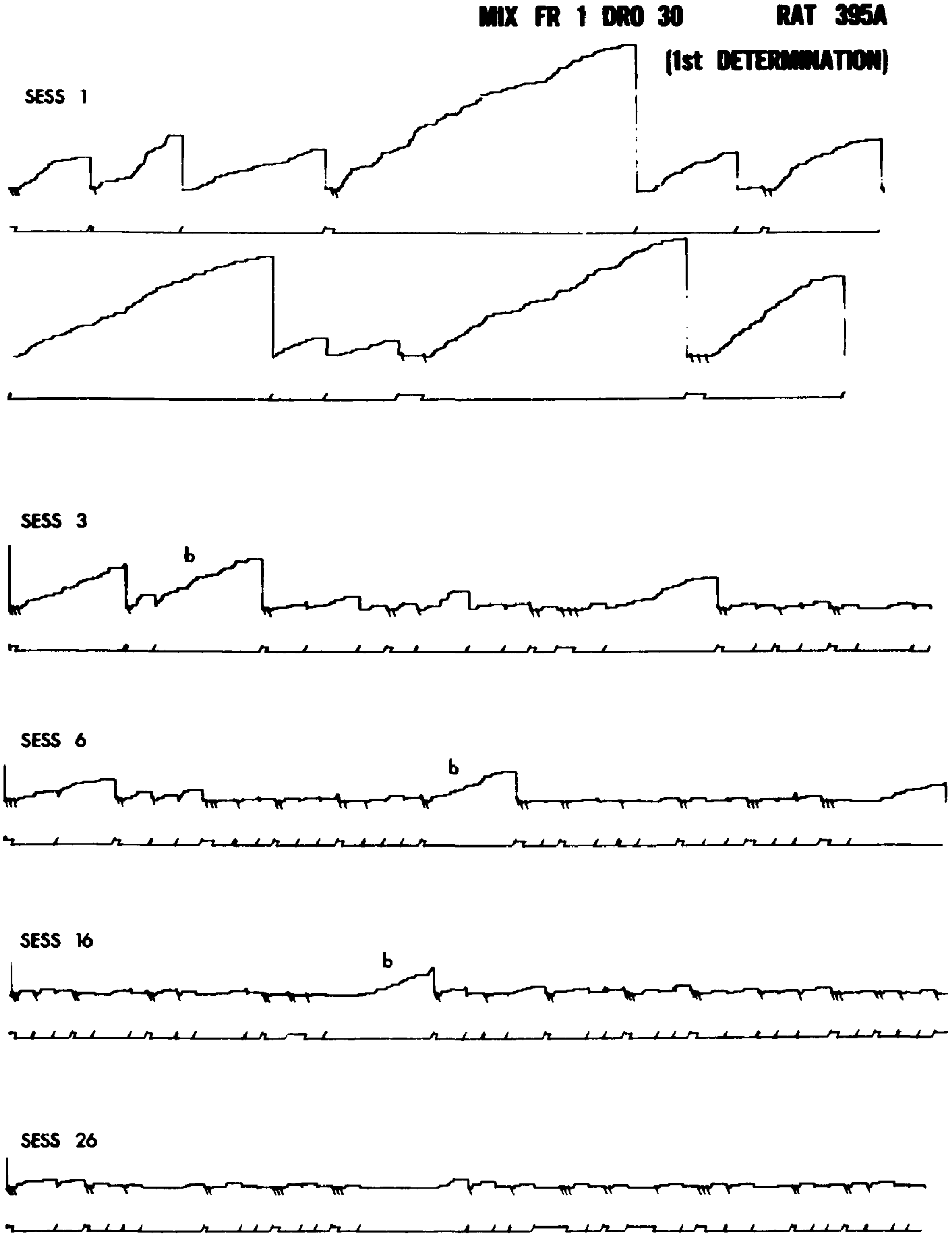

ANTERIOR
395 A
405 A
414 A
416 A
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
MILLILETERS PER KILOGRAM
120
105
90
75
60
45
30
15
3
1 3 5 1 3 5 7
PRE
POST
DAYS

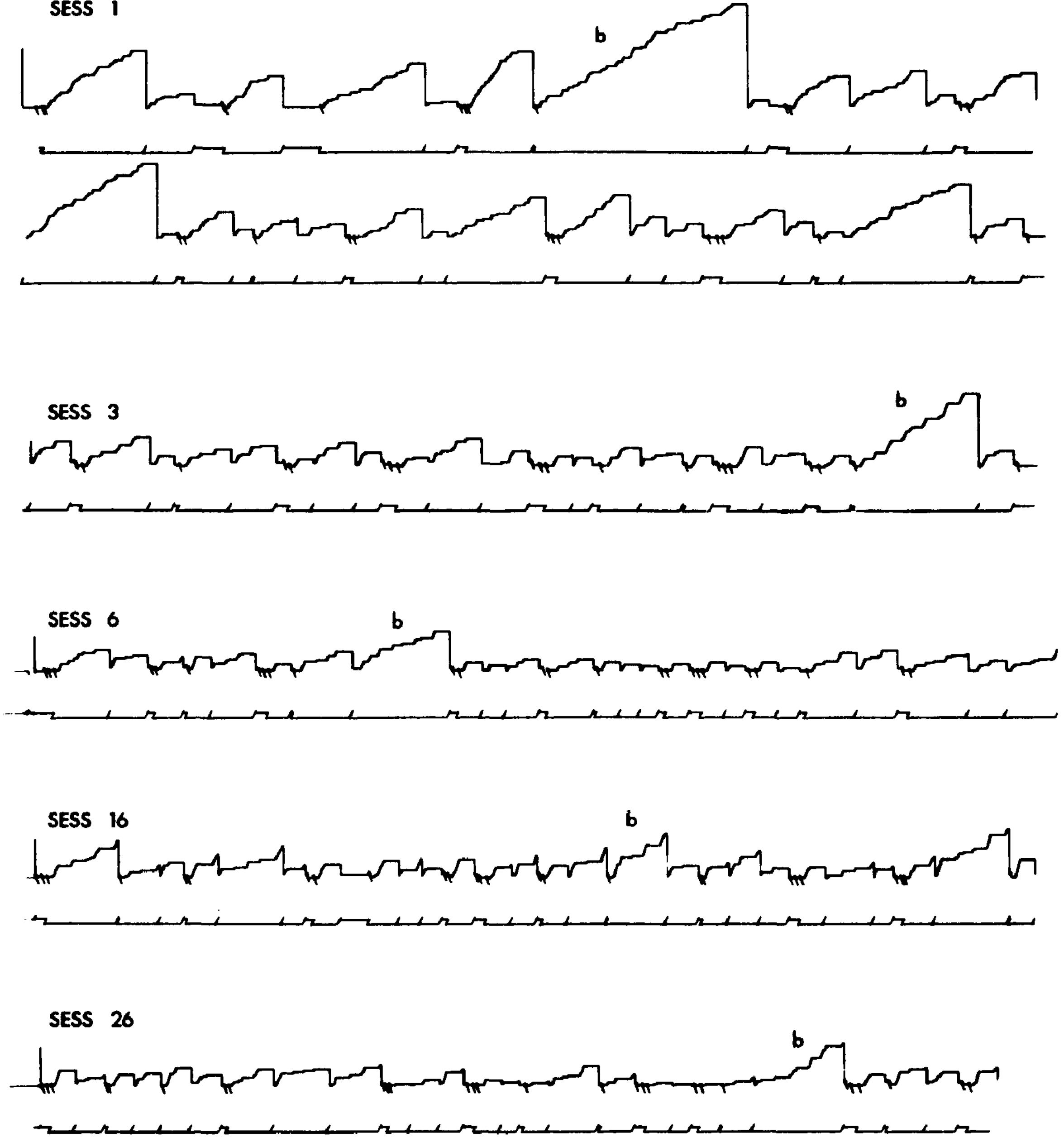
FIG. B. Cage tilts per minute over days in both light and dark conditions for individual rats. Activity measures were taken at end of testing on all Mix FR DRO schedules in Experiment I, approximately 11 months following surgery.

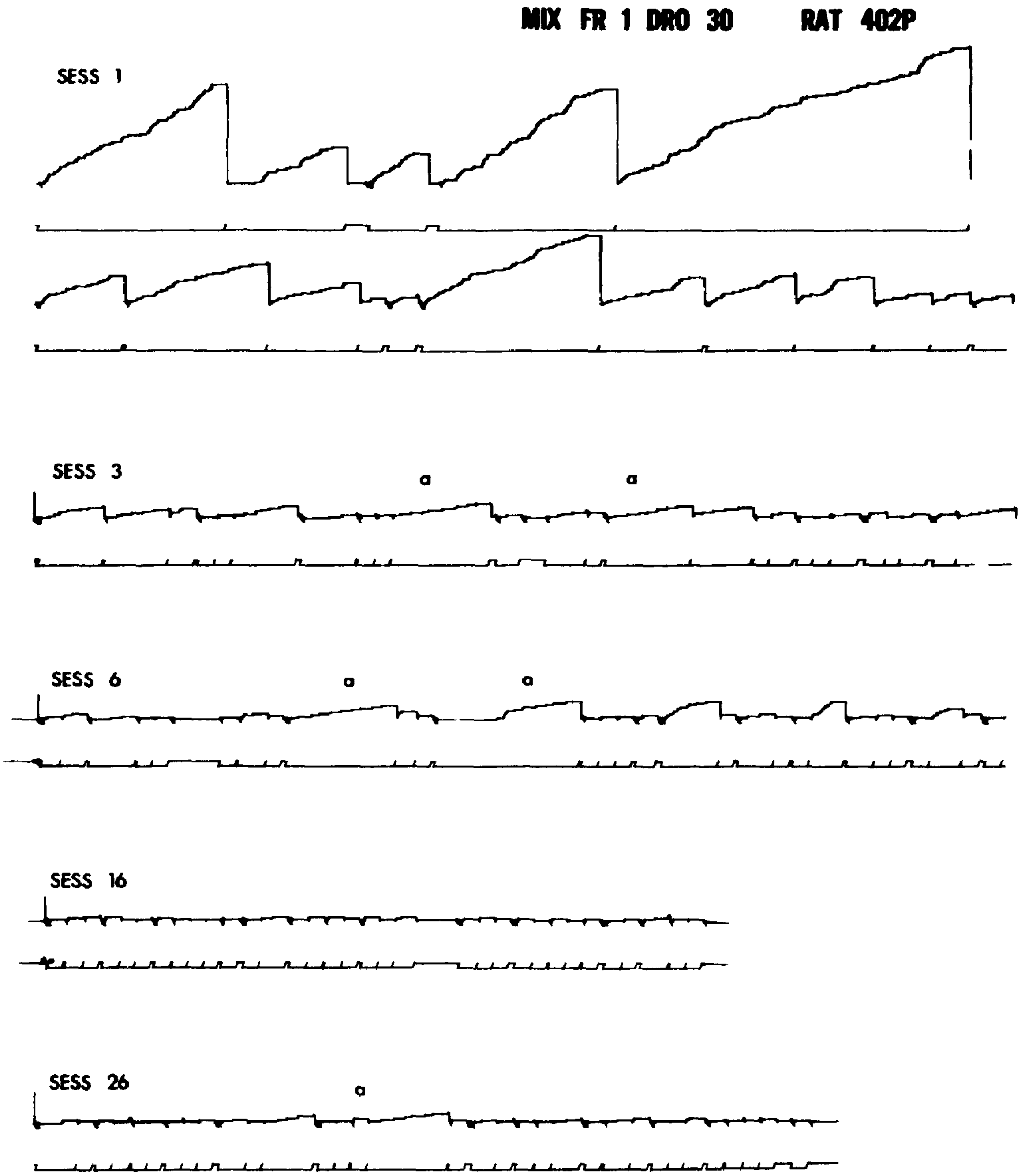

ANTERIOR
395 A
405 A
414 A
POSTERIOR
396 P
400 P
420 P
410 P
415 P
NORMAL
397 N
398 N
407 N
412 N
TILTS PER MINUTE
.30
.25
.20
.15
.10
.05
.01
1
2
3
4
5
DAYS
KEY
LIGHT
DARK

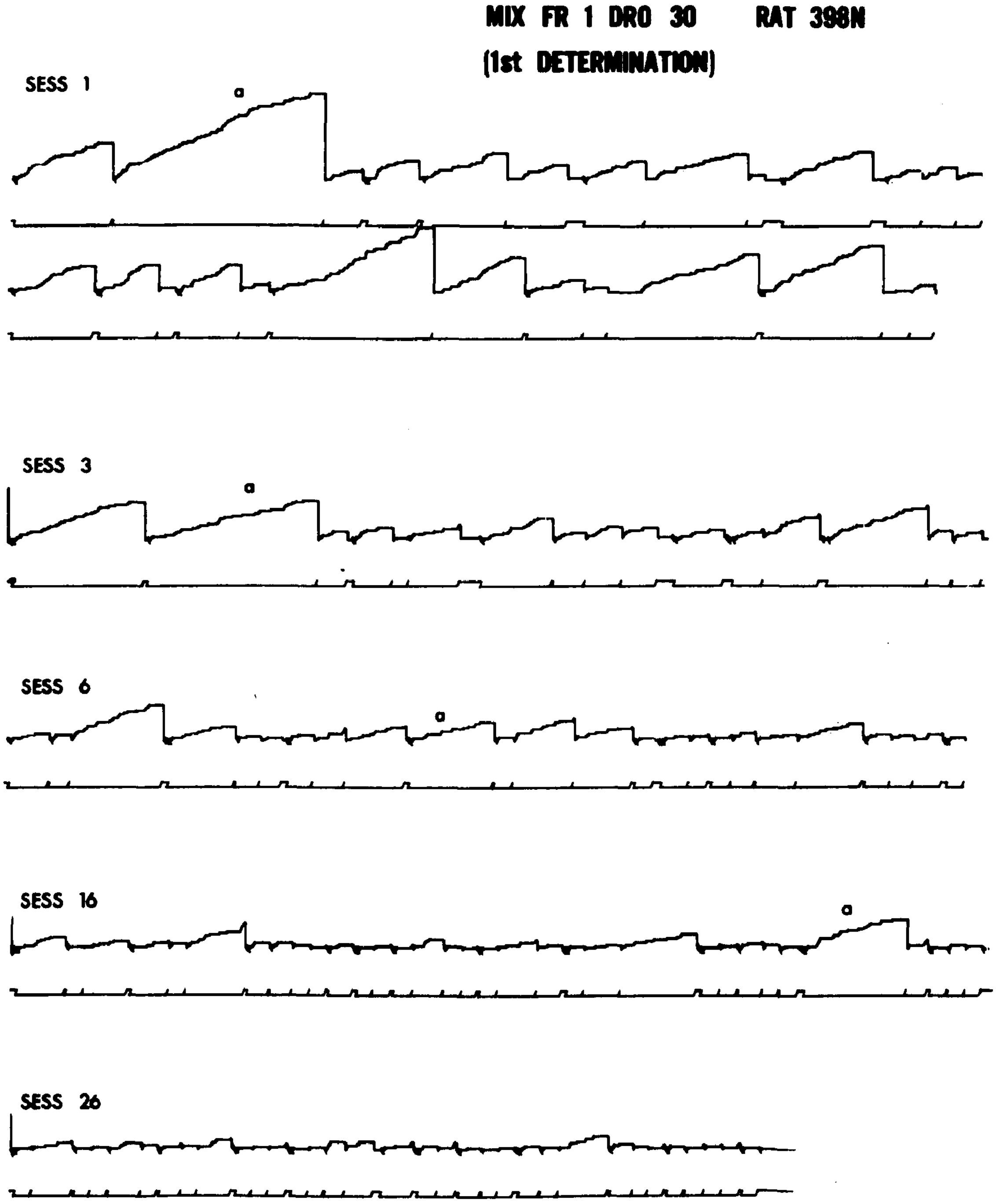
FIGS. C - P. Additional cumulative records of performance from sessions 1, 3, 6, 16, and 26 at FR levels 1 (first determination), 4, and 16, of the Mix FR DRO schedule. Scale is the same as that for cumulative records in text (Figs. 14 - 18)

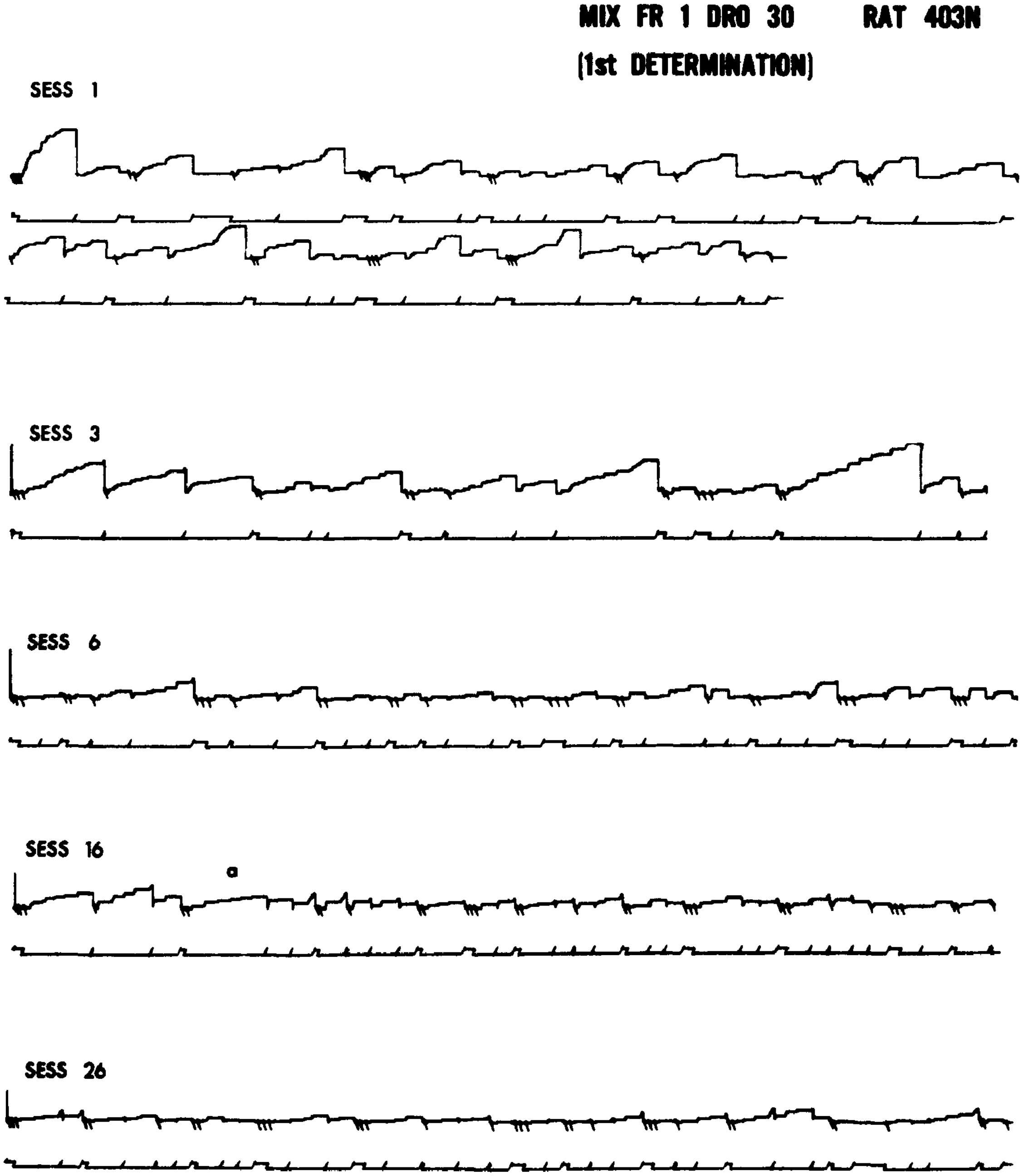

MIX FR 1 DRO 30
RAT 395A
(1st DETERMINATION)
SESS 1
SESS 3
b
SESS 6
b
SESS 16
b
SESS 26

MIX FR 1 DRO 30 RAT 405A
(1st DETERMINATION)

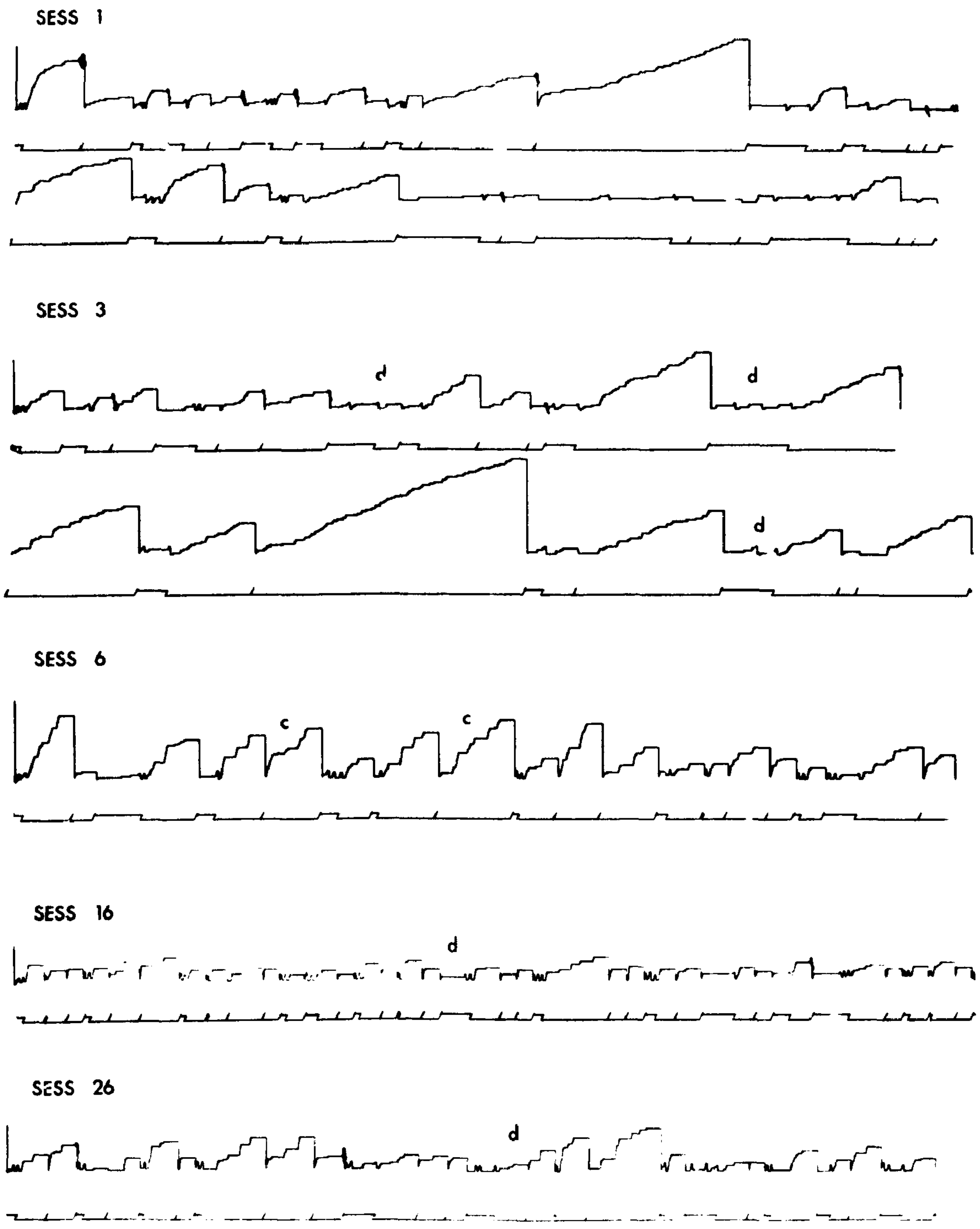


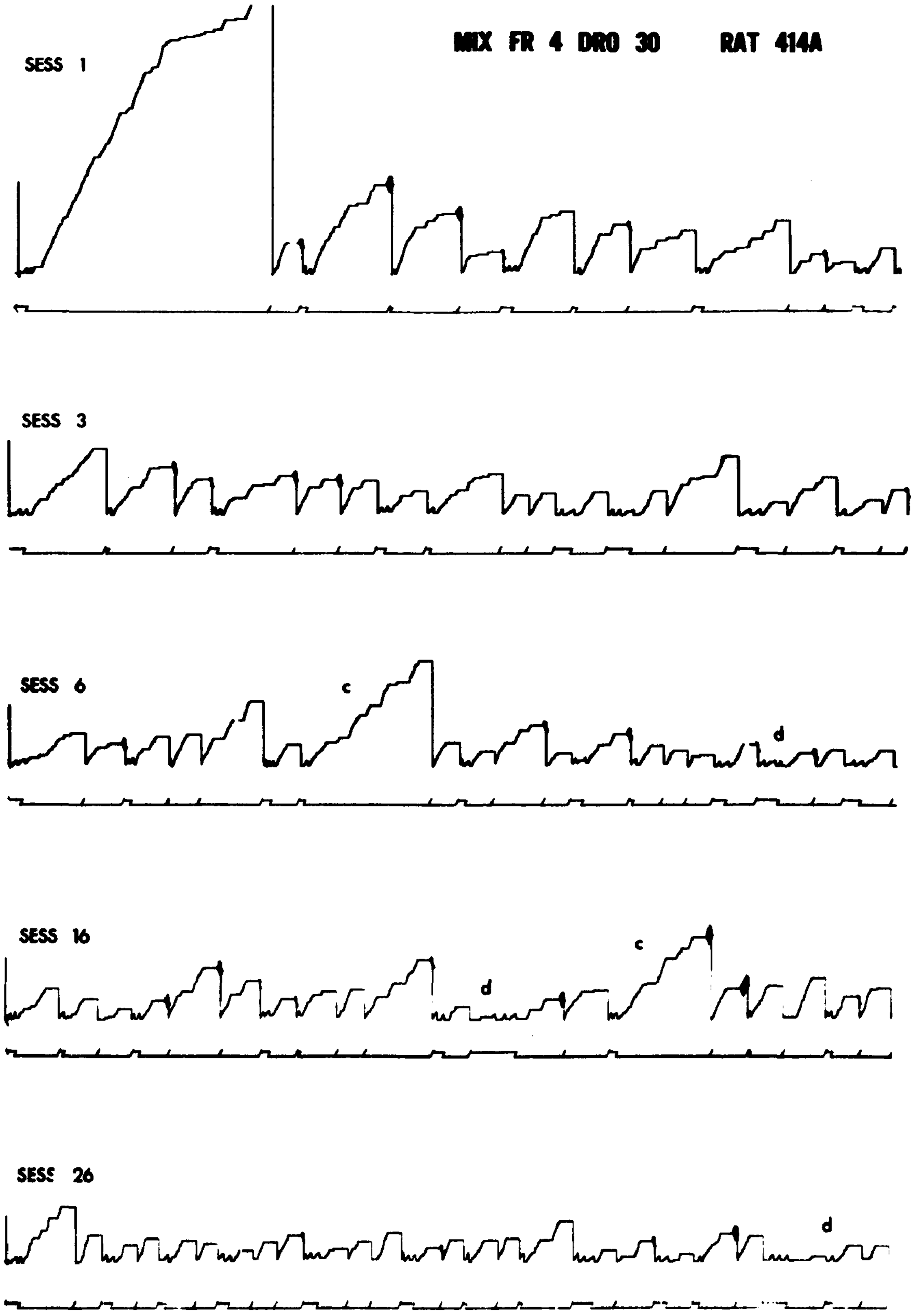

MIX FR 1 DRO 30
RAT 402P
SESS 1
SESS 3
a
a
SESS 6
a
a
SESS 16
SESS 26
a

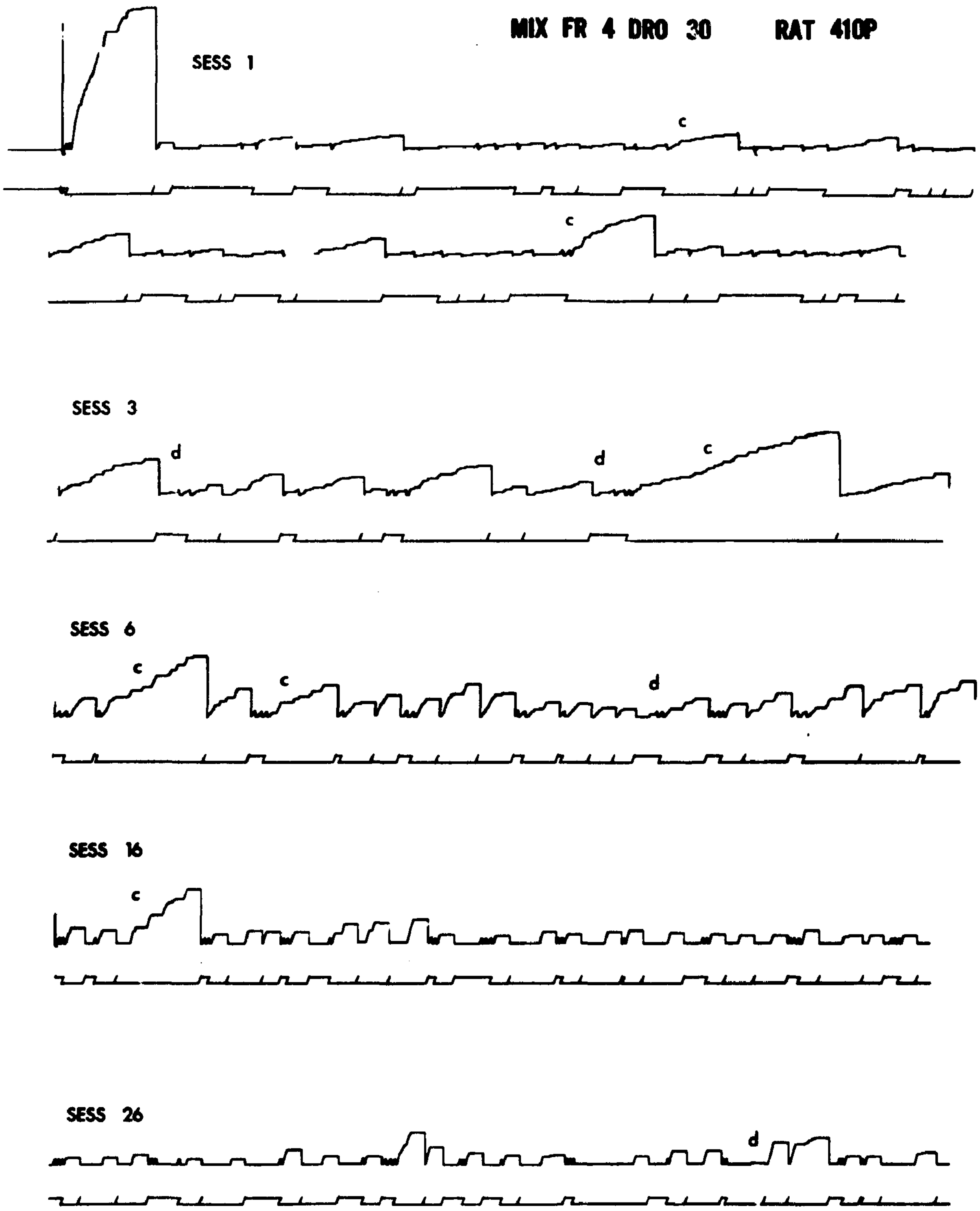

MIX FR 1 DRO 30
RAT 398N
(1st DETERMINATION)
SESS 1
a
SESS 3
a
SESS 6
a
SESS 16
a
SESS 26

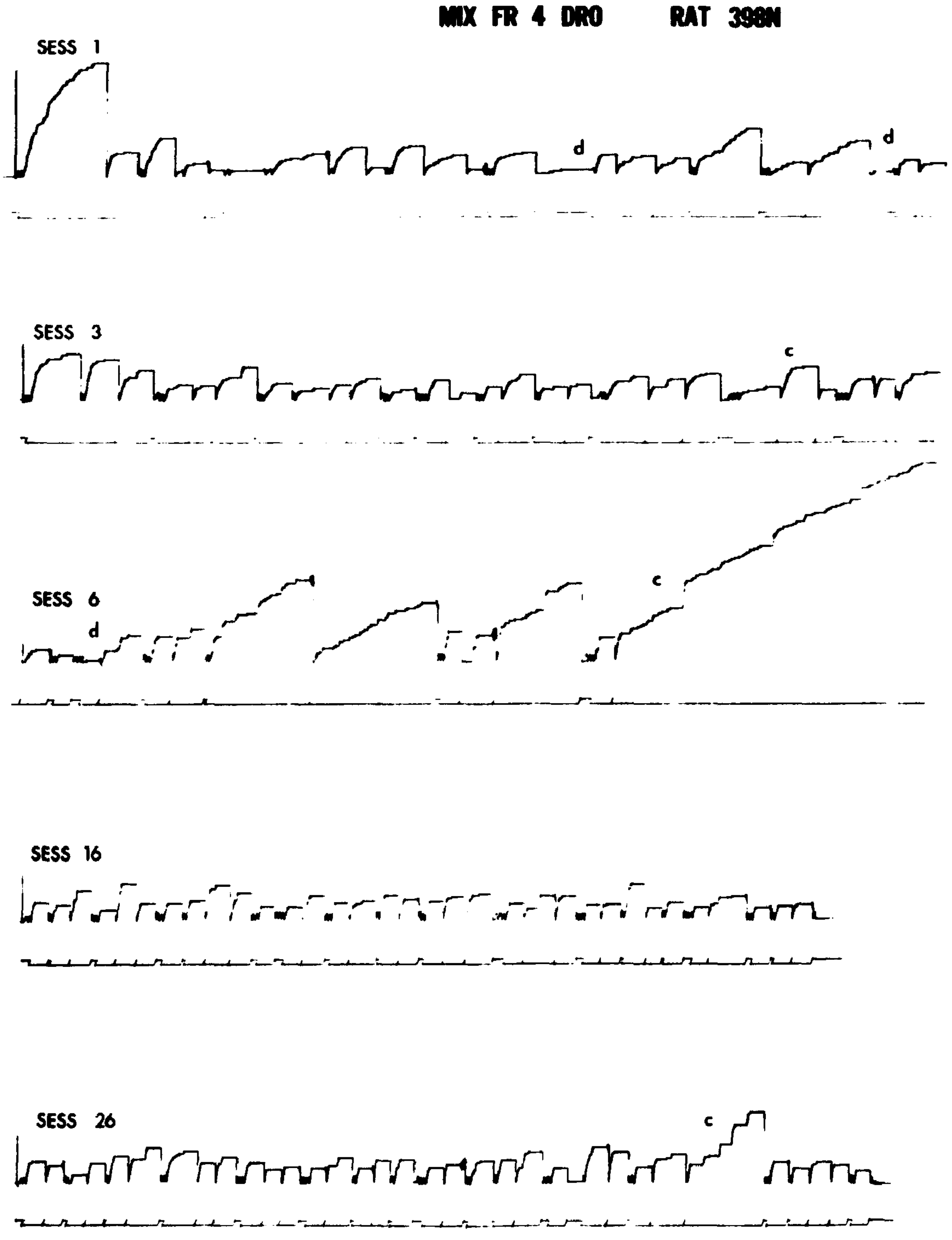

MIX FR 1 DRO 30 RAT 403N
(1st DETERMINATION)
SESS 1
SESS 3
SESS 6
SESS 16
a
SESS 26

MIX FR 4 DRO 30 RAT 395A

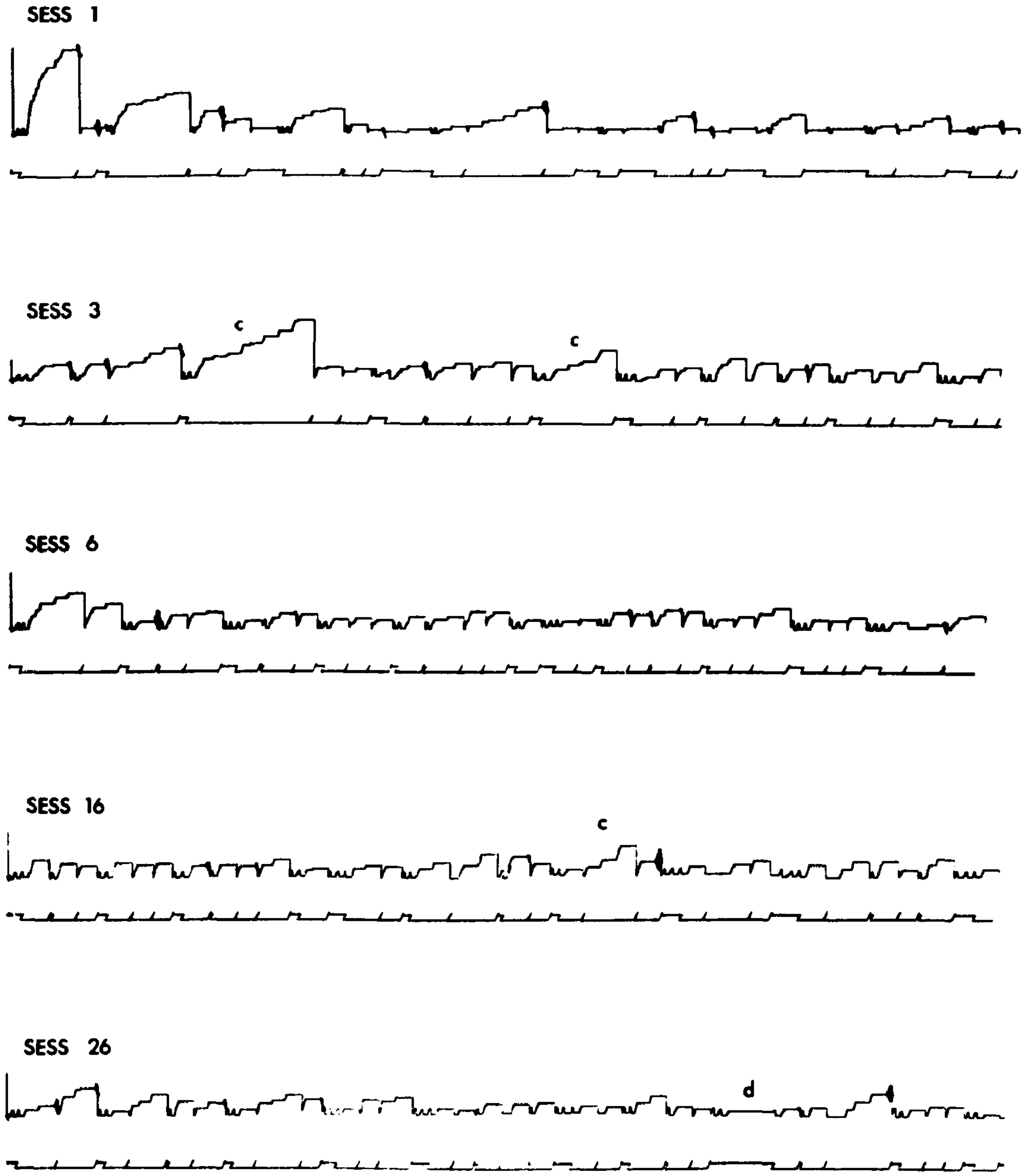



MIX FR 4 DRO 30
RAT 414A
SESS 1
SESS 3
SESS 6
c
d
SESS 16
d
c
SESS 26
d

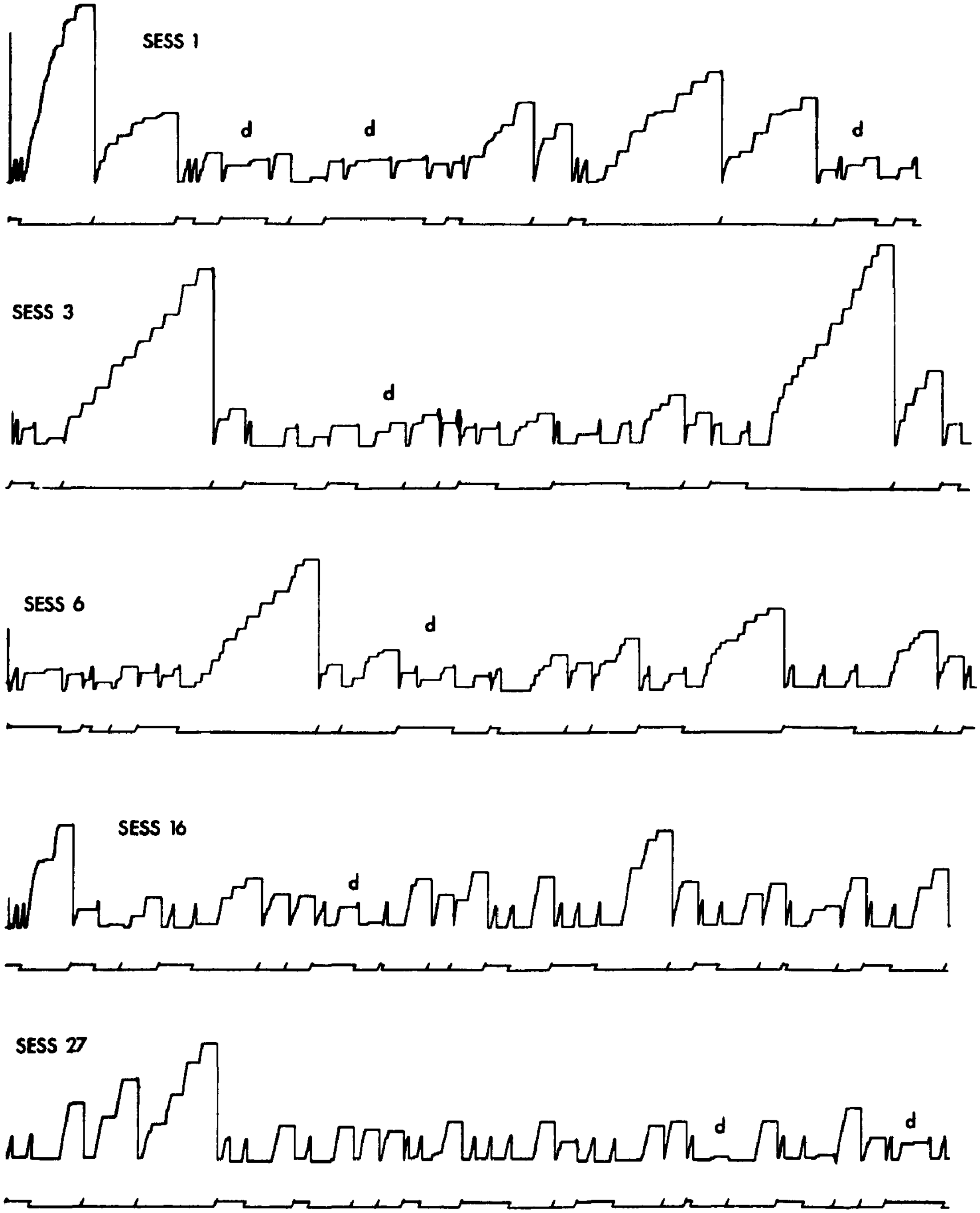

MIX FR 4 DRO 30
RAT 410P
SESS 1
c
c
SESS 3
d
d
c
SESS 6
c
c
d
SESS 16
c
SESS 26
d

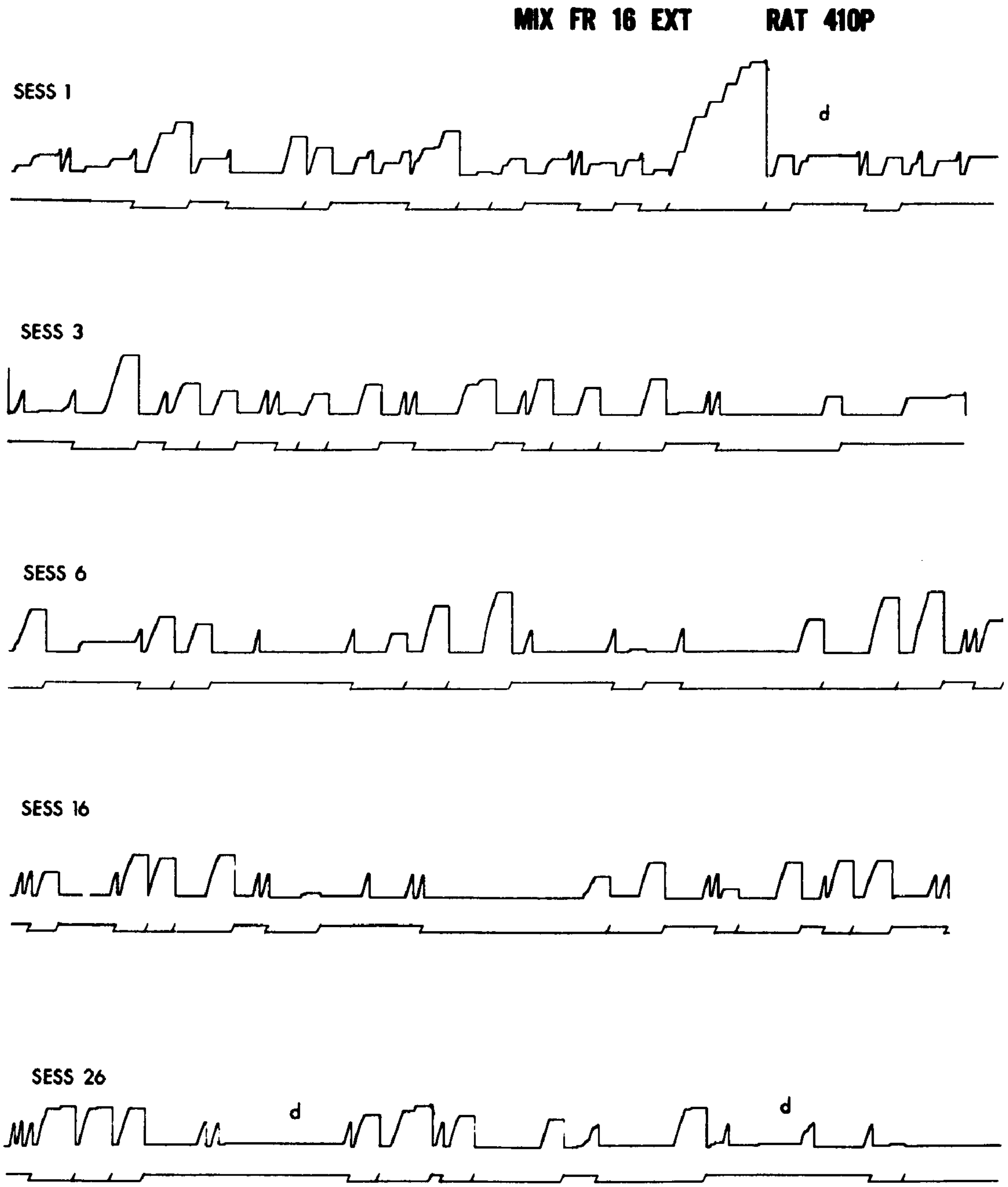

MIX FR 4 DRO
RAT 398N
SESS 1
d
d
SESS 3
c
SESS 6
d
c
SESS 16
SESS 26
c

MIX FR 4 DRO 30 RAT 403N

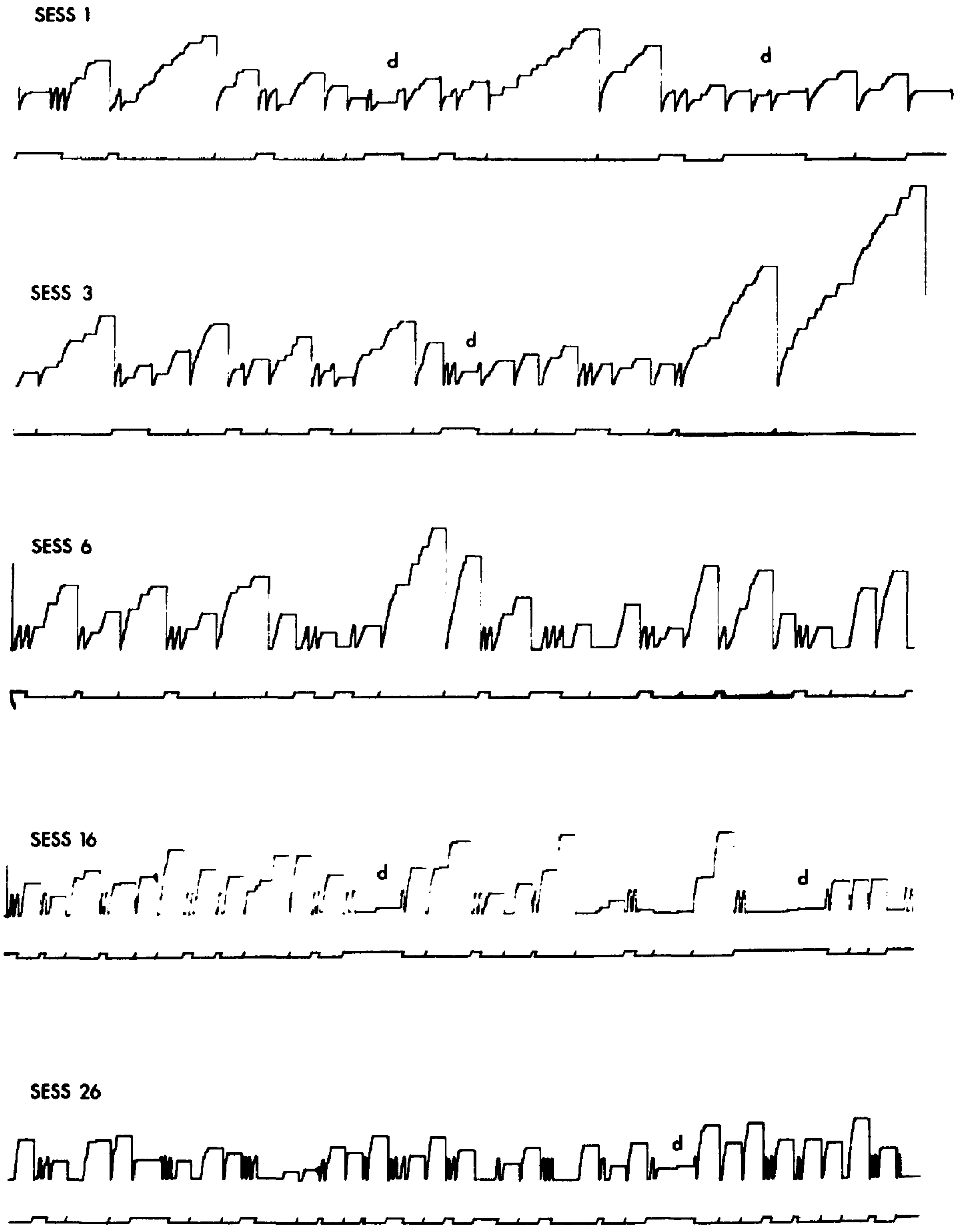


MIX FR 16 DRO 30 RAT 395A

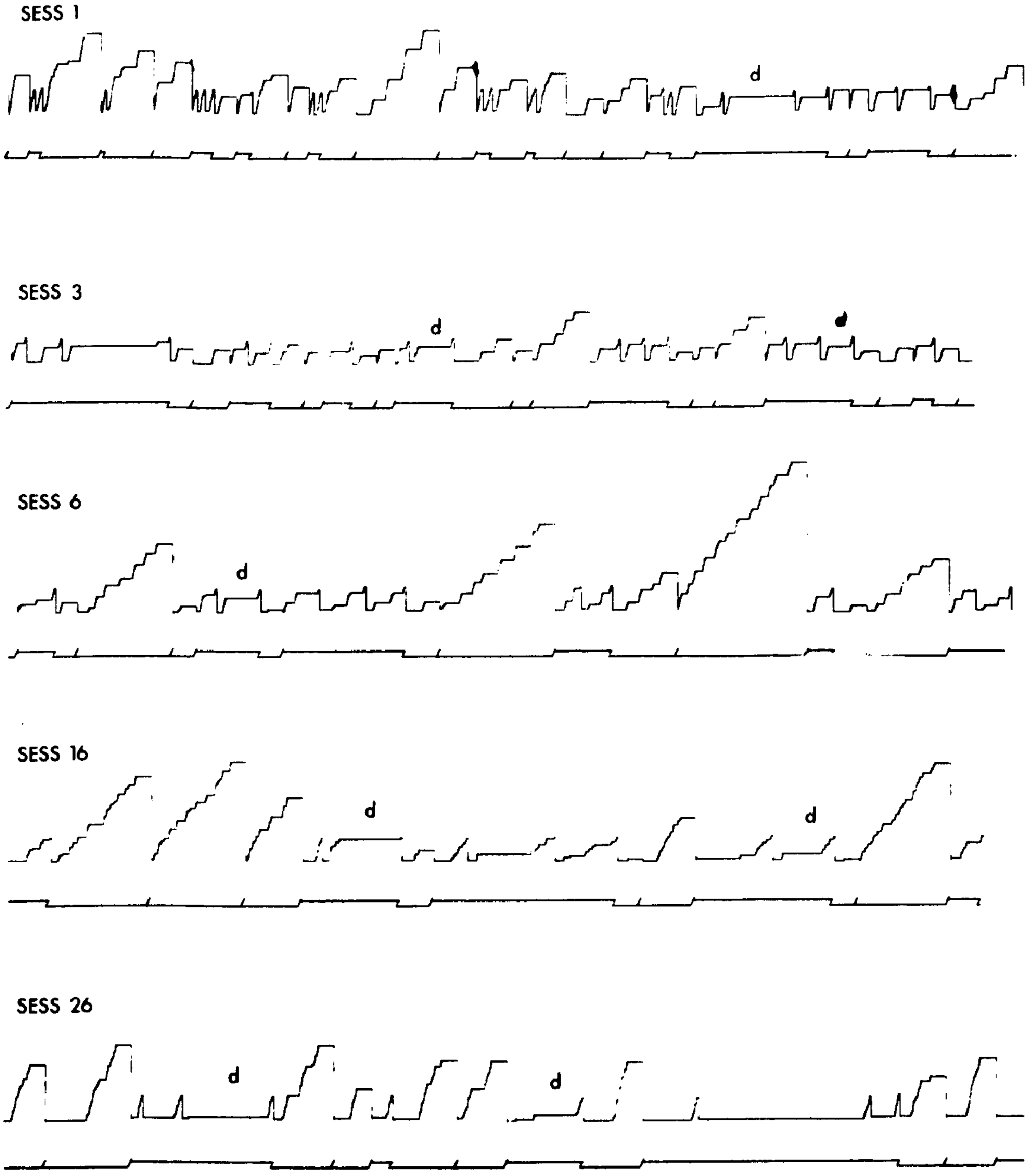



MIX FR 16 EXT
RAT 410P
SESS 1
d
SESS 3
SESS 6
SESS 16
SESS 26
d
d

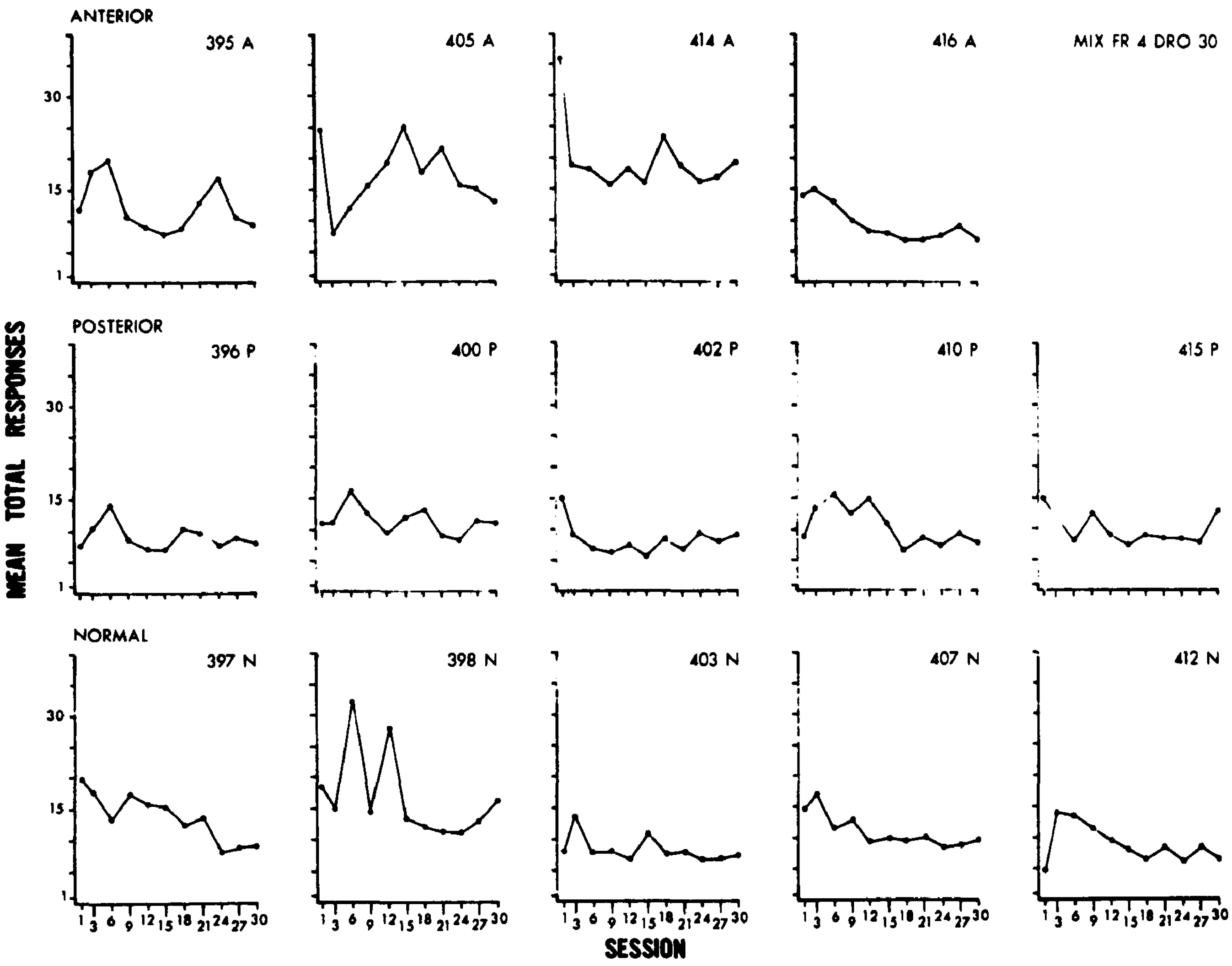
MIX FR 16 DRO 30 RAT 398N

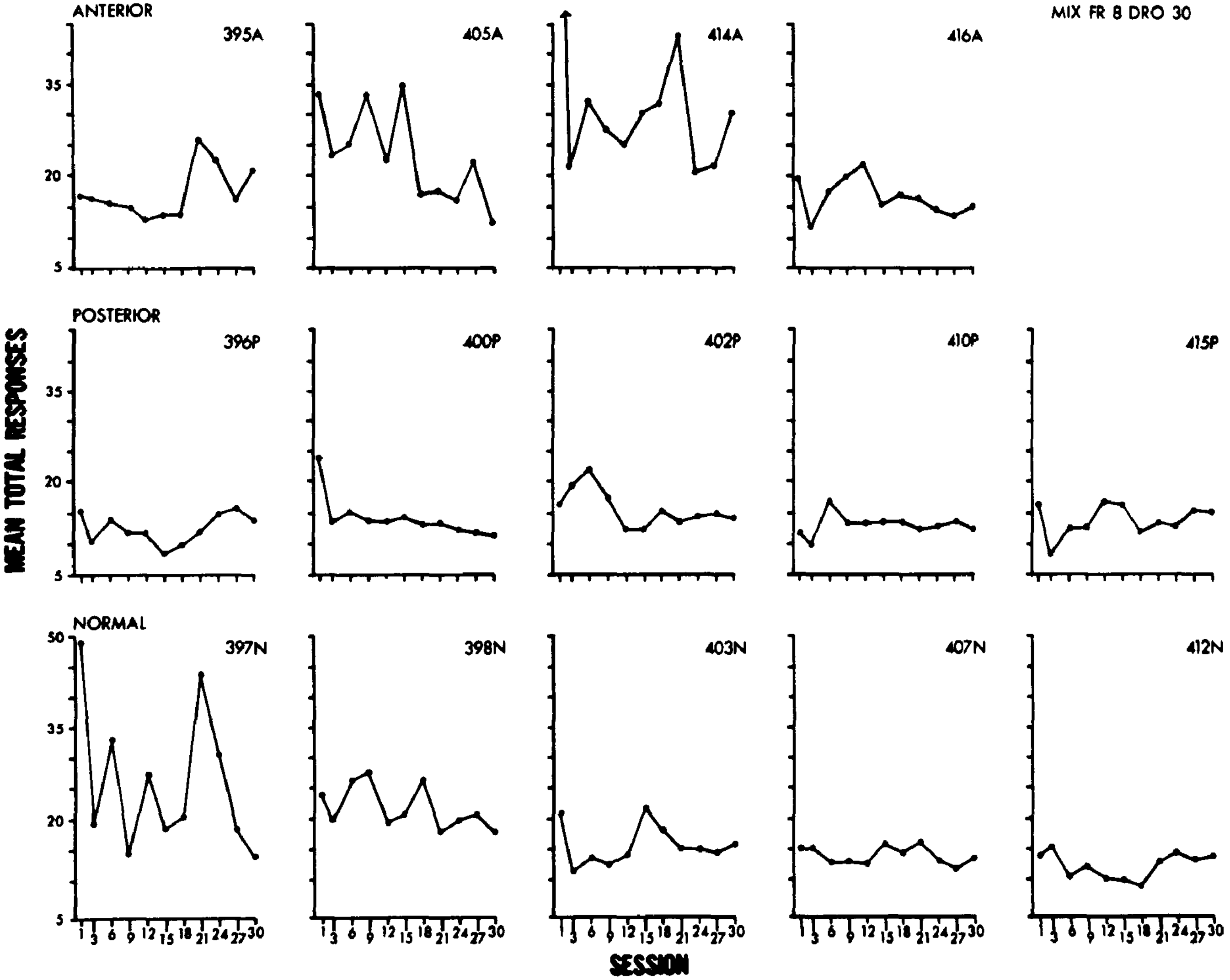


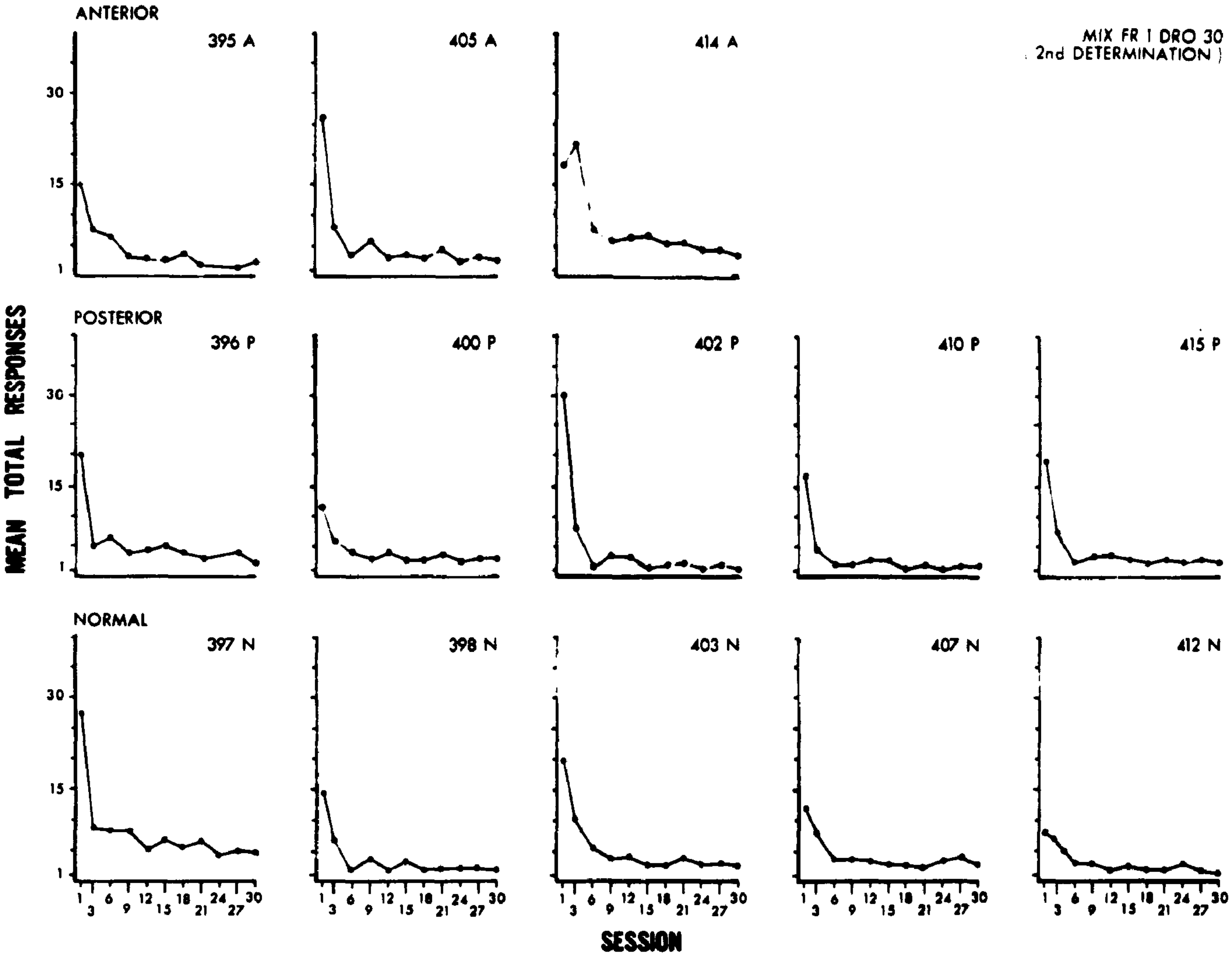
MIX FR 16 DRO 30 RAT 403N



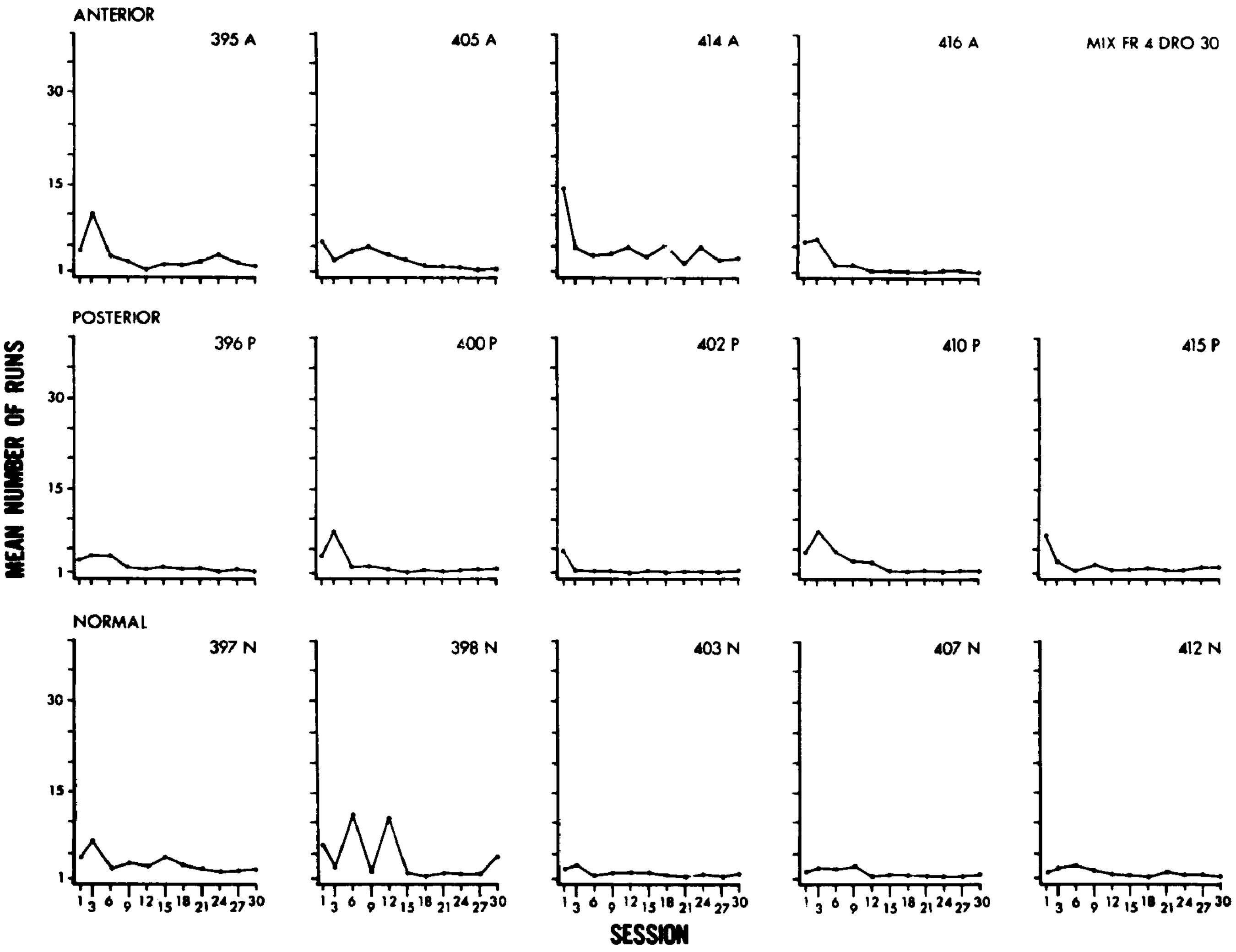
FIGS. Q, R, and S. Mean total responses for every third session for each animal at the FR 4, 8 and second determination of FR 1 level of the Mix FR DRO schedule. Each data point represents the session mean of the total number of responses emitted during each DRO period.

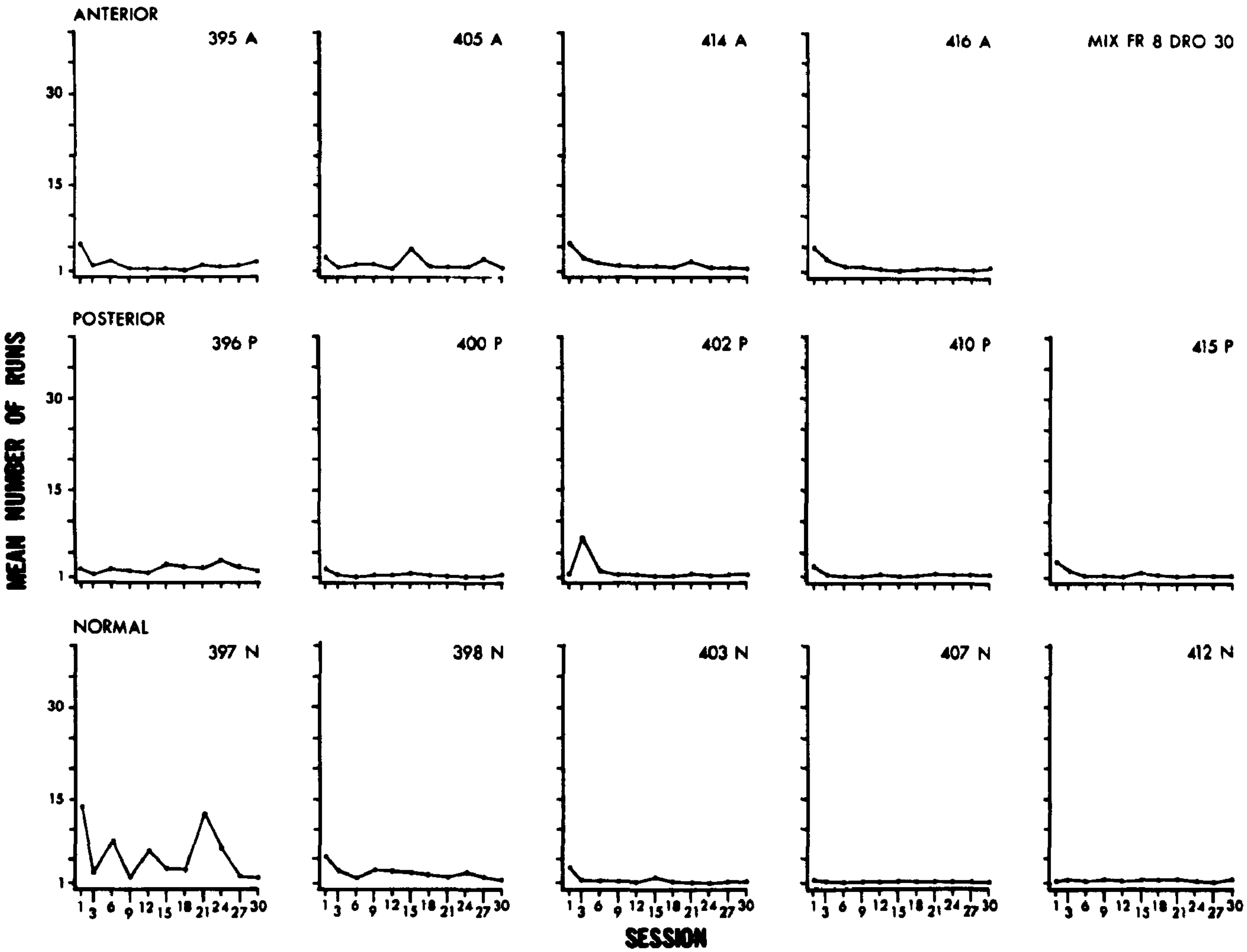

ANTERIOR
395 A
405 A
414 A
416 A
MIX FR 4 DRO 30
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
MEAN TOTAL RESPONSES
1
15
30
1 3 6 9 12 15 18 21 24 27 30
SESSION

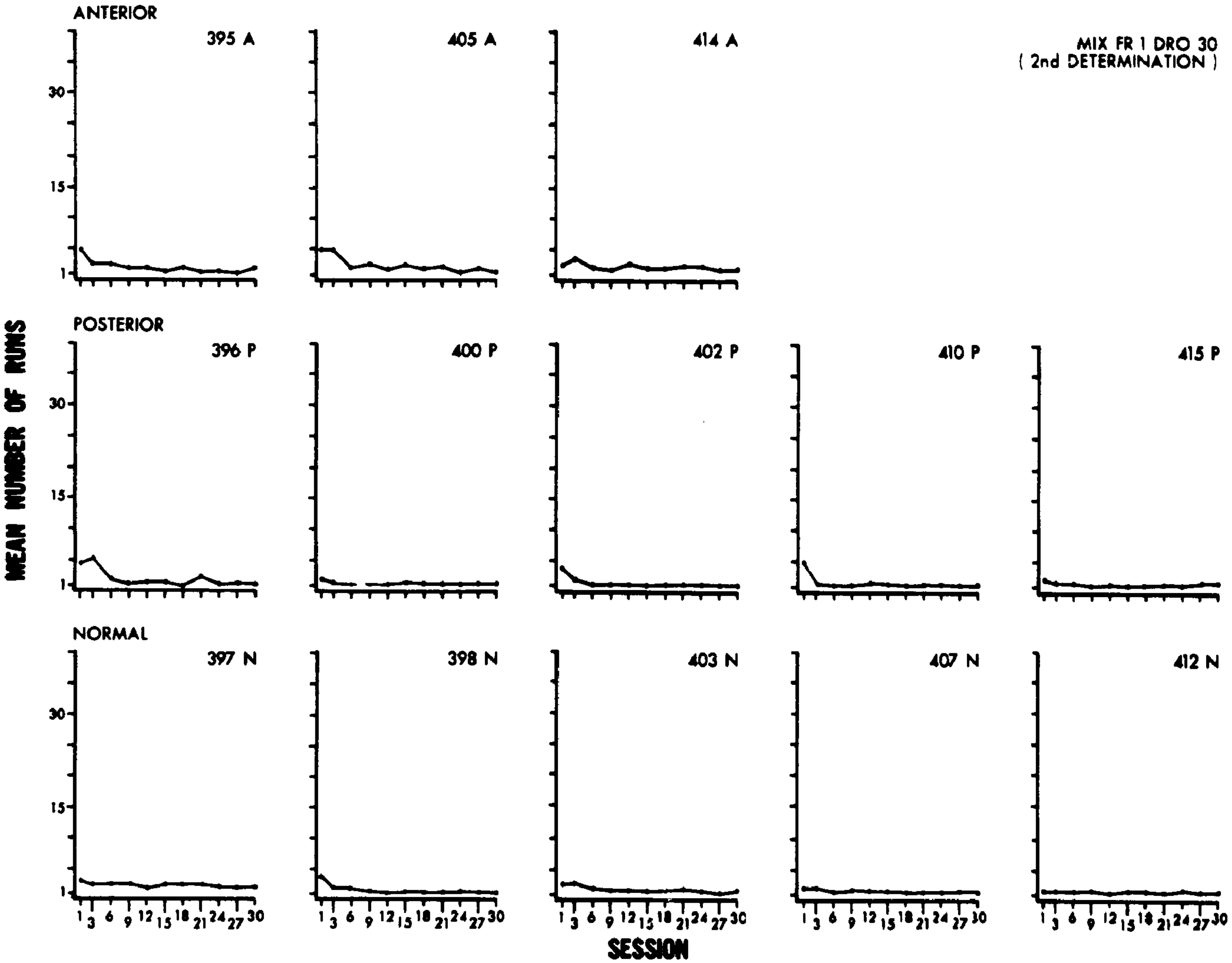

ANTERIOR
MIX FR 8 DRO 30
395A
405A
414A
416A
POSTERIOR
396P
400P
402P
410P
415P
NORMAL
397N
398N
403N
407N
412N
MEAN TOTAL RESPONSES
50
35
20
5
1
3
6
9
12
15
18
21
24
27
30
SESSION


ANTERIOR
395 A
405 A
414 A
MIX FR 1 DRO 30
(2nd DETERMINATION)
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
MEAN TOTAL RESPONSES
30
15
1
1 3 6 9 12 15 18 21 24 27 30
SESSION

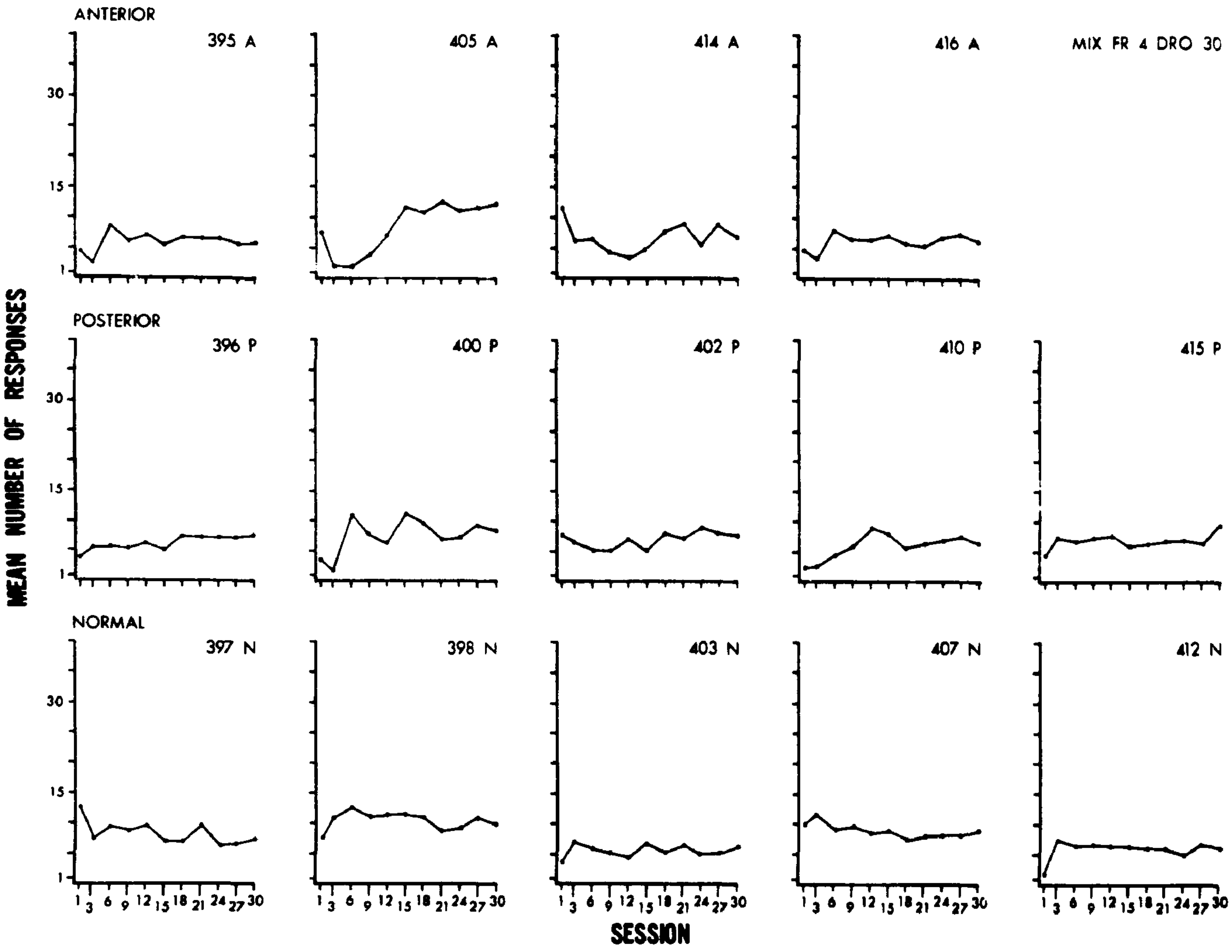
FIGS. T, U, and V. Mean number of runs in initial run in DRO for each animal at the FR 4, 8 and second determination of FR 1 level of the Mix FR DRO schedule. Each data point represents the session mean of the total number of responses emitted during each DRO period.

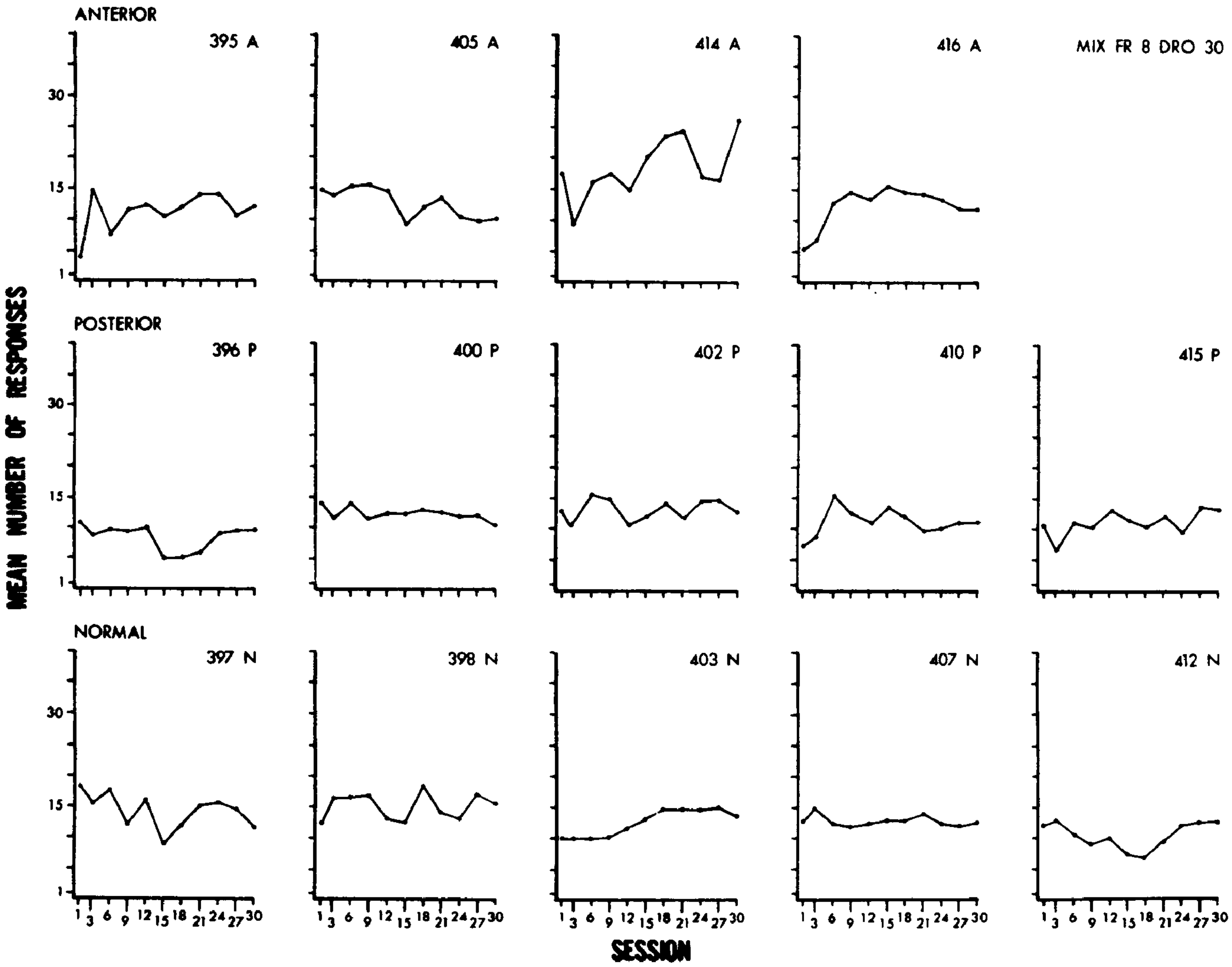

ANTERIOR
395 A
405 A
414 A
416 A
MIX FR 4 DRO 30
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
MEAN NUMBER OF RUNS
30
15
1
1 3 6 9 12 15 18 21 24 27 30
SESSION

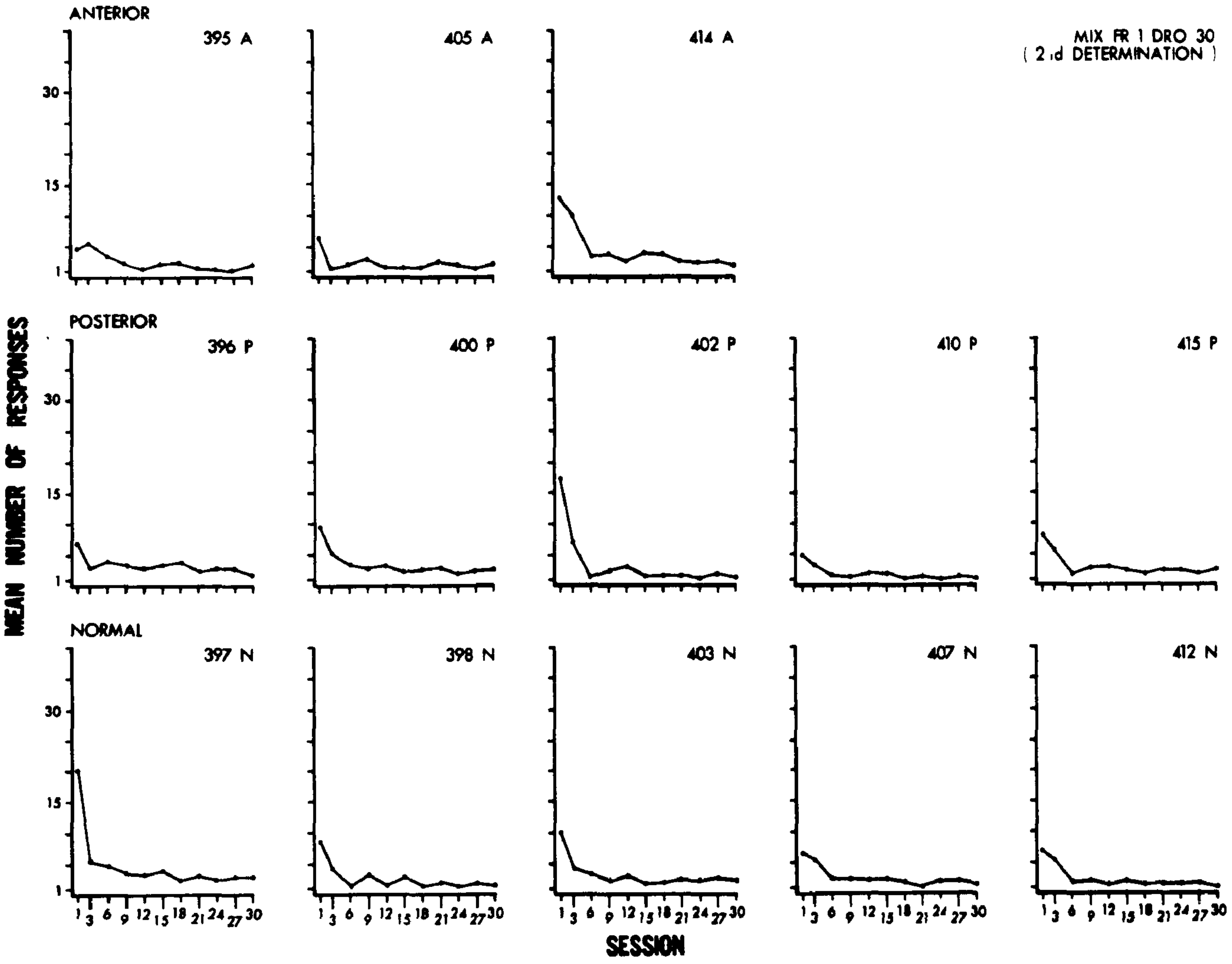

ANTERIOR
395 A
405 A
414 A
416 A
MIX FR 8 DRO 30
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
MEAN NUMBER OF RUNS
30
15
1
1 3 6 9 12 15 18 21 24 27 30
SESSION


MIX FR 1 DRO 30
(2nd DETERMINATION)
ANTERIOR
395 A
405 A
414 A
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
MEAN NUMBER OF RUNS
30
15
1
1 3 6 9 12 15 18 21 24 27 30
SESSION

FIGS. W, X, and Y. Mean number of responses per initial run in DRO for each animal at the FR 4, 8 and second determination of FR 1 level of the Mix FR DRO schedule, Each data point represents the session mean of the total number of responses emitted during each DRO period.


ANTERIOR
395 A
405 A
414 A
416 A
MIX FR 4 DRO 30
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
30
15
1
1 3 6 9 12 15 18 21 24 27 30
MEAN NUMBER OF RESPONSES
SESSION


ANTERIOR
395 A
405 A
414 A
416 A
MIX FR 8 DRO 30
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
MEAN NUMBER OF RESPONSES
30
15
1
1 3 6 9 12 15 18 21 24 27 30
SESSION


ANTERIOR
395 A
405 A
414 A
MIX FR 1 DRO 30
(2nd DETERMINATION)
POSTERIOR
396 P
400 P
402 P
410 P
415 P
NORMAL
397 N
398 N
403 N
407 N
412 N
MEAN NUMBER OF RESPONSES
30
15
1
1 3 6 9 12 15 18 21 24 27 30
SESSION

APPENDIX II

TABLE I

Estimates of Percent Caudate Tissue Destroyed, and Anterior and Posterior Bilateral Extent of Lesions

Rat and Group	Percent Tissue Destroyed+	Extent of Lesion*			
		Left Side		Right Side	
		Anterior	Posterior	Anterior	Posterior
Anterior					
184	21%	4.2	1.0	4.0	1.0
189	16%	4.6	2.0	3.8	1.4
191	16%	4.6	3.0	4.2	1.2
195	17%	4.2	1.8	3.0	0.8
197	14%	4.0	2.2	3.6	1.4
Posterior					
190	11%	2.0	0.8	1.4	0.0
193	11%	0.6	-0.8	1.4	-0.2

*All stereotaxic coordinates shown are relative to bregma; atlas of Pellegrino and Cushman (1967).

+Refers to percent tissue destroyed within that area of caudate containing the lesion; not to percent of total caudate.

TABLE J

Mean Total Responses Per DRO Period for Each Animal Over the Last Five Pre-Operative, First and Last Five Post-Operative Sessions of Experiment II

Rat and Group	Sessions		
	Pre-Operative	Post-Operative	
	11 - 15	1 - 5	11 - 15
Anterior			
184	1.7	7.4	5.1
189	2.3	6.4	4.7
191	8.2	6.5	4.8
195	2.4	5.3	3.6
197	2.2	3.2	2.9
Mean	3.4	5.7	4.2
Posterior			
190	2.0	2.2	1.2
193	2.3	3.7	1.7
Mean	2.2	3.0	1.5
Normal			
186	1.6	1.7	1.2
188	3.0	2.2	2.2
312	1.9	2.2	1.4
313	1.5	1.5	1.3
314	2.4	2.0	1.3
315	2.5	2.9	2.4
318	3.1	2.5	2.2
Mean	2.3	2.1	1.7

TABLE K

Mean Number of Runs Per DRO Period for Each Animal Over the Last Five Pre-Operative, First and Last Five Post-Operative Sessions in Experiment II

Rat and Group	Sessions		
	Pre-Operative	Post-Operative	
	11 - 15	1 - 5	11 - 15
Anterior			
184	1.3	4.4	2.7
189	1.6	5.1	3.0
191	4.0	4.0	2.7
195	1.8	4.5	2.2
197	2.3	2.7	1.7
Mean	2.2	4.1	2.5
Posterior			
190	1.2	1.4	1.1
193	1.4	2.0	1.4
Mean	1.3	1.7	1.3
Normal			
186	1.3	1.4	1.3
188	2.6	1.9	1.6
312	1.6	1.6	1.1
313	1.2	1.4	1.2
314	2.1	1.7	1.3
315	1.6	1.7	1.3
318	2.3	2.0	1.6
Mean	1.8	1.7	1.3

TABLE L

Mean Number of Responses Per Initial Run in DRO Periods for Each Animal Over the Last Five Pre-Operative, First and Last Five Post-Operative Sessions in Experiment II

	Sessions		
	Pre-Operative	Post-Operative	
Rat and Group	11 - 15	1 - 5	11 - 15
Anterior			
184	1.4	1.7	1.8
189	1.5	1.6	2.0
191	3.3	2.1	2.2
195	1.5	1.5	1.7
197	1.0	1.3	1.8
Mean	1.7	1.6	1.9
Posterior			
190	1.7	1.5	1.2
193	1.6	2.0	1.2
Mean	1.7	1.8	1.2
Normal			
186	1.3	1.3	1.1
188	1.2	1.2	1.3
312	1.2	1.5	1.3
313	1.2	1.0	1.1
314	1.1	1.0	1.0
315	1.7	2.0	2.0
318	1.6	1.3	1.5
Mean	1.3	1.3	1.3

TABLE M

Median Latencies of First Response in Reinforcement Periods Following Reinforcement and Following DRO for Each Animal Over the Last Five Pre-Operative, First and Last Five Post-Operative Sessions. Numerals Represent Lower Limit of Class Interval in Seconds

Rat and Group	Sessions					
	Pre-Operative		Post-Operative			
	1 - 5		1 - 5		11 - 15	
	Post FR	Post DRO	Post FR	Post DRO	Post FR	Post DRO
Anterior						
184	4.0	2.0	12.0	3.0	7.0	3.0
189	1.0	0.0	6.0	8.0	4.0	2.0
191	4.5	0.0	4.0	1.0	5.0	1.0
195	1.0	1.0	3.0	3.0	4.0	1.5
197	1.0	0.0	3.0	2.0	2.0	1.0
Mean	2.3	0.6	4.0	3.0	4.2	2.0
Posterior						
190	4.0	0.0	3.0	0.0	2.0	0.0
193	6.0	3.0	4.0	2.0	5.0	1.0
Mean	5.0	1.5	3.5	1.0	3.5	0.5
Normal						
186	1.0	1.0	1.0	1.0	1.0	0.5
188	1.0	1.0	0.0	1.0	1.0	0.0
312	1.0	1.0	1.0	0.0	1.0	0.0
313	4.0	1.0	2.0	1.0	4.0	1.0
314	2.0	0.0	0.0	0.0	0.5	0.0
315	2.0	0.0	2.0	0.0	1.0	0.0
318	1.0	0.0	1.0	0.0	1.0	0.0
Mean	1.7	0.7	1.0	0.4	1.3	0.2

REFERENCES

Albert, M., & Bignami, G. Effects of frontal, median cortical and caudate lesions on two-avoidance learning by rats. *Physiology and Behavior*, 1968, *3*, 141-147.

Allen, J. D., Mitcham, J. C., & Byrd, J. I. Effects of caudate lesions on the acquisition and retention of Sidman avoidance in the rat. *Psychonomic Science*, 1972, *27*, 157-160.

Barofsky, I., & Hurwitz, D. Within ratio responding during fixed ratio performance. *Psychonomic Science*, 1968, *11*, 263-264.

Battig, K., Rosvold, H. E., & Mishkin, M. Comparison of the effects of frontal and caudate lesions on delayed response and alternation in monkeys. *Journal of Comparative and Physiological Psychology*, 1960, *53*, 400-404.

Battig, K., Rosvold, H. E., & Mishkin, M. Comparison of the effects of frontal and caudate lesions on discrimination learning in monkeys. *Journal of Comparative and Physiological Psychology*, 1962, *55*, 458-463.

Berryman, R., Wagman, W., & Keller, F. S. Chlorpromazine and the discrimination of response produced cues. In L. Uhr & J. G. Miller (Eds.), *Drugs and Behavior*. New York: Wiley, 1960.

Boren, J. J. Response rate and resistance to extinction as functions of the fixed ratio. *Journal of Experimental Psychology*, 1961, *16*, 304-308.

Borst, A., Delacour, J., & Licouban, S. Effets, chez le rat, de lesions du noyau caude sur le conditionnement de response alternee. *Neuropsychologia*, 1970, *8*, 89-101.

Bossom, J. The effect of brain lesions on prism-adaptation in monkeys. *Psychonomic Science*, 1965, *2*, 45-46.

Bossom, J. The effect of lesions in caudate nucleus and superior colliculus upon adaptation to prisms in monkey. *Psychonomic Science*, 1972, *26*, 255-260.

Bowen, F. P. Visuomotor deficits produced by cryogenic lesions of the caudate. *Neuropsychologia*, 1969, *7*, 59-65.

Buchwald, N. A., Wyers, E. J., Carlin, J., & Farley, R. E. Effects of caudate stimulation on visual discrimination. *Experimental Neurology*, 1961, *4*, 23-36.

Buchwald, N. A., Wyers, E. J., Okuma, T., & Heuser, G. The "caudate-spindle." I: Electrophysiological properties. *Electroencephalography and Clinical Neurophysiology*, 1961, *13*, 509-518.

Buchwald, N. A., Heuser, G., Wyers, E. J., & Lauprecht, C. W. The "caudate-spindle." III: Inhibition by high frequency stimulation of subcortical structures. *Electroencephalography and Clinical Neurophysiology*, 1961, *13*, 525-530.

Buchwald, N. A., Wyers, E. J., Lauprecht, C. W., & Heuser, G. The "caudate-spindle." IV: A behavioral index of caudate-induced inhibition. *Electroencephalography and Clinical Neurophysiology*, 1961, *13*, 531-537.

Bullock, D. H., & Smith, W. C. An effect of repeated conditioning-extinction upon operant strength. *Journal of Experimental Psychology*, 1953, *46*, 349-352.

Butters, N., & Rosvold, H. E. Effect of caudate and septal nuclei lesions on resistance to extinction and delayed-alternation. *Journal of Comparative and Physiological Psychology*, 1968, *65*, 397-403.

Campbell, B. A., & Lynch, G. S. Cortical modulation of spontaneous activity during hunger and thirst. *Journal of Comparative and Physiological Psychology*, 1969, *67*, 15-22.

Chorover, S. L., & Gross, C. G. Caudate nucleus lesions. Behavioral effects in the rat. *Science*, 1963, *141*, 826-827.

Davis, G. D. Caudate lesions and spontaneous locomotion in the monkey. *Neurology*, 1958, *8*, 135-139.

Deadwyler, S. A., & Wyers, E. J. Effects of interpolated caudate and septal stimulation on DRL performance in rats. *Proceedings*, 78th Annual Convention, American Psychological Association, 1970, 241-242.

Dean, W. H., & Davis, G. D. Behavior changes following caudate lesions in rhesus monkeys. *Journal of Neurophysiology*, 1959, *22*, 524-537.

Denny-Brown, D. *The Basal Ganglia and their Relation to Disorders of Movement*. London: Oxford University, 1962.

Dews, P. B. Analysis of effects of psychopharmacological agents in behavioral terms. *Federation Proceedings*, 1958, *17*, 1024-1030.

Divac, I. Effects of prefrontal and caudate lesions on delayed response in cats. *Acta Biologiae Experimentalis*, 1968a, *28*, 149-167.

Divac, I. Functions of the caudate nucleus. *Acta Biologiae Experimentalis*, 1968b, *28*, 107-120.

Divac I. Frontal lobe system and spatial reversal in the rat. *Neuropsychologia*, 1971, *9*, 175-184.

Divac I., Rosvold, H. E., & Szwarcbart, M. K. Behavioral effects of selective ablations of the caudate nucleus. *Journal of Comparative and Physiological Psychology*, 1967, *63*, 184-190.

Ervin, F. R., & Anders, T. R. Normal and pathological memory: Data and a conceptual scheme. In F. O. Schmitt (Ed.), *The Neurosciences, Second Study Program*. New York: Rockefeller University, 1970.

Felton, M., & Lyon, D. O. The post-reinforcement pause. *Journal of the Experimental Analysis of Behavior*, 1966, *9*, 131-134.

Ferster, C. B., & Skinner, B. F. *Schedules of Reinforcement*. New York: Appleton-Century-Crofts, 1957.

Fitzsimons, J. T. Thirst. *Physiological Review*, 1972, *52*, 468-561.

Fox, S. S., Kimble, D. P., & Lickey, M. E. A role for the caudate nucleus in motor response inhibition. Mental Health Research Institute, The University of Michigan, Preprint 116, August, 1963.

Fox, S. S., Kimble, D. P., & Lickey, M. E. Comparison of caudate nucleus and septal-area lesions on two types of avoidance behavior. *Journal of Comparative and Physiological Psychology*, 1964, *58*, 380-386.

French, G. M. The frontal lobes and association. In J. M. Warren, & K. Akert (Eds.), *The Frontal Granular Cortex and Behavior*. New York: McGraw-Hill, 1964.

Gellerman, L. W. Chance orders of alternating stimuli in visual discrimination experiments. *Journal of Genetic Psychology*, 1933, *42*, 207-208.

Green, R. H., Beatty, W. W., & Schwartzbaum, J. S. Comparative effects of septo-hippocampal and caudate lesions on avoidance behavior in rats. *Journal of Comparative and Physiological Psychology*, 1967, *64*, 449-452.

Gregory, R. L. The brain as an engineering problem. In W. H. Thorpe, & D. L. Zangwill (Eds.), *Current Problems in Animal Behavior*. Cambridge, England: Cambridge University, 1961.

Gross, C. G., Chorover, S. L., & Cohen, S. M. Caudate, cortical, hippocampal and dorsal thalamic lesions in rats: Alternation and Hebb-Williams maze performance. *Neuropsychologia*, 1965, *3*, 53-68.

Grossman, S. P. A Textbook of Physiological Psychology. New York: Wiley, 1967.

Hansing, R. A., Schwartzbaum, J. S., & Thompson, J. B. Operant behavior following unilateral and bilateral caudate lesions in the rat. Journal of Comparative and Physiological Psychology, 1968, 66, 378-388.

Hearst, E. Some persistent problems in the analysis of conditioned inhibition. In R. A. Boakes, & M. S. Halliday (Eds.), Inhibition and Learning. New York: Academic Press, 1972.

Hearst, E., Besley, S., & Farthing, G. W. Inhibition and the stimulus control of operant behavior. Journal of the Experimental Analysis of Behavior, 1970, 14, 373-409.

Hodos, W. Progressive ratio as a measure of reward strength. Science, 1961, 134, 943-944.

Jung, R., & Hassler, R. The extrapyramidal motor system. In J. Field, H. W. Magoun, & V. E. Hall (Eds.), Handbook of Physiology, Section I: Neurophysiology, Vol. III. Washington, D. C.: American Physiological Society, 1960.

Kelleher, R. T. Chaining and conditioned reinforcement. In W. K. Honig (Ed.), Operant Behavior, Areas of Research and Application. Appleton-Century-Crofts, 1966.

Kelleher, R. T., & Gollub, L. R. A review of positive conditioned reinforcement. Journal of the Experimental Analysis of Behavior, 1962, 5, 543-597.

Keller, F., & Schoenfeld, W. N. Principles of Psychology. New York: Appleton-Century-Crofts, 1950.

Kirkby, R. J. Caudate nucleus lesions and maternal behavior in the rat. Psychonomic Science, 1967, 9, 601-602.

Kirkby, R. J. The caudate nucleus and active avoidance: A comment on the report of Winocur and Mills. Psychonomic Science, 1970, 18, 269.

Kirkby, R. J., & Kimble, D. P. Avoidance and escape behavior following striatal lesions in the rat. Experimental Neurology, 1968, 20, 215-227.

Kitsikis, A. The suppression of arm movement in monkeys: Threshold variations of caudate nucleus stimulation. Brain Research, 1968, 10, 460-462.

Konorski, J. Integrative Activity of the Brain. Chicago: University of Chicago Press, 1967.

Laursen, A. M. Movements evoked from the region of the caudate nucleus in cats. Acta Physiologica Scandinavica, 1962, 54, 175-184.

Laursen, A. M. Corpus striatum. Acta Physiologica Scandinavica, 1963, 59, 1-106.

Liles, S., & Davis, D. G. Athetoid and choreiform hyperkinesis produced by caudate lesions in the cat. Science, 1969, 164, 195-197.

Lynch, G. S. Separable forebrain systems controlling different manifestations of spontaneous activity. Journal of Comparative and Physiological Psychology, 1970, 70, 48-59.

MacLean, P. D. Cerebral evolution and emotional processes: New findings on the striatal complex. Annals of the New York Academy of Sciences, 1972, 193, 137-149.

Martin,J.P. The Basal Ganglia and Posture. Philadelphia: Lippincott, 1967.

McLennan, H., Emmons, P. P., & Plummer, P. M. Some behavioral effects of stimulation of the caudate nucleus in unrestrained cats. Canadian Journal of Physiology and Pharmacology, 1964, 42, 329-339.

Mechner, F. Probability relations within response sequences under ratio reinforcement. Journal of the Experimental Analysis of Behavior, 1958a, 1, 109-121.

Mechner, F. Sequential dependencies of the length of consecutive response runs. Journal of the Experimental Analysis of Behavior, 1958b, 1, 229-233.

Mettler, F. A. Relation between the pyramidal and extrapyramidal function. Proceedings of the Association for Research in Nervous and Mental Disease, 1942, 21, 602-655.

Mettler, F. A. Perceptual capacity, functions of the corpus striatum and schizophrenia. Psychiatric Quarterly, 1955, 29, 89-111.

Mettler, F. A., & Crandell, A. Relation between Parkinsonism and psychiatric disorders. Journal of Nervous and Mental Disease, 1959, 6, 551-563.

Mettler, F. A., & Mettler, C. C. The effects of striatal injury. Brain, 1942, 65, 242-255.

Meyer, M. E. Response inhibition of rats with frontal ablations and caudate-putamen lesions. Psychonomic Science, 1967, 8, 375-376.

Mikulas, W. L. Effects of lights at the choice point on spatial alternation and position learning by normal rats and rats with bilateral lesions of the caudate nucleus. Psychonomic Science, 1966, 5, 275-276.

Mikulas, W. L., & Isaacson, R. L. Impairment and perseveration in delayed tasks due to bilateral lesions of the caudate nucleus in rats. Psychonomic Science, 1965, 3, 485-486.

Miles, R. C. Learning by squirrel monkeys with frontal lesions. In J. M. Warren, & K. Akert (Eds.), *The Frontal Granular Cortex and Behavior*. New York: McGraw-Hill, 1964.

Mitcham, J. D., & Thomas, R. K. Comparison of substantia nigra and caudate nucleus lesions on avoidance learning in rats. *Journal of Comparative and Physiological Psychology*, 1972, *81*, 101-107.

Neill, D.B., & Grossman, S. P. Behavioral effects of lesions on cholinergic blockade of the dorsal and ventral caudate of rats. *Journal of Comparative and Physiological Psychology*, 1970, *71*, 311-317.

Olton, D. S., & Isaacson, R. L. Importance of spatial location in active avoidance tasks. *Journal of Comparative and Physiological Psychology*, 1968, *65*, 535--539.

Pellegrino, L. J., & Cushman, A. J. *A Stereotaxic Atlas of the Rat Brain*. New York: Appleton-Century-Crofts, 1967.

Platt, J. R., & Senkowski, P. C. Response-correlated stimulus functioning in homogeneous behavior chains. In J. H. Reynierse (Ed.), *Current Issues in Animal Learning*. Lincoln, Nebraska: University of Nebraska, 1970.

Potegal, M. Role of the caudate nucleus in spatial orientation in rats. *Journal of Comparative and Physiological Psychology*, 1969, *69*, 756-764.

Powell, R. W. Probability of reinforcement and fixed-ratio responding: A model. *The Psychological Record*, 1970, *20*, 73-80.

Pribram, K. H. Neocortical function in behavior. In H. F. Harlow, & C. N. Woolsey (Eds.), *Biological and Biochemical Bases of Behavior*. Madison, Wisconsin: University of Wisconsin, 1958.

Rescorla, R. A. Pavlovian conditioned inhibition. *Psychological Bulletin*, 1969, *72*, 77-94.

Rosvold, H. E. The prefrontal cortex and caudate nucleus: A system for effecting correction in response mechanisms. In C. Rupp (Ed.), *Mind as a Tissue*. New York: Haeber Medical Division, Harper & Row, 1968.

Rosvold, H. E., & Delgado, J. M. R. The effect on delayed-alternation test performance of stimulating or destroying electrically structures within the frontal lobes of the monkey's brain. *Journal of Comparative and Physiological Psychology*, 1956, *49*, 365-372.

Rosvold, H. E., & Szwarcbart, M. K. Neural structures involved in delayed-response performance. In J. M. Warren, & K. Akert (Eds.), *The Frontal Granular Cortex and Behavior*. New York: McGraw-Hill, 1964.

Rubenstein, E. D., & Delgado, J. M. R. Inhibition induced by forebrain stimulation in the monkey. *American Journal of Physiology*, 1963, *205*, 941-948.

Rye, M. M., & Elder, S. F. A suggestion concerning the anesthetization of the rat. *Journal of the Experimental Analysis of Behavior*, 1966, *9*, 243-244.

Schmaltz, L. W., & Isaacson, R. L. Effects of caudate and frontal lesions on retention and relearning of a DRL schedule. *Journal of Comparative and Physiological Psychology*, 1968, *65*, 343-348.

Schoenfeld, W. N., & Farmer, J. Reinforcement schedules and the "behavior stream." In W. N. Schoenfeld (Ed.), *The Theory of Reinforcement Schedules*. New York: Appleton-Century-Crofts, 1970.

Schwartzbaum, J. S., & Donovick, P. J. Discrimination reversal and spatial alternation associated with septal and caudate dysfunction in rats. _Journal of Comparative and Physiological Psychology_, 1968, _65_, 83-92.

Skinner, B. F. _Contingencies of Reinforcement: A Theoretical Analysis._ New York: Appleton-Century-Crofts, 1970.

Smith, B. H. _Principles of Clinical Neurology_. Chicago: Yearbook Medical, 1965.

Stamm, J. S. Electrical stimulation of monkey's prefrontal cortex during delayed-response performance. _Journal of Comparative and Physiological Psychology_, 1969, _67_, 535-546.

Thompson, R. L. Effects of lesions in the caudate nuclei and dorso-frontal cortex on conditioned avoidance behavior in cats. _Journal of Comparative and Physiological Psychology_, 1959, _52_, 650-659.

Thompson, R. L. The roles of striatum in adaptive behavior. _Project Report_, College of Physicians and Surgeons, Columbia University, 1963a.

Thompson, R. L. Effects of caudate lesions on performance of monkeys in mixed fixed ratio-extinction schedules. _American Psychologist_, 1963b, _18_, 464. (Abstract).

Thompson, R. L., Ayers, W. J., & Mettler, F. A. Comparison of effects of frontal and caudate lesions on timing and discrimination in monkeys. _American Psychologist_, 1962, _17_, 384. (Abstract).

Thompson, R. L, & Mettler, F. A. Permanent learning deficit associated with lesions in the caudate nucleus. _American Journal of Mental Deficiency_, 1963, _67_, 526-535.

Unger, R. An analysis of behavioral changes associated with lesions of the caudate nucleus in rats. Unpublished Ph.D. Dissertation, Harvard University, 1966.

Weiss, B., Laties, V. G., Siegel, L., & Goldstein, D. A computer analysis of serial interactions in spaced responding. Journal of the Experimental Analysis of Behavior, 1966, 9, 619-626.

Weissman, A. The behavioral effects of repeated exposure to three mixed extinction schedules. Journal of the Experimental Analysis of Behavior, 1960, 3, 115-122.

Whittier, J. R., & Orr, A. Hyperkinesia and other physiological effects of caudate deficit in the adult albino rat. Neurology, 1962, 12, 529-539.

Wilson, M. P., & Keller, F. S. On the selective reinforcement of spaced responses. Journal of Comparative and Physiological Psychology, 1953, 46, 190-193.

Winocur, G., & Mills, J. A. Effects of caudate lesions on avoidance behavior in rats. Journal of Comparative and Physiological Psychology, 1969, 68, 552-557.

Winocur, G., & Mills, J. A. The caudate nucleus and active avoidance: Reply to Kirkby. Psychonomic Science, 1970, 18, 270.

Wolf, G., & DiCara, L. Progressive morphologic changes in electolytic brain lesions. Experimental Neurology, 1969, 23, 529-536.

Wyers, E. J., & Deadwyler, S. A. Duration and nature of retrograde amnesia produced by stimulation of the caudate nucleus. Physiology and Behavior, 1971, 6, 840-846.

Wyers, E. J., Peeke, H. V. S., Williston, J. S., & Herz, M. J. Retroactive impairments of passive avoidance learning by stimulation of the caudate nucleus. Experimental Neurology, 1968, 22, 350-366.