THE UNIVERSITY OF MANITOBA

PROBING THE BEHAVIORAL EFFECTS OF NEO-NATAL GAMMA IRRADIATION IN THE RAT

by

DAVID R. HUMPHREYS

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF MASTER OF ARTS

DEPARTMENT OF PSYCHOLOGY

WINNIPEG, MANITOBA

MAY, 1971

Abstract

A recent interpretation of a motor agnosia to account for irradiated animals failure to adjust sequencing of motor chains during periods of nonreinforcement was tested in two experiments each designed to examine the effect of changing reinforcement schedule on the sequencing of bar presses.

The first study was employed to look at timing of the initial bar press to an intermittent CS when the reinforcement availability trailed the CS in time, (Delayed Conditioned Approach). Results showed that after an initial transient oscillation (Halasz, 1967) in which both irradiates and controls behaved similarly, the irradiated animals were significantly lower in response latency in the steady state. In addition it was found that irradiated rats responded at significantly higher rates between trials during which no reinforcement occurred and that this tendency decreased although did not reach control values after the insertion of a between trial second order DRL (trial abort Halasz, 1968).

The second study was employed to investigate the effects of changing the ratio demand in discriminated FR behavior. The results showed that irradiates and controls behaved similarly to changes in FR reinforcement schedule. Irradiates showed significantly higher responses rates between trials than did controls, which was decreased but not reduced to control values by inserting a second order between trial DRL.

Both experiments showed that neonatally irradiated rats seem to respond during periods of nonreinforcement as compared to control animals. The results were interpreted as a perseveration of response sets (Mishkin, 1964) although it is noted that reinterpretation of a motor agnosia hypothesis is a plausible alternative.

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It has been pointed out by Hicks (1953), Rugh (1959), and reiterated countless times Rugh & Grupp (1959), Furchtgott (1956, 1963), Rugh (1962), that proliferating tissues are particularly radiosensitive. It follows that developing organisms will be affected to a much greater extent by ionizing radiations than will the adult. Hicks (1953) has presented a timetable of developmental malformations produced by ionizing radiation. By irradiating mice and rats at known points during gestation and examining subsequent histological changes in the embryos and fetuses it has been shown that different areas of the nervous system are preferentially malformed as a function of the particular time of the radiation insult (Hicks, Brown & D'Amato, 1957; Hicks, D'Amato & Lowe, 1959). Rugh (1962) has compared the reported anomalies reported following human fetal X-irradiation with prenatal irradiation of the mouse and rat. As would be expected the observed lesions were shown to be a function of varying dosage levels as well as the time during gestation at which irradiation occurred. Brizzee et al. (1962) found variations in the thickness of cerebral cortical layers during prenatal exposure of rats to total body irradiation. Yamazaki et al. (1962) found changes in cerebellar size, cell density and arrangement of the folia in neonatally irradiated rats while Brownson (1962) investigated changes in the cerebellum purkinje cells, granual cells, pyramidal cortex, hypothalamus, brainstem, meninges, blood vessels and glia in rats irradiated as adults with massive doses of X-irradiation. It seems clear from these reports that the developing nervous system is sensitive to radiation during different periods of development and that specific parts may be preferentially affected over the time course of development.

Hicks (1958) has presented the case for using radiation as a tool

for studying mammalian developmental neurology. In addition to his timetable of lesion specificity and gestation period he has been able to study the migration of cells from the neuroectoderm to cortex as well as the rates of development of phylogenetically different parts of the brain. Following the example of Hicks, studies conducted by Cowen & Geuller (1960) showed long term changes resulting from prenatal X-irradiation. Subjects were exposed in utero to 250 rads at different stages of gestation. The animals were later sacrificed for examination between the ages of 3-19 months. It was shown that forebrain defects were most severely produced during days 15-16 while cerebellar lesions were prominent with later exposure dates.

Clemente et al. (1960) and Yamazaki et al. (1962) studied the effects of postnatal X-irradiation and found a prominence of cerebellar malformations, thus confirming the earlier timetables of Cowen & Geuller and Hicks. Kosmarskaya & Barashnev (1958) irradiated rats between postnatal days 1 and 33 with doses of 250 to 500 rads. They reported a decrease in the size of the brain when irradiation occurred within the first five days. Irradiation on day 14 postnatal did not result in a decrement in size but differences in shape were noted. These authors also noted the sensitivity of the cerebellum during the immediate postnatal period.

That the interest in the anatomical results of ionizing radiation should be soon followed by interest in possible behavioral effects of radiation was to be expected. By far the greatest amount of work regarding the effects of radiation on psychological processes has been done with adult animals where the radiation treatment has been administered after the nervous system has fully differentiated. In an investigation of possible CR disturbances Nemonov (cited by Stahl, 1959) failed to find

any changes in a salivary CR following a 1.5-kilorad cranial exposure with X-rays. An additional dose of 2.2 kilorads produced a reduction in CR magnitude. Lyman et al. (1933) found salivary CR changes with high dose exposure in dogs. Abdullin (1962) found disturbances in a conditioned leg flexion reflex after exposure of rabbits to 500 rads head only. It was found however that the disturbance was transient and full CR magnitude had recovered within two days. Livshits (1960) found that 2-3 weeks after a period of daily exposures (25 rads) totalling 600 rads, dogs exhibited a period of reduced amplitude of both salivary UR and CR.

Studying the effects of irradiation on instrumental conditioning, Arnold (1962) found no significant differences in either aquisition or retention of a simple bar pressing task after rats had been exposed to 2500 rads head only at the age of 90 days. Irradiating adult rhesus monkeys, Davis & McDowell (1962) found manifestations of disturbance in movement, activity, and visual survey. The animals were given two doses of 3000 rads each to various regions of the brain. Frontal lobe irradiated monkeys showed hyperactivity within a few days. Those animals irradiated in both frontal and posterior regions showed enhanced visual attention and some changes in posture. Harlow (1962) has cited evidence that the adult cerebral cortex may be more radioresistant than many other neural structures and offers this as an explanation for the observations of no adverse effects on learning in many adult irradiated animals. Brown and McDowell (1962) using rhesus monkeys report an increase in the concentration of attention which will result in a facilitation of performance on tasks placing a premium on attention to the location of the food reward. In the same animals there was a decrease in performance on tasks requiring attention to peripherally placed stimuli. Riopelle (1962) found few per-

manent losses in chimpanzees irradiated with 375-400 rads of gamma radiation over 12 hours. On a few tests conducted some years later some of the irradiated animals were found to be significantly inferior to control animals.

It would seem apparent that there are no easy explanations to account for the wide variation in results from experiments on adult irradiation. It is paradoxical that some authors report no effects with high dosages while others report deficits with much smaller doses. One obvious variable must be kept in mind, the means of irradiation. Whole body irradiation is going to have secondary effects on behavior which cannot be directly attributed to the nervous system. Such effects might include muscular weakness, changes in blood constituents and general metabolic rate, damage to peripheral receptors, digestion, and a host of others, (Kimeldorf & Hunt, 1965). It is clear that whether or not behavioral manifestations will occur after irradiation depends upon the behavioral measures taken. It would seem difficult at this point to attempt to attribute to radiation lesion in the adult any one particular behavioral result that remains constant from species to species and from method to method. Furthermore, it has been pointed out by Ordy et al. (1968) that often large dosages are required to produce any microscopic changes in the nerve cells of the adult animal. If such be the case it seems reasonable that lesion produced behavioral changes in the adult animal would be difficult to diagnose, especially in smaller dosages.

Behavioral Studies on the Irradiated Developing Nervous System

With the nervous system being more sensitive to ionizing radiation during its development it would seem logical that behavioral manifestations

might be more prominent when tested. On this assumption many investigators turned toward prenatal and neonatal exposure to radiation as a means of producing and investigating behavioral changes.

In one of the earlier studies, Furchtgott, Echols, & Openshaw (1958), subjected rats to 100 to 300 rads of X-rays on days 14 through 18 of the gestation period or neonatally. The rats were subsequently tested in a Lashley 3 maze at the age of 45-50 days. Learning deficits were inversely related to age at irradiation. In the group irradiated between day 14-15, 100 rads was effective while those subjects irradiated neonatally, 300 rads was necessary to produce decrements. Graham, Marks, and Ershoff (1959) tested brightness discrimination in rats which had been irradiated on day 10-18 of the gestation period. They found that rats irradiated on day 10 or 18 with 150 rads did not differ from controls, whereas 150 rads on day 14 or 300 rads on day 18 decreased the rate of acquisition of the task. These data coincide with the finding of Hicks (1953) that day 14 is the time of maximum sensitivity of the cerebral cortex to radiation.

Piotkovskiy & Kolomeitseva (as cited by Furchtgott, 1963) irradiated rats on day 18 and found acquisition and extinction of and instrumental response were significantly a slower than control animals. Haefner (as cited by Furchtgott, 1963) irradiated mice to 235-350 rads on days 6, 12, or 14 of gestation. He found a decrease in running speeds in a maze when they were tested as adults. It might be noted here that Sharp (1961) conducted an experiment to see if irradiation of the pregnant mother alone was sufficient to contribute to the deficits observed in the offspring. In one group he shielded the abdomen and irradiated the head and thorax only. In the other group the abdomen and legs were irradiated as well as the rest of the body. The offspring of the mothers which had been irrad-

iated with the abdomen shielded did not differ from a third control group, while those offspring whose mothers had received total body irradiation demonstrated impairment. It seems reasonable to conclude therefore that deficits observed in the offspring can be attributed to direct radiation effects.

Furchtgott & Wechkin (1962) found that avoidance conditioning in a Mower-Miller box was more rapid in rats which had been irradiated with 200 rads on day 16 than control subjects. Comparing these results with an earlier finding (Furchtgott & Echols, 1958a) where rats irradiated in utero showed greater activity and increased fearfulness, the authors hypothesized an emotionality enhancement in these animals. They attributed the faster acquisition of avoidance conditioning in prenatally irradiated rats to a hyperactivity and increased emotionality. In a more recent investigation, Sharp (1954) reported that rats irradiated with 200 rads on day 16 of gestation acquire a conditioned emotional response more rapidly than control animals. In a similar experiment by Deagle & Furchtgott (1968), rats irradiated with 200 rads on day 16 of gestation showed a faster acquisition of passive avoidance behavior. The same animals were superior in acquiring an active avoidance response also. Since passive avoidance calls for inhibiting responding these authors criticized earlier interpretations of increased avoidance behavior being due to hyperactivity alone; instead an emphasis was placed on increased emotionality.

Experiments by Kaplan (1962) showed that animals irradiated during gestation exhibited deficits in maze behavior but not in a bar pressing task. Kaplan also found differences in maze performance in irradiated rats depending upon the age of testing. These findings once again draw attention to the importance of a consistent and reliable diagnostic mea-

sure for assessing behavioral effects of lesions.

The Analysis of Radiation Produced Motor Deficits

Furchtgott & Echols (1958b) investigated locomotor coordination in rats irradiated between day 14 of gestation and neonatally. The behavioral task was to locomote with all four legs on two narrow parallel bars. Increments in the width between the bars were made in a step-view fashion until the animal could no longer negotiate the distance. Rats irradiated with only 50 rads on days 14-15 showed lower scores. In general it was found that there was an inverse relationship between age at the time of irradiation and the minimal effective dose. In a subsequent study, Furchtgott, Echols, & Dees (1960) reported deficits with dosages as low as 25 rads. Sharp (1961) reported similar data for rats tested at the age of 40 and 90 days. He did not, however, find any differences in irradiates and controls when tested at the age of 140 days. It should be kept in mind that the failure to find differences in the two groups as the animals grow older might reflect a decrement in performance in controls due to age rather than the lack of effect in the experimental animals. Wechkin, Elder & Furchtgott (1961) observed a deficit in the ability of rats irradiated on day 16-18 of gestation to climb an inclined plain. As in previous similar experiments the dose used was 200 rads. It is interesting to note that Furchtgott & Echols (1958a) found an increase in general activity in rats irradiated on days 14-18, while rats irradiated neonatally were hypoactive. These same authors found that prenatal irradiation enhanced locomotion in an open field, whereas neonatal irradiation inhibited it. Levinson (1962) found slower maze running in neonatally irradiated rats as well as more errors

in the maze. In a series of experiments by Wallace & Altman (1970a, 1970b) rats were irradiated neonatally in single and successive doses. The subjects were tested in 24 hour activity wheels in addition to a number of motor tasks such as pole climbing and weight pulling. In rats which had been given a single dose of 200 rads postnatally there were no significant motor disturbances when tested as adults. In animals which were given repeated doses of 200 rads on successive days there was a marked reduction in activity wheel scores as well as an obvious impairment in coordination and strength as demonstrated by the pole climbing and weight pulling tests.

The investigation into radiation produced motor deficits is interesting in two respects. Since it has been shown that different parts of the motor system develop at different periods of time it is not surprising to find different or even opposite motor disturbances depending on the time of the exposure to radiation. Therefore it may be that radiation could be used as a tool to probe the different components of the motor system during optimum periods of development, thus possibly revealing an insight into their individual roles in complex motor behavior. It is also of interest that many of the behavioral disturbances--some motoric, some not--are accompanied by obvious lesions in motor areas as revealed by histology. It has always been a keen point of interest to the psychologist to correlate anatomical deformations of the brain to observed behavioral changes. In this regard it is of interest to note that Yamazaki et al. (1962) found that the outstanding histological finding in neonatally irradiated rats is an atrophy of the cerebellum. This is in contrast to the findings in prenatally irradiated animals which show an atrophy of the forebrain with no significant change in the size of the cerebellum. Brownson (1962) found in postnatally

irradiated rats a decrease in the number of granule and purkinje cells in the cerebellar cortex along with signs of motor tremor. Schjeide et al. (1962) found a decrease in the size of the cerebrum and cerebellum of rats irradiated at two days of age. Altman, Anderson, & Wright (1969) changes in the external granule layer of the cerebellum in neonatally irradiated rats. It seems fair to conclude that irradiation between gestation days 14-18 produce most prominent changes in the cerebral cortex, especially in the thickness of cortical layers and also overall size. To what extent this will differentially affect learning tasks as opposed to pure motor skills tests is not clear. It would seem that those areas of the motor system (pyramidal cortex, basal ganglia, striatum) which reside in the cerebrum will be affected most by prenatal irradiation and to a lesser extent by neonatal irradiation. In contrast it appears that the cerebellum is most likely to be affected by neonatal exposure and to a lesser extent by prenatal irradiation. Therefore, motor disturbances observed as a result of selectively irradiating prenatally or postnatally may be related to the role of these different structures in the organization and integration of motor behavior. Unfortunately there is little solid evidence with which one can substantially attribute as being specifically induced motor impairments. The fact that prenatal rats are hyperactive and neonatal rats are hypoactive is of not much help in assessing the relative role of the cerebrum and cerebellum in motor behavior. The possibility exists that radiation produced changes in general activity may not be related to lesions observed in motor areas but rather a change in various metabolic functions that are unrelated to the nervous system itself but may run a somewhat parallel time course in development. In both clinical and animal experimentation there is no clear cut relation

between the anatomical locus of lesions and the degree of general activity, Mountcastle (1968). It is important to note that in Wallace and Altman's work they found motor coordination deficits resulting from exposure neonatally. It is interesting that this finding parallels their report of cerebellar malformations. It is quite possible that the low scores reported on the pole climbing and weight pulling tests can be attributed to cerebellar lesion.

The Cerebellum and Motor Behavior

It seems likely that with such obvious changes in the cerebellum after neonatal irradiation some behavioral motor component would be affected. The ease with which diagnosing such an impairment may on the other hand be somewhat difficult. As has been stated by Gilman & McDonald (1967a, 1967b) cerebellar insult is usually accompanied by a later compensation period where the organism regains the use of motor components. It has been reported by a number of authors, (Guyton, 1967; Mountcastle, 1968; Ruch & Patton, 1965; Glaser & Higgins, 1966) that cerebellar ablation results in an immediate ataxia and hypertonus in animals but that within weeks to months compensation occurs. It is possible that motor deficits caused by cerebellar irradiation might undergo compensation and hence obvious disturbances would escape measurement if the animals were tested as adults. This has two direct implications. First, if neonatally irradiated animals are to be tested for cerebellar related motor deficits, it might be of value to initiate testing soon after the lesion was introduced before compensation has taken place. A point worth remembering is that with radiation produced lesions in the cerebellum, the typical syndrome is reduction in cell numbers and malformation of cellular layers rather than

total absence of tissue as is the case with most experimental and many clinical lesions. Since functional connections still presumably exist--although they may be deranged--the resultant motor disturbances may be more elusive than in the case of other types of lesions. The second implication is that although compensation does occur it seems quite likely that some residual disturbance should still exist even in the adult. The degree of subtlety of these residual disturbances is likely to be a function of several variables, namely the kind of lesion (such as radiation as opposed to ablative), the size and magnitude (dose level), the age, and degree of compensation. The challenge then to psychologists and physiologists alike is to design measurements which are sensitive enough to reveal possible residual disturbances yet not overly contaminated by uncontrolled intervening variables.

In a series of recent investigations (Halasz, 1968; Halasz & Cheng, 1969; Halasz et al., 1970) an attempt has been made to quantify various components of conditioned behavior in a timing problem in hopes that contributions from such things as hyperactivity, ability to inhibit a previously reinforced response, and adjustment to changes in schedule could be partitioned and looked at individually. The basic paradigm consists of a delayed conditioned response (DCA) which are modifications of a conditioned approach response originally used in research with drugs and the central nervous system, (Halasz & Marazzi, 1964). In the DCA paradigm the reinforcement availability trails the onset of the conditioned stimulus for a given period of time. Initially the reinforcement availability coincides with the CS; after a period of training the delay is inserted in ramp, step or impulse-like form and subjects learn to adjust their response latencies to the level of the new demand, (Halasz, 1968). In

shorter delay demands, the adjustment of the response latency occurs because of the lack of reinforced premature bar presses. In longer delay periods there is a small steady error, a minimum deviation between the actual latencies and that demanded by the schedule, (Halasz, 1967). An additional penalty contingency can be inserted where very premature responses result in postponement of scheduled reinforcement availability. This acts to move response latencies closer to the demand level. Finally, another contingency has been introduced (trial abort) where a scheduled CS if preceded by a bar press within a given length of time is postponed for a period of time. This assures that the animal is not adventitiously pressing the bar at the onset of the CS. This contingency has been termed a between trial second order DRL, (Halasz, 1967).

In applying this DCA paradigm to the analysis of neonatal irradiated rats some interesting findings have arisen that are relevant to the behavioral disturbances associated with cerebellar lesions. In one investigation changes in the schedule demand of the DCA paradigm were instituted in ramp, step, and impulse like fashion (Halasz et al., 1970). In the condition of delay with no penalty, irradiated animals (200 rads on 3rd postnatal day) continued to bar press through periods of nonreinforcement and latencies did not adjust to the new delay demand level as did control animals. However, when the penalty contingency was inserted the irradiates adjusted to control values in response latency. In general the major finding was that the irradiated animals responded more than controls during periods of nonreinforcement including inter-trial intervals. At first glance one might conclude that these findings represented the general inability of the animal to inhibit; however, this cannot be so since when forced to prolong response latencies via the penalty contingency, the ir-

radiates did not differ from control animals. An alternative explanation of hyperactivity might suffice were it not for the fact that running wheel data was taken an hour before each training session resulting in the finding that irradiated animals were not as active as controls; thus confirming the earlier findings of Furchtgott & Echols (1958). In a prior series of unpublished pilot studies, Halasz (personal communication, 1969) studied the effects of introducing a penalty contingency which was shorter than the delay contingency. That is to say, the very premature bar presses were punished with the postponement of scheduled reinforcement availability, while bar presses which occurred after a given time yet before the onset of reinforcement availability during the delay period, merely went unreinforced. This type of paradigm establishes a tolerably low level of premature responses. It was found that control animals adjusted their response latencies near the level of the demanded delay while the irradiates adjust near the penalty value. Thus, when forced to inhibit premature responses the irradiated animals will adjust response latencies to the level demanded by the penalty only. Non reinforcement during the CS by itself does not appear to be a sufficient inhibitory stimulus to irradiated animals as it appears to be in control animals.

It seems rather paradoxical that nonreinforcement either during the CS (premature latencies) or during the inter-trial interval (prolonged responding after termination of the CS) does not act to reduce inappropriate responding, whereas postponement of a CS either in the trial abort contingency or in the penalty contingency is an effective inhibitory stimulus. Such disturbances have been interpreted by Halasz et al. (1970) as a "motor agnosia" where the organism is unable to relate the reinforcement consequences of each individual motor component in a series. While retaining

the ability to adjust the entire sequence, this interpretation has interesting implications with regard to two opposing theories concerning the mechanisms of delayed response and timing behavior. It should be noted that this interpretation of motor agnosia is not the classical interpretation which has been applied to observed dysmetria in cerebellar clinical syndromes (Kappers, Huber & Crosby, 1936).

The Possible Role of the Cerebellum in Delayed Response and Temporal Discrimination

Pavlov (1927) has described inhibition of delay as resulting from the CS in conjunction with its prolonged duration acting as a compound stimulus which gains inhibitory properties because of lack of immediate reinforcement by the UCS. Many modern investigators have experimented with variations of a delayed response paradigm (cf. Warren & Akert, 1964). Much of the research so far conducted favors the hypothesis of cortical inhibition as a mechanism mediating the delayed response with special attention to the frontal lobe (Konorski, 1964, 1967; Mishkin et al. 1962; Pribram, 1955; Rosvold & Szwarcbart, 1964). On the other hand there are those who think that mediation of the delayed response is a function of postural mechanisms. Wilson & Keller (1953) have observed that as increments in a differential reinforcement of low rates (DRL) schedule, viz., 10, 20, 25 sec., are instituted that not only is there a decrease in response rate but each subject develops clearly defined "collateral" behavior which is incompatable with bar pressing. It has been suggested that this other behavior serves as a discriminative stimulus for the timed response. As the delay interval increases more links are added to the collateral chain. The strength of such a collateral chain is

thought to be maintained by conditioned reinforcers provided by the stimuli arising from attending the collateral chain. Laties et al. (1965) have supported such a hypothesis by showing that modification of collateral behavior via certain drugs also modifies the timed response. Konorski (1967) has emphasized the importance of kinesthetic-spatial relationships in delayed response and Stamm (1970) has found that frontal monkeys made errors in delayed alternation and visual discrimination by developing strong postural habits. He also noted that the severity of the lesion effect was directly related to the amount of motor involvement in the task, (monkeys in a restraining chair performed better than those in a maze).

To what degree the cerebellum is involved in spatial-kinesthetic association is not clear. Botterell & Fulton (1938) found that removal of the ansiform lobe of the cerebellum in monkeys produced a peculiar disturbance. Monkeys were allowed to run down an alleyway or corridor; upon coming to the end the animals ran headlong into the wall as if it were not there. These authors reported that the animals didn't appear to be ataxic. This is in contrast to the most frequent reports of cerebellar injury, particularly the anterior lobe, (Mountcastle, 1968). Typical clinical syndromes resulting from cerebellar damage are intention tremor, the tendency for a limb to escillate immediately prior to its attainment of a desired position; disdiadokokinesia, the inability to perform rapidly alternative movements--apparently a reflection of impaired anticipatory motor adjustments; dysmetria, errors in the range of movement (in touching a point arresting the action before reaching it, or shooting past it, deviation in line of movement (eg., carrying food to the ear instead of the mouth) and asynergia, a lack of cooperation

between muscles. (Mountcastle, 1968; Kappers, Huber, & Crosby, 1936). It has been suggested (Ruch & Patton, 1965; Mountcastle, 1968; Thach & Evarts, 1969) that different parts of the cerebellum serve different functions with regard to the integration of motor behavior. If this is so it might explain some of the disparities in experimental and clinical findings.

The interpretation of a motor agnosia (Halasz, et al.,1970) with regard to temporal discrimination poses a number of interesting questions with regard to the function of the cerebellum. These authors reported the cerebellar deformation which is typical of neonatal irradiated animals. Because of its unique anatomical arrangement (cf. Eccles, et al., 1967) the cerebellum has often been referred to as a proprioceptive organ, (Guyton, 1967). This fact makes it an attractive candidate for a mediator in behavior which is dependent on kinesthetic input. There are those who feel that the cerebellum plays a role in modifying or actually initiating motor movement in conjunction with the cerebral pyramidal system, (Thach & Evarts, 1969; Marr, 1969; Thach, 1970a, 1970b). Others, however, look at the cerebellum as merely a "fine regulator" or error detector which functions to make corrections in force and direction of fine movement, (Henatsch et al., 1964; Guyton, 1967; Mountcastle, 1968). Under the second hypothesis one would not expect that individual responses in a chain would be affected by cerebellar lesion exept in terms of their force, duration and accuracy. It would be assumed that each individual response was organized at some level other than the cerebellum (this should be taken to include the possible cerebellar modification of an output organized elsewhere; ie., an executive role as opposed to a regulative role). If the initiation of individual components in a motor chain

were independent of the status of the cerebellum one should expect to see disturbances in the fine regulation of each component. If on the other hand the sequencing of components of a motor chain is cerebellar dependent--that is, the cerebellum works to some extent to modify the programming of components in a chain after the chain has once been initiated--then disturbances in motor chaining might conceivably be disturbed in some way. A motor agnosia hypothesis would certainly favor the latter. If one assumes that first, the disturbance seen in the irradiated animals in question is a deficit in the ability to relate individual components in a motor chain to reinforcement contingencies and second, that the observed defect is due to the cerebellar lesion, then the obvious conclusion is that the cerebellum is more than just an error detector in fine movement. These assumptions imply that the cerebellum has an integrative organizational function with respect to the programming of motor sequences. It should be noted here, that while it is more parsimonious to attribute the motor agnosia hypothesis to the cerebellum in light of the observed lesions, it is not necessary to do so.

If the cerebellum is involved in the organization and integration of components in a motor chain, a deficit in this function might be revealed by changing the demanding contingencies which require the animal to make adjustments in motor sequencing. If such a deficit is a generalized inability to make adjustments in motor chains in response to changing reinforcement contingencies, one would expect that impairments in a number of situations where motor chaining is involved would be indicated.

It is the purpose of this study to investigate this interpretation of the motor agnosia hypothesis with regard to neonatally irradiated animals. The experiments reported here will be directed toward the

possible elucidation of the role of the cerebellum in motor chaining, in the hope that information may be generated by which tentative conclusions might be reached. As already indicated the underlying assumption is that a motor agnosia as discussed by Halasz et al. (1970), is manifested by a generalized inability to adjust motor sequences in response to changing reinforcement contingencies while retaining the ability to adjust the entire sequence. To test this assumption of generality two experiments will be performed, each utilizing a different conditioned behavior under the control of two types of reinforcement contingency, where it is assumed that in each case there is a common mediating process,--namely motor chaining.

In the first experiment reinforcement contingencies control the motoric chaining which presumably mediates the temporal spacing of initial bar presses to a signal. In the second, reinforcement contingencies control the number of actual components in a motor chain without reference to their temporal occurance. If there is a generalized inability in irradiates to adjust components of a chain then deficits would be expected in both experiments.

Experiment #1

The DCA paradigm (Halasz, 1968) has been used to investigate the temporal spacing of a conditioned response where the animal has to delay the initial response to the onset of a time randomized CS. For longer delay periods a penalty contingency has been used to force the response latency within a tolerable level of steady error. In addition a between trial second order DRL (trial abort) has been inserted which postpones a scheduled CS occurrance if it is preceded within a determined length of

time by a bar press. In previous studies (Halasz, 1968; Halasz et al., 1970) when a delay in reinforcement availability was inserted, reinforcement availability was protracted beyond the length of the CS for a time equal to the delay, such that each trial would be accompanied by the same amount of reinforcement availability regardless of the value of the delay. This resulted in some bar presses being reinforced in the absence of the conditioned stimulus. In the following experiment in order to determine whether or not the higher incidence of bar pressing in the intertrail interval was due to such an arrangement of reinforcement contingencies or to some other effect, reinforcement availability always terminated with the termination of the CS. This resulted in decreased total time of reinforcement availability as the delay contingencies were inserted. In addition, no penalty condition was used in this experiment in order that delayed responses in both control and experimental animals could be observed in long delay periods without the addition of further reinforcement contingencies.

Method

Subjects: Eight male albino rats of the Holtzman strain were used. The rats were divided into two groups of four each. Each animal in one group was paired with its litter mate in the other group. One group was irradiated with ^{60}Co at a dose of 200 rads on neonatal day three. The other group served as a control group and was run through a sham irradiation procedure to control for handling effects. The animals were housed individually in a 6 X 6 X 8 wire cages with free access to food at all times. All animals were 60 days of age at the onset of training procedures.

Apparatus: A modified Skinner box which was programmed for water reinforcement on the right lever was used for all training. Reinforcement was delivered by means of a solenoid operated dipper positioned in the center of the front panel. Each dipperful of water contained approximately 2-3 drops. The CS, a 1000 hz tone from an Eico model 377 audio generator was delivered through a speaker in the front panel of the Skinner box. The intensity setting on the audio generator was 80 db. The right lever was connected to a strain gauge and wired to a DC amplifier which was connected to a Hewlett-Packard 130C oscilloscope for measuring amplitude and time course of individual bar presses. Bar presses were photographed with a HP197a oscilloscope camera. Response latencies were measured by a printout counter which began to count at the onset of each CS and terminated with the first bar press. All programming of experimental contingencies was accomplished by means of BRS logic units. Counters were hooked up to record the number of bar presses during the CS, the number of bar presses during the inter trial interval and the number of aborted trials.

Procedure: At the initiation of training each subject was put on a 23 hour water deprivation schedule. The rats were trained daily in one hour sessions. After the end of each training session each rat was given 15 minutes free access to water before it was removed from the home cage. Post session water consumption was measured and recorded.

Training consisted of four distinct phases: CRF and SD conditioning, trial abort conditioning, first delay insertion (step-type increase), and second delay insertion.

CRF-SD conditioning: Animals were shaped to press the lever in the ab-

sence of any tone until steady bar pressing was observed. At this point, the CS which was 30 seconds in duration and randomized over an average one minute interval. Responses during the CS were reinforced while those between trials were not. Response latencies were measured at this point and continued throughout the duration of the experiment. Each session consisted of 40 trials.

Trial Abort conditioning: After total responses / session in each group had stabilized the trial abort contingency was inserted. The duration of the trial abort was 15 seconds. Any response occurring 15 seconds prior to the next scheduled CS as determined by the randomizer was postponed for another randomized cycle. The number of aborted trials in each session for each group was recorded.

First step-like delay: When response latencies had stabilized, the reinforcement delay contingency was introduced. Reinforcement availability trailed the onset of the CS by 10 seconds which resulted in a total reinforcement time of 20 seconds per trial. The procedure for instituting the step delay was to allow the animal full access to reinforcement for the first 20 trials of the session. The last 20 trials were with the delay contingency. Response latencies for the first 20 and last 20 trials were computed separately. Subsequent training sessions were run with the delay contingency in effect.

Second Step-like delay: When response latencies had stabilized from the initial delay, a second 10 second delay was inserted in the same manner as before, which resulted in reinforcement availability trailing the onset of the CS by 20 seconds; thus, total reinforcement time in each trial was 10 seconds.

Photographs were taken of the sequential distribution of bar presses

during all phases of training although not continually from day to day. The onset of the CS was programmed to trigger the sweep of the oscilloscope and the shutter of the scope camera. Thus, the sequential distribution, amplitude (force) and duration of each individual bar press could be photographed.

At the termination of the experiment the animals were sacrificed, their brains perfused and histology was done to determine the extent of radiation produced lesions. Special attention was directed to the size of the cerebellum and cerebrum as compared to control animals in addition to deformation of cortical layers in the cerebellum. Photographs of coronal sections of the cerebellum were taken.

Results

Figure 1 graphically shows the daily mean response latency of each group. Four days of baseline responding under the CS-Trial abort phase are compared with the following days during which the two step-like increases in delay were implemented. The graph does not differ significantly from that shown. A T-test for differences between the mean of the group daily means was conducted. In all T-tests taken the estimated standard error was computed according to Hays (1963) for differences between group means with small N. It was found that prior to the first delay increment there were no significant differences ($t=.21.6df\ P<.8$, Appendix II, Table 2) in response latencies between the control and irradiated groups. Latencies are expressed in eighths of seconds. On the two days that a change in delay was implemented the mean latency of each subject was computed over the entire fourty trials so that each daily mean used in computing the group mean would have equal number. A T-test for the three days fol-

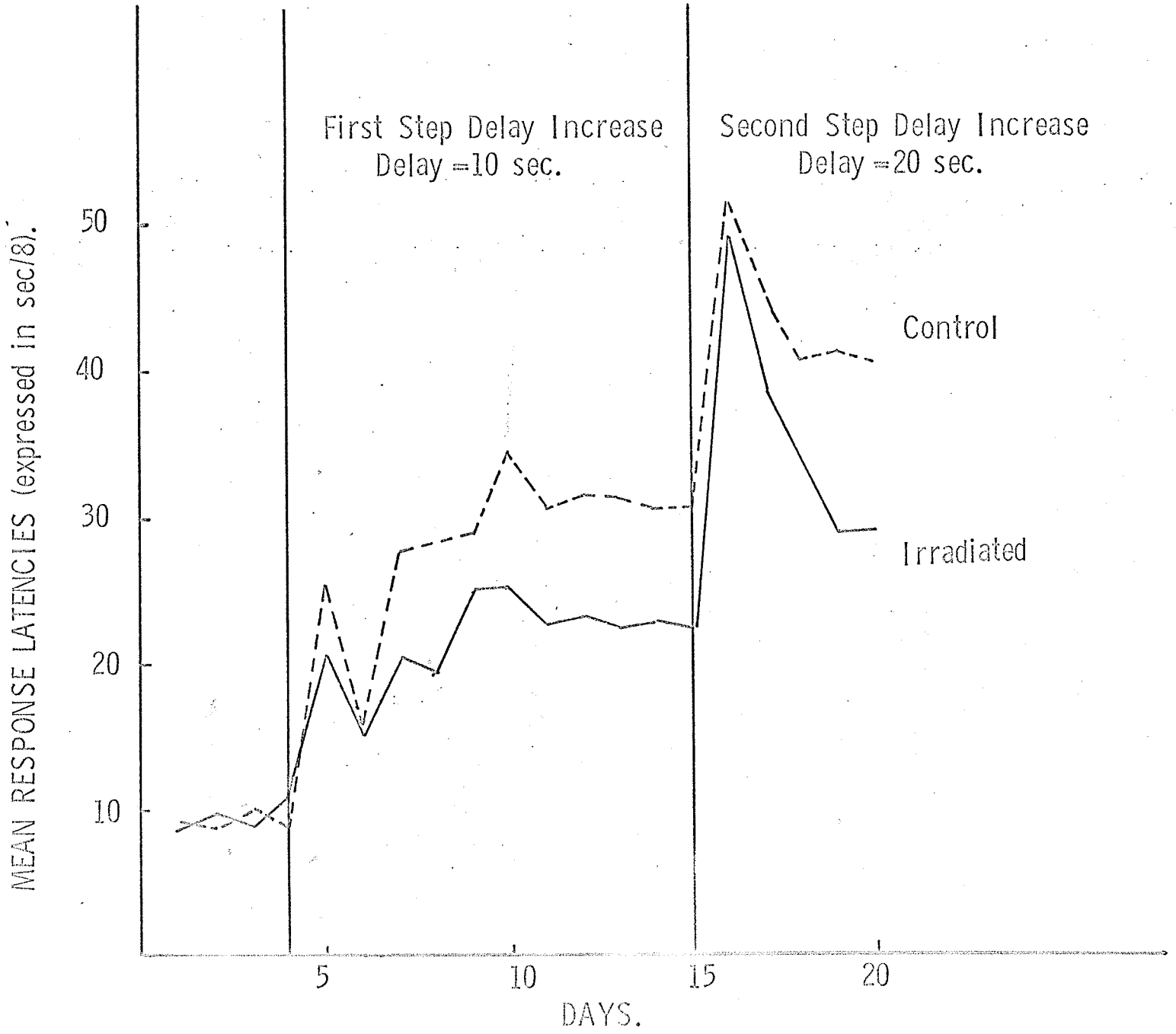
First Step Delay Increase
Delay =10 sec.
Second Step Delay Increase
Delay =20 sec.
Control
Irradiated
MEAN RESPONSE LATENCIES (expressed in sec/8).
50
40
30
20
10
5
10
15
20
DAYS.

Figure 1

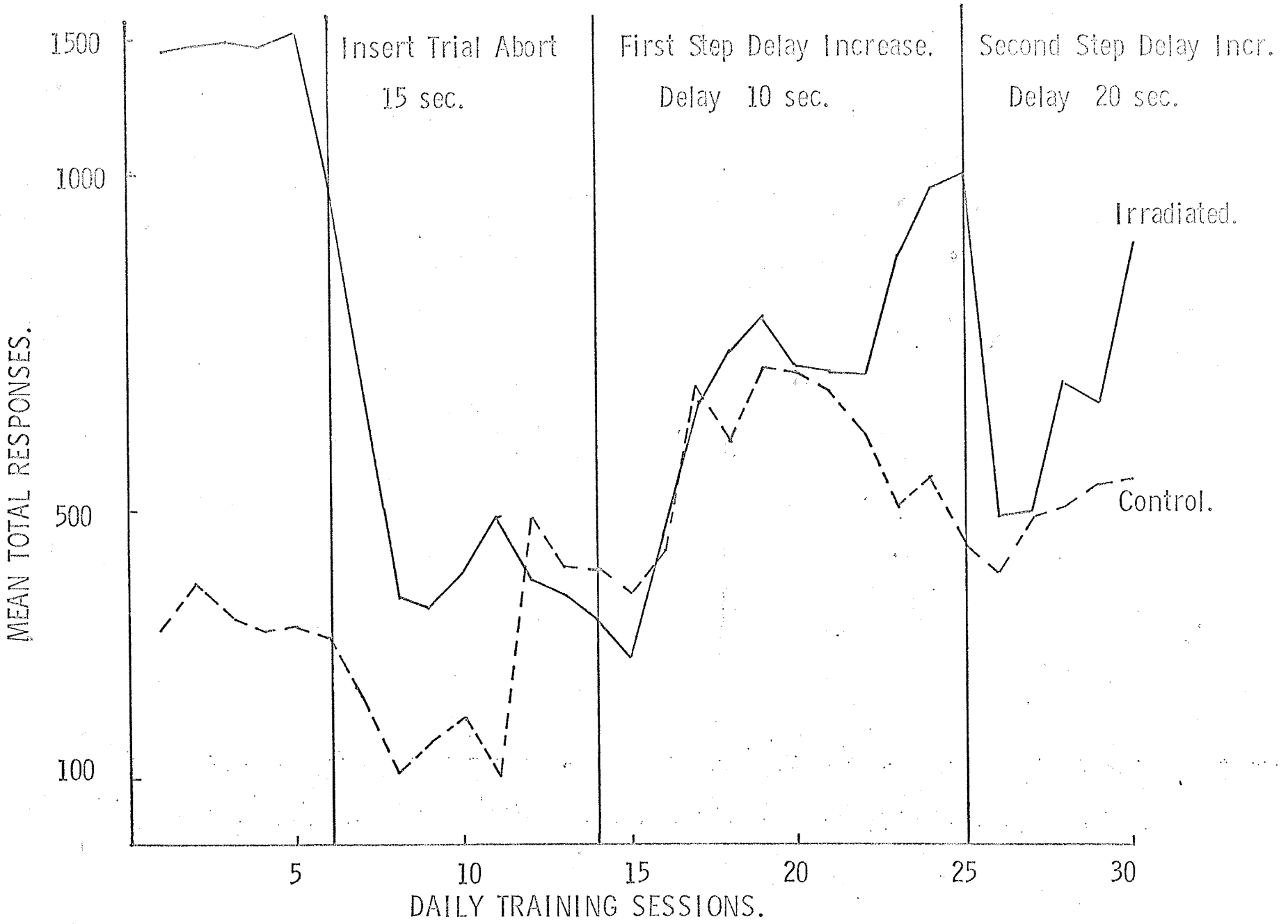
1500
1000
500
100
MEAN TOTAL RESPONSES.
Insert Trial Abort
15 sec.
First Step Delay Increase.
Delay 10 sec.
Second Step Delay Incr.
Delay 20 sec.
Irradiated.
Control.
5
10
15
20
25
30
DAILY TRAINING SESSIONS.

Figure 2

lowing the first delay increment showed no significant differences in response latency ($t = .733$, 4df $P < .5$, Appendix II, Table 2). It can be seen in Figure 1 that both groups response latency increased with the insertion of the first delay. After initial oscillations each group stabilized over a five day period preceding the second delay increase. It is interesting to note that the irradiated group stabilized at a much lower response latency than did control animals. The differences over a four day period prior to the second delay increase were significant ($t=23.0$, 6df $P < .001$, Appendix 11, Table 2). With the second increment in the delay (20 seconds) both groups were shown to increase their response latencies in the same fashion as with the first delay. The response latencies of the two groups during the first three days following the second increase in delay were not significantly different ($t=.81$ 4df $P < .2$, Appendix 11, Table 2). The last three days show a significant difference ($t=4.8$, 4df $P < .01$, Appendix 11, Table 2). Again, irradiated animals adjusted to a lower level than did control animals. The increase in delay was not as effective in increasing response latencies in the irradiated group as in controls. It might be interesting to note that it appears that the recovery from the second delay is greater in both animals but more so for the irradiated group. This finding is in agreement with the previous findings of Halasz (1967) in normal animals where he notes as steady error in response latencies where the animals do not completely adjust to the level demanded by the schedule change. This effect seems particularly prominent in long delay periods such as in this experiment. The demanded level of the first delay increment would be 80 on the graph (seconds/8) and 160 for the second delay increment. Thus, a large steady error is obvious in both groups under both delay conditions.

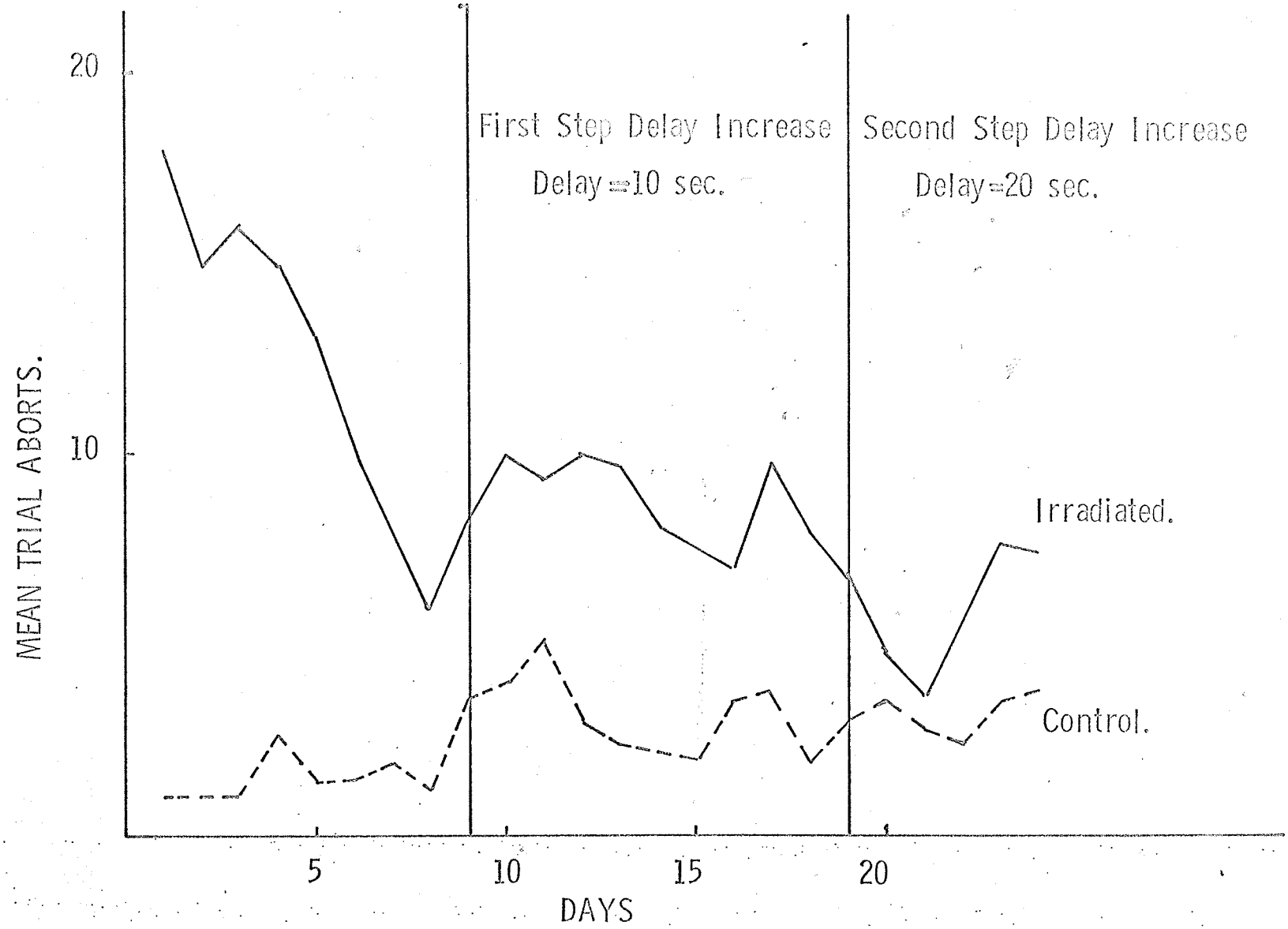
20
10
MEAN TRIAL ABORTS.
First Step Delay Increase
Delay=10 sec.
Second Step Delay Increase
Delay=20 sec.
Irradiated.
Control.
5
10
15
20
DAYS

Figure 3

In Figure 2 daily group means (Appendix 11, Table 3) are graphed over training sessions. Shaping data has been omitted. The number of total responses during CS conditioning in the irradiated group is significantly different ($t=17.4$, 10df, $P<.001$, Appendix 11, Table 4). With the insertion of the trial abort contingency the total responses in both groups initially decrease. The last three days of trial abort conditioning prior to the first delay increment are not significantly different with respect to total responses ($t=2.25$, 4df, $P<.05$, Appendix 11, Table 4). With the first delay both groups increase total responses at about the same level for the first six days. In the following four days the number of total responses in the irradiated group is significantly larger ($t=3.1$, 6df, $p<.05$, Appendix 11, Table 4). With the onset of the second delay, there are no significant differences ($t=.98$, 4df, $P<.2$, Appendix 11, Table 4) in total responses for the first three days. The last three days show that total responses in the irradiated group was significantly higher ($t=3.54$, 4df, $P<.05$, Appendix 11, Table 4) than control animals. It is apparent that irradiated animals respond more times in a training session than control animals during the initial training phases. When the trial abort contingency is introduced it acts as a force by which the irradiates learn to inhibit such high rates of responding. It would appear however, that this may be a transient effect as evidenced by a tendency of the irradiated group to return to high levels of responding. It should be pointed out that the insertion of the delay contingency results in both groups increasing the total number of responses per session. This however, is much more obvious in irradiated animals than in control subjects.

Insight into what extent the higher total responses in irradiates

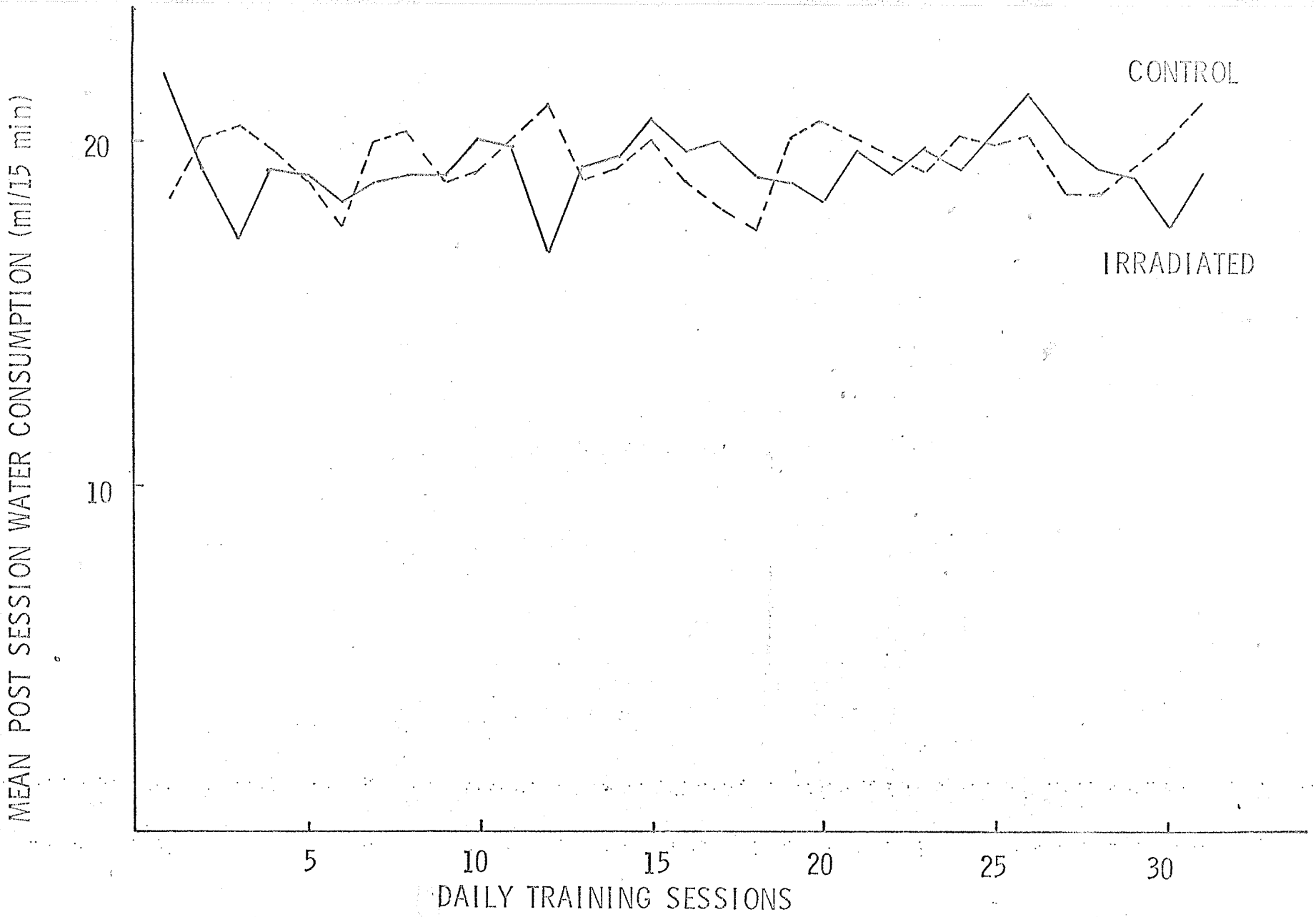
CONTROL
IRRADIATED
MEAN POST SESSION WATER CONSUMPTION (ml/15 min)
20
10
5
10
15
20
25
30
DAILY TRAINING SESSIONS

Figure 4

as compared to controls is during the inter-trial interval is gained by referring to Figure 3. Daily group mean trial aborts are graphed across training sessions, (Appendix 11, Table 5). At the onset of trial abort conditioning the irradiated animals make significantly more trial aborts ($t=3.86$, 14df, $p<.001$, Appendix 11, Table 5) during the following eight days than do controls. With the introduction of the first delay it is shown that over the following eleven days the irradiated group makes significantly more trial aborts than control animals. ($t=4.5$, 20df, $P<.001$, Appendix 11, Table 5). In the five days following the second delay irradiated animals make significantly more trial aborts than controls ($t=1.93$, 8df, $P<.05$, Appendix 11, Table 5). It is obvious that irradiated animals make significantly more trial aborts throughout the experiment than control animals. It should be kept in mind that since the onset of any CS is randomized and since the trial abort contingency always precedes the CS by 15 seconds, this measure should be a fairly good estimation of the relative frequency of inter-trial responding. It would seem, therefore, that a portion of the higher total responses in irradiated animals can be accounted for during the inter-trial interval. It is important to realize that the trial abort contingency results in a decrease in total responses in addition to a decreasing number of trial aborts as the experiment progresses; however, in this experiment the irradiated group never settled down to control levels in either total responses or trial aborts. That the introduction of the delay contingencies had any systematic effects on the number of trial aborts as a dependent measure was not apparent.

To control for possible motivational differences in the two groups, post-training session water consumption levels were recorded each day.

Figure 4 shows group mean water consumption (Appendix 11, Table 6) over training session. It is obvious that the two groups did not differ significantly in the amount of water consumed after each training session.

Appendix 111 contains photographs taken from two control (top) and two irradiated (bottom) animals during various phases of conditioning. There appears tentative evidence that bar presses (upward deflection) in irradiates may be accompanied by motor tremor, although at this point such a conclusion is highly speculative and a more systematic investigation is necessary in order to reveal any consistent motor tremor.

Appendix Vl contains histological data from samples of both groups of this experiment. The most prominent effect was marked cerebellar deformation which confirms earlier investigations. Caliper measurements were taken of the forebrain in irradiated and control animals and it was found that the overall size of the forebrain was smaller in irradiates than in controls, however, it was difficult to tell whether the difference in size was significant or not. A more detailed histological exploration of the forebrain is required before definite conclusions can be made, as to the nature of forebrain damage in these neonatally irradiated animals.

Discussion

The results of this experiment have demonstrated a number of points concerning the behavior of irradiated animals in a delayed conditioned approach paradigm. Response latencies have been used as rough indices of timing behavior where the experimenter has been looking at the adjustment of such latencies in response to new reinforcement contingencies. In the absence of a delay contingency there is no difference in the CR latencies to CS of irradiated and control animals. The two groups behave similarly in the initial periods of delay although the irradiates tend to be affected to a lesser extent. This may reflect different mediating mechanisms in the transient and steady state which may be differentially affected. In both delay periods investigated in this experiment it was found that the irradiated animals are more resistant to the forces which tend to increase the response latencies of normal animals. It would be presumptuous to call this a deficiency in timing ability since even the control animals did not adjust to the level demanded by the schedule change. In addition, with the additional contingency added (penalty) Halasz et al. (1970) have shown that irradiated animals do not differ from controls in their ability to inhibit early responding. It would be more appropriate to term the observed effect a resistance to the effects of non-reward. That this idea is further supported in this experiment is evidenced by the higher total number of responses with parallel higher numbers of trial aborts which reflect the relative frequency of responding during the inter-trial interval. Clearly, the outstanding observation that seemed consistent throughout the entire experiment was that irradiated rats have a strong tendency to respond in periods of nonreinforcement. The fact that introduction of the trial abort contingency

serves to decrease the inter-trial responding might lead to an interesting hypothesis. The effect of the trial abort is to postpone the occurrence of a CS which is associated with reinforcement. It seems paradoxical to talk in terms of resistance to non-reward and at the same time observe that a postponement of a stimulus associated with reward, or second order DRL, is effective in partially removing inter-trial responding. It is interesting that the penalty contingency, not employed in this experiment, operationally performs a similar function --that is to postpone (after terminating) a stimulus associated with reinforcement. It would appear that removing or postponing a stimulus associated with reward is effective in forcing the animal to inhibit, while actual responding (either during the stimulus in delay period or in the absence of it) without reinforcement is not so effective. Such evidence appears to favor the motor agnosia hypothesis which states that the animal cannot relate the reinforcement consequences of the behavior to individual components in a motor chain. Presumably the CS triggers the initiation of the motor chain which is then taken over by different parts of the brain which might be affected by the radiation lesion. In this line of reasoning, one draws a distinction between the initiation of a motor chain and the execution of the individual components in the chain. That there is no doubt a difference between ballistic type movements and exploratory or finer movements (Konorski, 1967; Granit et al. 1956) and the mechanisms involved in their initiation and execution seems to be in line with clinical findings; however, in this experimental situation the initial motor component (first bar press) is no different in topography from the second or last one. It seems unreasonable that the nervous system would involve one structure with the initial bar press

and then switch to another system for the execution of the rest of the chain. Furthermore, if one assumes that timing (inhibition during a given period of time) behavior is mediated by collateral chaining (this need not necessarily be so) then operationally we have in a period of time during the CS, while the rat is inhibiting the bar press in the delay and/or penalty situation, the interaction of two motor chains each consisting of individual components. If the motor agnosia interpretation can be generalized to such collateral behavior it would seem that the deficit would prevent the successful switching from one chain (collateral) to another (bar pressing). In other words, if the animal has the inability to relate reinforcement to individual components of a motor chain, why wouldn't the animal continue to emit collateral chaining once it has started? Future experimentation is needed where an experimental situation is such that motor chains and their individual components are precisely identified so that one is able to study initiation and execution of motoric behavior.

Experiment #2

If the motor agnosia syndrome as described by Halasz et al. (1970) is generalized to all behaviors mediated via motor chaining, one would expect that the sequencing of individual bar presses in an FR schedule of reinforcement might be affected if the reinforcement contingencies were to be suddenly changed. This should be so if one defines a motor chain to reinforcement consequences. The fixed ratio has been described as a schedule where individual bar presses serve as conditioned reinforcer for the subsequent motor components (Ferster & Skinner, 1957). It seems quite obvious that the conditioned stimulus for subsequent bar presses in an FR chain are set up by the proprioceptive activity generated by previous bar presses. Since the cerebellum is an ideal candidate for a proprioceptive organ, and in line with the motor agnosia hypothesis, one might hypothesize that it is the interaction between proprioceptive input to the cerebellum in conjunction with changing reinforcement contingency demands that result in adjustment of motor sequences in the normal animal or the failure to do so in the irradiated animal. This experiment tests the effects of sudden changes in the demand of an FR schedule on performance levels which utilize motor chaining. In order that similar comparisons could be made to Experiment #1 the same basic experimental setup was used. The subjects were reinforced for FR behavior to a CS but not between trials. The only difference in this experiment was the nature of the response to be adjusted--the number of individual responses rather than the temporal spacing of the initial response.

Method

Subjects: Eight male albino rats of the Holtzman strain were used. The rats were divided into two groups of four each. Each animal in one group was paired with its litter mate in the other group. Radiation procedures were carried out in identical fashion as in the first experiment, with the control group getting a sham radiation session. Animals were all housed in 6 X 6 X 8 wire cages with free access to food. The animals were 60 days old at the onset of training.

Apparatus: All equipment reported in Experiment #1 was utilized in this experiment in the same fashion. In addition a Lehigh Valley 240-50 stepper was used to count FR responses. Reinforcement was water delivered through the centrally located dipper. The strain gauge, DC amplifier, HP 130C oscilloscope and the HP 197A oscilloscope camera were used to photograph the sequential distribution of bar presses in a trial. Counters measured the number of bar presses during the CS, inter-trial interval and number of aborted trials.

Procedure: At the onset of training each subject was put on a 23 hour water deprivation schedule. The rats were trained daily in one hour sessions. After the end of each training session each rat was given 15 minutes free access to water before it was removed from the cage. Postsession water consumption was measured and recorded. Training was broken down into three phases; FR shaping and CS conditioning, trial abort conditioning, and step-like increment in ratio demand.

FR-CS conditioning: Animals were initially shaped in the absence of the CS (tone) to a criterion of FR-10. When steady responding was apparent

the CS was introduced and responses during the inter-trial interval were not reinforced. The duration of the CS was 30 seconds and its onset was randomized over an average one minute interval. Each session consisted of forty trials. Response latencies were recorded in the same manner as in Experiment #1.

Trial abort conditioning: When total responses/session had stabilized the trial abort contingency was introduced in the same manner as Experiment #1. The duration of the trial abort contingency was 15 seconds. The number of aborted trials was recorded on a counter.

Step-like increase in ratio demand: When the number of aborted trials approached an asymptote and as the total responses/session stabilized an increment in the ratio demand was made. The procedure for instituting the changes was to allow the first 20 trials to reinforce on an FR 10 and the second 20 trials reinforced on an FR 50 on that day. Subsequent training sessions were run with the new ratio demand in effect.

Photographs of the distribution of bar presses during trials were taken in all phases of the experiment although not every day. As in Experiment #1 the oscilloscope was triggered by the onset of the CS as was the shutter of the camera.

After the termination of the experiment animals were sacrificed and histology was performed as in Experiment #1.

Results

Figure 5 shows group daily mean latencies over training sessions as in Experiment #1. Means were computed over the entire experimental duration and a t test for difference between group means was taken, (Appendix IV, Table 1). There were no significant differences (t=.5,

40df, P<.50, Appendix IV, Table 1) in response latency between the two groups during the experiment.

Figure 6 shows group daily mean total responses over training sessions. The graph is divided into the four experimental phases, FR shaping, Conditioned Stimulus-FR training. Trial abort conditioning, and ratio increment training. During the FR shaping phase the differences between the Control and irradiated groups were not significant (t=.59, 10df, P<.50, Appendix IV, Table 2). During the phase of CS conditioning where the animals were rewarded on an FR-10 during the CS and not rewarded between trials, the irradiates were not significantly higher in total responses than control animals (t=1.18, 12df, P<.10, Appendix IV, Table 2). In the trial abort conditioning phase, the total responses of the irradiates was significantly higher (t=2.0, 16df, P<.05, Appendix IV, Table 2) than control animals. It should be pointed out that total responses in both groups decreased during the trial abort phase. During the ratio increment phase the irradiated animals were significantly higher in total responses (t=3.8, 8df, P<.01, Appendix IV, Table 2) than control animals. Total responses in both groups increased after the initiation of the ratio increment.

It is of prime importance to know in what part of the training session the irradiated animals emitted their extra responses, particularly during the ratio increment phase. Figure 7 shows the daily group mean trial aborts during trial abort conditioning and ratio increment phases. A difference between daily group means was computed (Appendix IV, Table 3) for each training session shown on the graph. A t-test for difference between differences for trial abort and ratio increment phases was conducted. There were no significant differences (t=0, df12, Appendix IV,

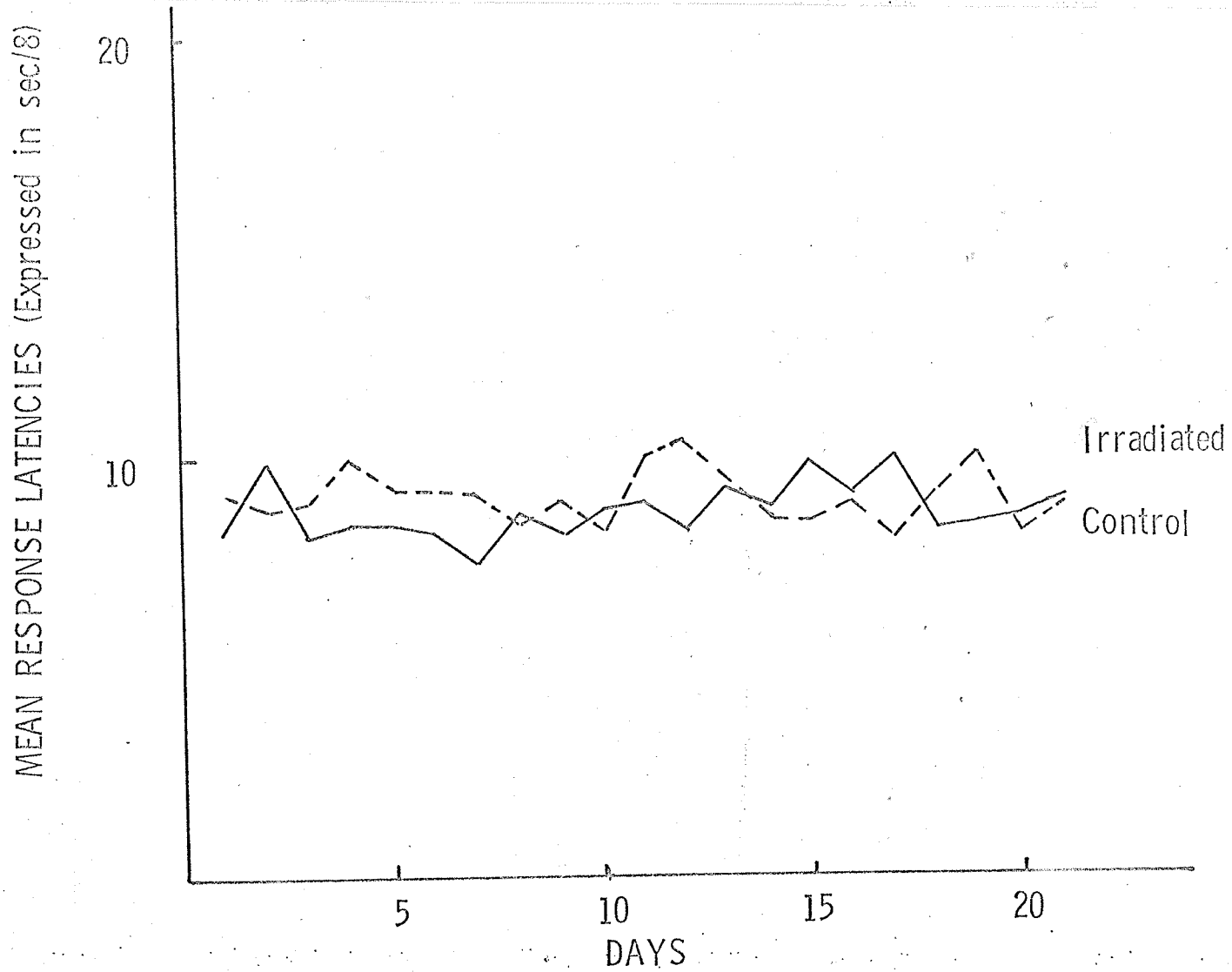
MEAN RESPONSE LATENCIES (Expressed in sec/8)
20
10
Irradiated
Control
5
10
15
20
DAYS

Figure 5

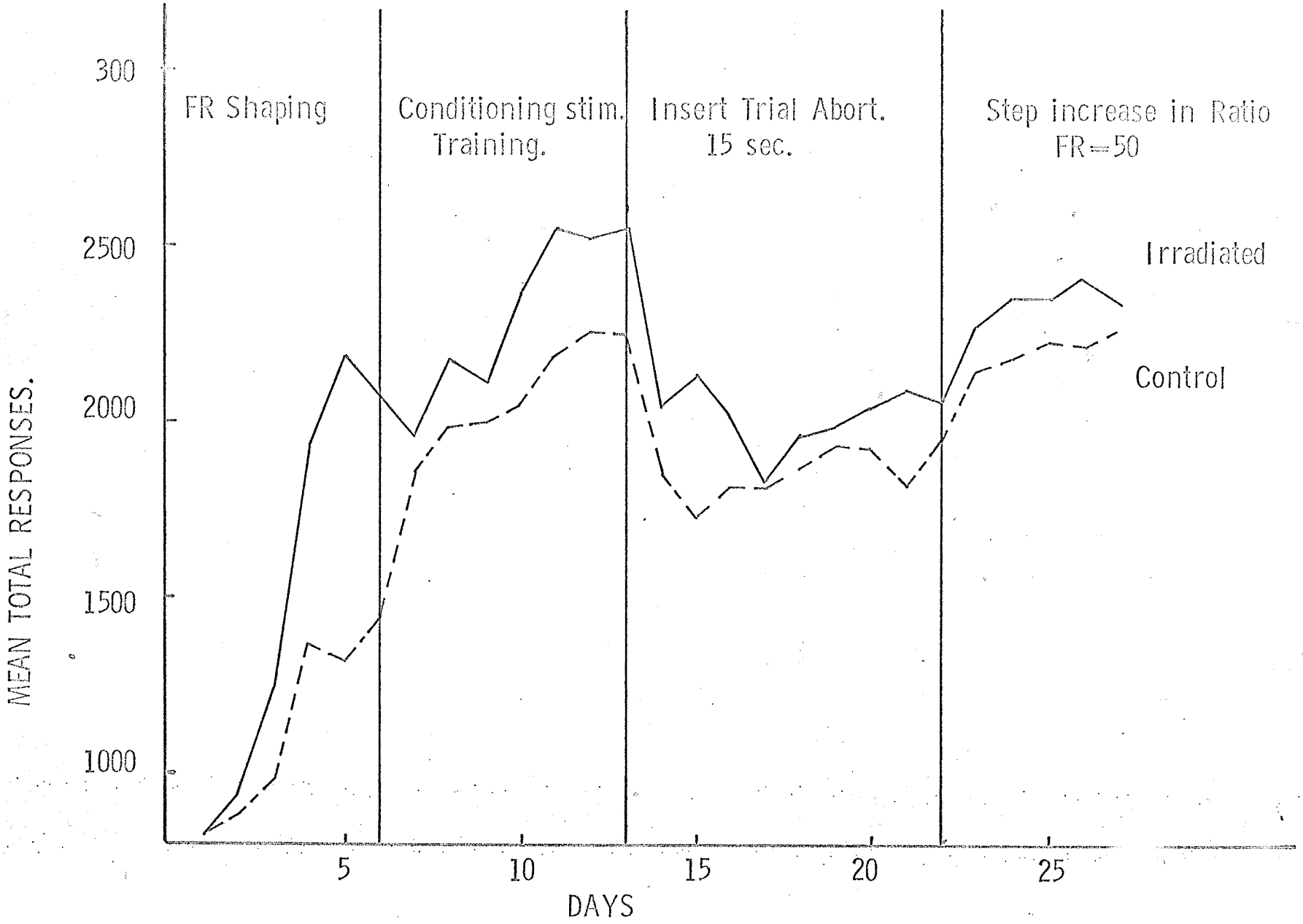
300
2500
2000
1500
1000
MEAN TOTAL RESPONSES.
FR Shaping
Conditioning stim. Training.
Insert Trial Abort. 15 sec.
Step increase in Ratio FR=50
Irradiated
Control
5
10
15
20
25
DAYS

Figure 6

Table 3) indicating that the difference in number of trial aborts in the experiment remained relatively constant as shown by the graph of Figure 7. The number of trial aborts during the trial abort conditioning phase was significantly higher in irradiates ($t=2.4$, 16df, P .025, Appendix IV, Table 3) and also in the ratio increment phase ($t=9.7$, 8df, P .001, Appendix IV, Table 3). This indicates that the higher total responses in the irradiates could be attributed to responding during the inter-trial interval. The fact that total responses in both groups increased with the ratio increment (which is not surprising; Ferster & Skinner, 1957) indicates that adjustment to the new ratio demand was successful and that it affected both groups similarly (Figure 7).

In general, both groups behaved similarly in all phases of the experiment with a tendency of the irradiated group to respond at higher rates and more frequently during the inter-trial interval.

In order to control for possible motivational differences in the two groups, measures of postsession water consumption was taken. These values (ml) are graphically represented in Figure 8. It is obvious that no significant differences were found in water levels between the two groups.

Appendix V contains photographs from two irradiated and two control animals (top and bottom respectively) during different phases of experimentation. The possibility of a tremor phenomenon was not apparent as in Experiment #1. It is difficult to reach any kind of formative conclusion from the pictures available on these FR animals.

Histological samples from the two groups in this experiment again showed (Appendix VI) typical cerebellar deformation found in Experiment #1, in addition to slightly smaller forebrain size in the irradiates.

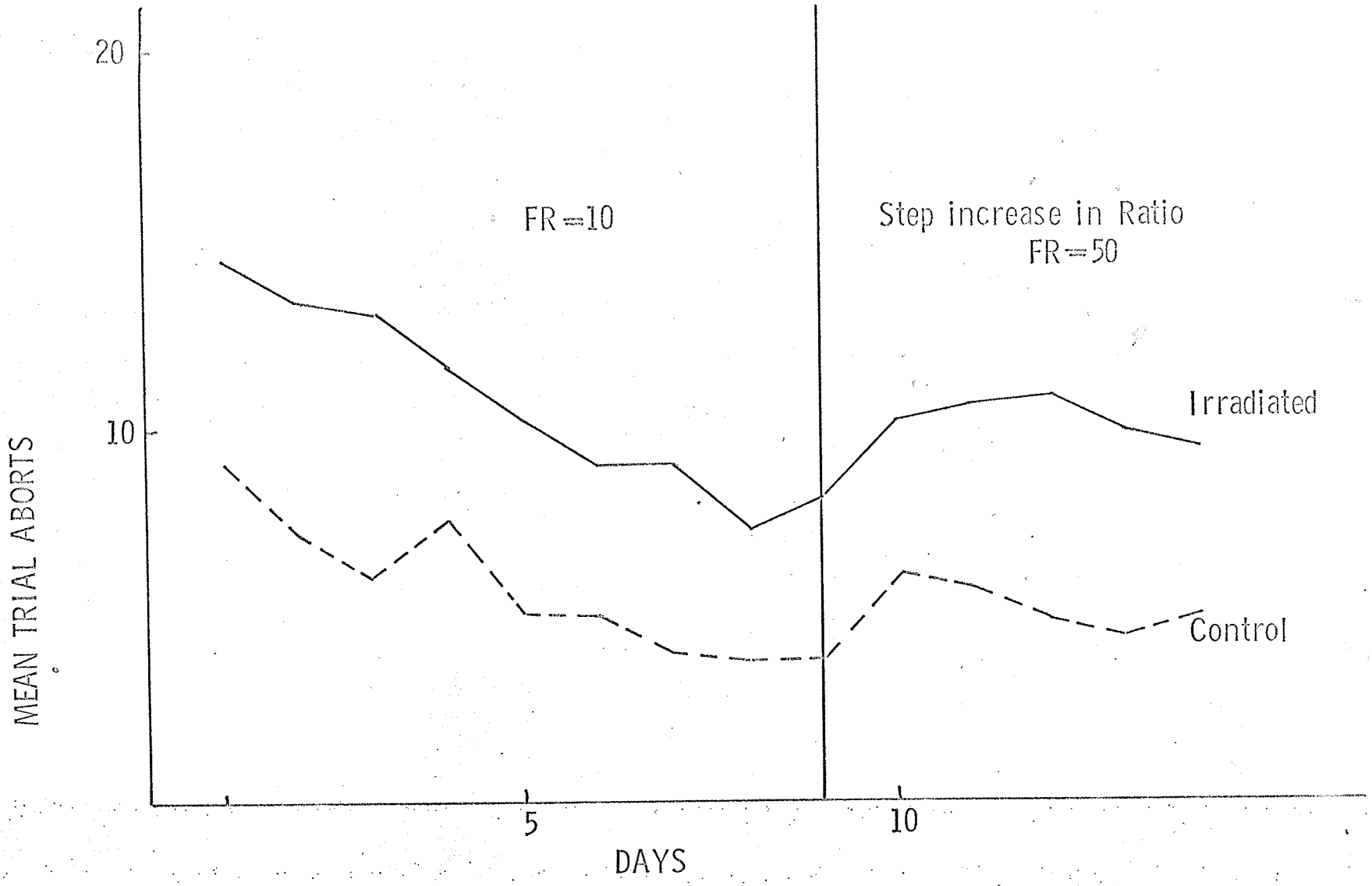
20
10
MEAN TRIAL ABORTS
FR=10
Step increase in Ratio
FR=50
Irradiated
Control
5
10
DAYS

Figure 7

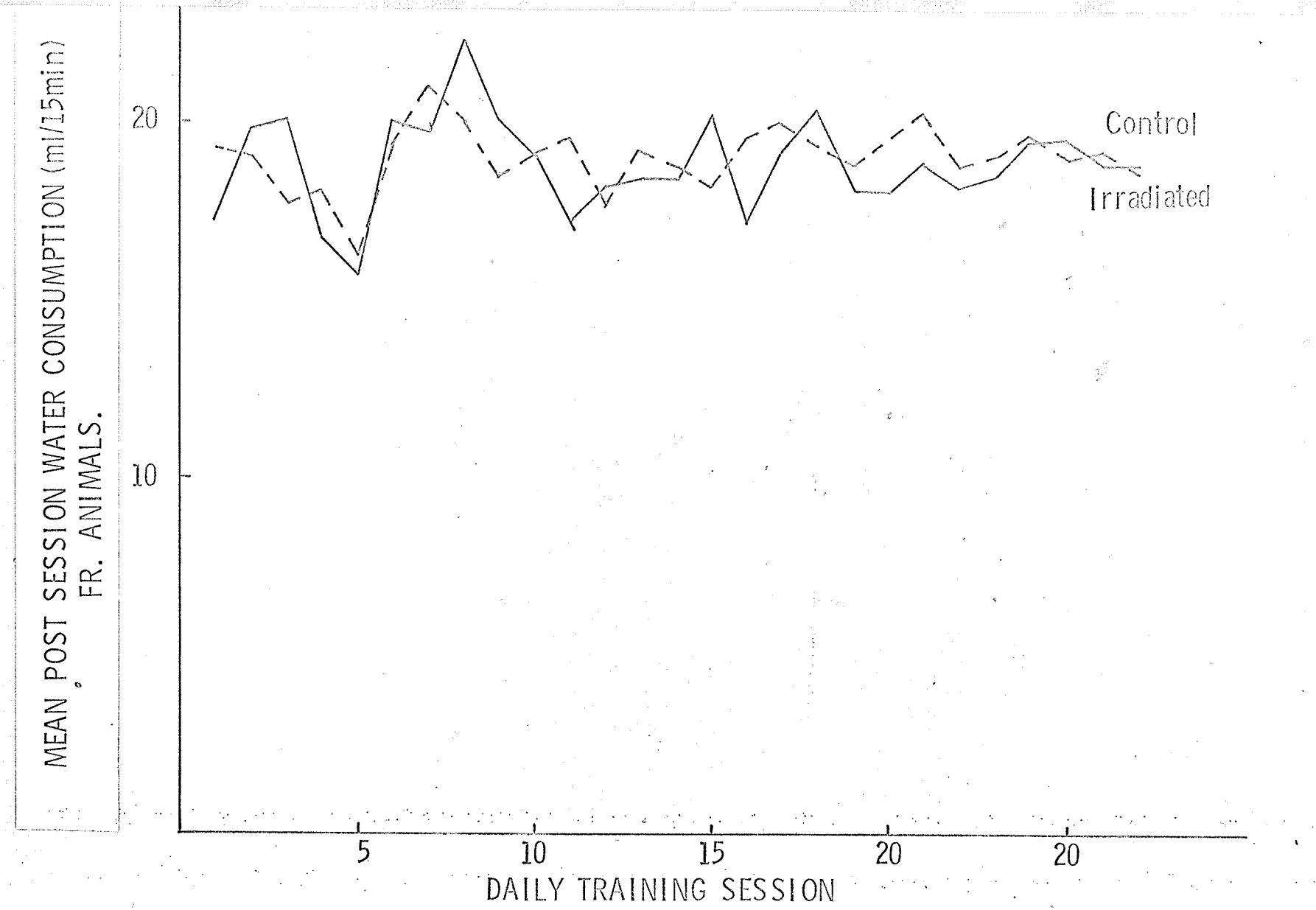
MEAN POST SESSION WATER CONSUMPTION (ml/15min)
FR. ANIMALS.
20
10
Control
Irradiated
5
10
15
20
20
DAILY TRAINING SESSION

Figure 8

Discussion

The results of this experiment have shown that irradiated animals and controls behaved similarly in all phases of training with a tendency of the irradiated group to respond at higher rates and more frequently during the inter-trial interval. Increments in FR demand resulted in increased responses in both groups. It is interesting to note that while the insertion of the trial abort contingency resulted in a reduction in total responses in both groups, the irradiated group made consistently more trial aborts throughout the experiment than did the control animals. It is difficult to decide whether this reflects an incomplete ability to inhibit or whether inhibitory ability in both groups is equal but the irradiated group makes more trial aborts because initial inter-trial responses were higher than control values. If such is the case the data obtained here would suggest a tendency of the irradiated group to respond more during periods of nonreinforcement --a finding that was supported by the data from Experiment #1. Another important similarity between the two experiments is the lack of differences in drive level between the two groups as shown by the graphs of postsession water consumption. This is interesting because in both experiments the irradiated group responded at higher rates and more often during inter-trial interval.

The response latencies of both groups did not significantly vary over the entire experiment which is not surprising. The consequences of the initial bar press after the onset of the CS remained unchanged after the increment in ratio demand. This is contrary to the condition found in Experiment #1 where the initial bar press was the particular response affected by the changing reinforcement schedule.

General Discussion and Conclusions

The major results of these two experiments can be summarized as follows:

1) Changes in delay demand results in irradiated animals adjusting their response latencies at lower levels than control animals. Effects are prominent in the steady state as opposed to the transient. This has been shown for two values of delay change. Insertion of the trial abort contingency in delay animals resulted in a lowering of response rates in both control and irradiated animals. Irradiated animals in the delay problem made consistently more trial aborts than did control animals which reflects a greater tendency to respond during the inter-trial interval.

2) Animals in the FR problem behaved similarly throughout the duration of the experiment with a tendency of the irradiated group to respond at higher rates and more frequently during the inter-trial interval. The irradiated group in the FR problem made consistently more trial aborts throughout the experiment than did control animals.

3) In both experiments drinking levels in both groups did not differ significantly from each other.

4) The most outstanding histological difference in both experiments was deformation of the cerebellum in irradiates with some indication that forebrain size was decreased.

These data would indicate that the most consistent finding in both experiments was that irradiated animals tend to respond excessively during periods of nonreinforcement. It is interesting to note that in both experiments the irradiated groups made consistently more trial aborts throughout the experiment. This has two possible interpretations; one, that the

ability to inhibit a previously reinforced response (Mishkin, 1964), is impaired, or that inhibition per se is not affected but that the greater number of trial aborts is a result of higher initial response rates during the inter-trial interval. Either interpretation would favor a hypothesis of response perseveration (Mishkin, 1964) which is a frequent observation in monkeys with forebrain damage, specifically the frontal lobes. Milner (1964) has observed similar perseveration in humans with forebrain damage. Brutkowski (1964) has argued that perseveration of response sets observed in frontal animals is a reflection of differences in drive strength. Such an interpretation is not supported by Mishkin's work nor by the present study. It cannot be stated definitively here that the ability to inhibit is not affected as in a previous series of experiments (Halasz et al. 1970). A possible explanation may lie in the fact that in this experiment the subjects were irradiated on day three postnatal while in the previous work these authors irradiated on day two. Recent evidence by Altman & Das (1970) has underlined the importance of the postnatal day irradiation was accomplished and the nature of the lesion effects. His data would indicate a marked difference in cerebellar lesions between day two and three. It is unfortunate that in the present experiment, more quantitative measures of forebrain histology were not taken. The behavioral results of the experiments would certainly favor a forebrain interpretation in light of the vast literature on forebrain damage and perseverative responses. One is still left to explain the results of these experiments in light of the marked cerebellar lesions which were observed. One might hypothesize that although the observed lesions are prominent, at the age of training compensation of motor deficits have occurred. A possible alternative has been suggested by Halasz et al. (1970) in which a motor agnosia hypothesis was offered.

As has been mentioned this interpretation is taken to mean that the animal cannot relate the reinforcement consequences of individual components in a motor chain, while retaining the ability to adjust entire sequences. The validity of such a hypothesis is entirely dependent upon the real function of the cerebellum. If the cerebellum is involved in the programming of motor components of chain then such an interpretation would seem appealing. If on the other hand the cerebellum is involved in fine regulation of individual motor components with no involvement in their programming, a motor agnosia hypothesis would be difficult to support. Unfortunately this study sheds no light on either position. One might look, however, at typical clinical syndromes of cerebellar lesion which have been mentioned. Among the more prominently reported clinical manifestations (Mountcastle, 1968) are intention tremor, the tendency for a limb to oscillate immediately prior to its attainment of a desired position; disdiadokokinesia, the inability to perform rapidly alternative movements--apparently a reflection of impaired anticipitory motor adjustments; dysmetria, errors in the range of movement (in touching a point, arresting the action before reaching it, or shooting past it, deviation in line of movement (eg., carrying food to the ear instead of the mouth) and asynergia, a lack of cooperation between muscles. These types of manifestations would support the idea of the cerebellum as performing a purely regulative role as an error detector for individual motoric components as well as gross movements. As has been mentioned, Marr (1969) on the other hand suggests that the cerebellum plays a vital role in the execution and regulation of skilled movements while Evarts & Thach (1969) have implied that the cerebellum may play a role in the programming of motor movements in cooperation

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with the cerebellum. Until more information can be attained on the precise role of the cerebellum in motor behavior, the present interpretation of the motor agnosia hypothesis must be considered highly speculative. Points from the present study, however, are worth mentioning here. If the motor agnosia was a general manifestation of an inability to adjust motoric chaining then one might expect more difficulty in irradiated animals in an FR problem. If, however, the deficit was specific to temporal delay problems and timing, the present data tends to support such a hypothesis. A further point here is that one wonders how a deficit in motor chaining could be manifested in a paradigm where the initial response to the CS is the one which is most affected by changing reinforcement schedule rather than later components of a motoric chain. Although it is more parsimonious to attribute the observed effects to a perseveration of response set, the alternative to this is to re-examine the present interpretation of the motor agnosia hypothesis. Rather than a generalized inability to adjust motor chains to changes in reinforcement schedule, as assumed here, an agnosia reflected only in specific kinds of motoric sequencing may be a plausible alternative.

In conclusion these data support an interpretation of perseveration of response sets (Mishkin, 1964) while at the same time they cannot totally refute a motor agnosia hypothesis. It has been pointed out that this study has underlined the importance of doing exaustive histological analysis of radiation produced lesions. At this point one is still unable to more easily understand the function of the cerebellum and the relationships which exist between function and observed lesions in neonatally irradiated rats.

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APPENDIX 1

Table 1a

FR Group: Data for C1

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1		59			17 ml.
2		520			20
3		890			19
4		1260			15
5		1350			17
6		1420			19
7	9.6	1826	929		21
8	7.4	2028	1229		24
9	7.8	2163	1596		19
10	9.1	1998	1426		16
11	8.8	2056	1690		19
12	9.4	2198	1710		21
13	7.9	2077	1598		18
14	8.2	1824	1540	8	19
15	8.6	1573	1260	6	21
16	7.5	1640	1492	5	18
17	9.4	1810	1546	10	19
18	9.4	1940	1650	4	21
19	8.1	2036	1690	4	20
20	7.6	1841	1600	3	19
21	11.4	1930	1736	6	17
22	10.4	2013	1698	4	18
23	9.6	2260	1562	6	19
24	9.2	2194	1694	5	19
25	8.6	2198	1656	4	18
26	7.1	2246	1794	5	18
27	9.6	2250	1733	5	20

Table 1b

FR Group: Data for C4

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1		85			14
2		500			20
3		990			19
4		1460			18
5		1220			18
6		1520			21
7	9.6	1765	1028		19
8	7.8	1890	1269		21
9	8.3	1910	1090		20
10	7.3	2097	1629		17
11	8.1	2410	1925		14
12	9.6	2362	1820		15
13	8.7	2234	1882		19
14	7.8	1898	1550	11	21
15	8.3	1941	1530	7	20
16	10.7	1801	1696	5	17
17	7.6	1744	1542	6	14
18	9.4	1768	1595	4	15
19	13.1	1819	1569	3	19
20	7.9	1914	1757	5	20
21	8.4	1770	1535	3	22
22	8.6	1965	1702	2	15
23	9.2	2169	1571	5	19
24	8.8	2254	1694	6	14
25	9.1	2268	1716	4	18
26	8.4	2196	1780	5	19
27	8.6	2240	1776	4	18

Table 1c

FR Group: Data for C3

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1		51			21
2		380			19
3		1090			18
4		1560			17
5		1620			14
6		1460			21
7	9.2	1950	1385		19
8	9.5	2012	1590		23
9	9.8	2064	1636		23
10	8.2	2128	1523		19
11	14.0	2261	1648		16
12	9.7	2219	1795		15
13	9.2	2300	1820		19
14	8.3	1965	1670	7	16
15	8.1	1637	1428	9	20
16	7.5	1842	1440	5	18
17	8.5	1864	1695	6	14
18	8.8	1838	1616	7	21
19	7.9	1996	1720	8	19
20	9.4	1821	1696	5	19
21	8.6	1924	1650	4	20
22	8.9	2003	1725	5	18
23	9.3	2401	1796	7	18
24	10.3	2150	1824	6	19
25	8.6	2273	1716	6	20
26	9.5	2318	1862	4	19
27	7.9	2374	1882	5	19

Table 1d

FR Group: Data for C2

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1		120			18
2		590			20
3		960			25
4		1220			17
5		1060			14
6		1340			19
7	7.9	1926	1096		20
8	8.6	2041	1182		27
9	9.4	1862	940		19
10	8.1	1980	1296		21
11	8.9	2069	1380		17
12	12.6	2212	1528		18
13	11.4	2281	1695		21
14	9.6	1767	1430	10	20
15	8.1	1858	1505	7	20
16	8.8	1963	1658	9	18
17	8.1	1820	1668	8	18
18	8.7	1990	1677	5	21
19	9.4	1954	1628	5	20
20	7.9	2036	1616	3	19
21	10.6	1797	1541	2	17
22	9.4	1814	1528	4	19
23	7.9	2016	1454	6	18
24	8.5	2135	1646	6	19
25	8.7	2109	1739	4	21
26	8.4	2114	1751	5	19
27	8.6	2096	1796	6	20

Table 1e

FR Group: Data for R1

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1		67			17
2		490			18
3		1260			18
4		1620			19
5		1810			15
6		1760			21
7	6.4	1995	1450		21
8	6.9	2086	1740		20
9	8.0	2143	1520		18
10	7.8	2136	1863		17
11	10.2	2590	1794		16
12	7.8	2480	1748		19
13	11.6	2610	1800		18
14	8.4	2230	1670	12	19
15	10.2	2126	1595	16	21
16	7.4	2040	1650	11	18
17	8.2	1910	1725	13	18
18	7.9	1840	1657	9	19
19	8.4	1990	1710	7	22
20	9.6	2048	1700	10	23
21	10.1	2001	1798	6	19
22	7.8	2162	1834	9	17
23	9.4	2246	1740	11	18
24	11.4	2330	1668	13	17
25	8.2	2375	1719	9	21
26	7.5	2310	1649	7	24
27	9.2	2329	1706	10	19

Table 1f

Delay Group: Data for R2

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1	9.3	1159	298		19
2	8.8	1356	395		21
3	9.0	1336	266		18
4	12.5	1529	325		18
5	12.4	1581	348		17
6	8.4	1493	290		19
7	9.4	925	94	19	19
8	7.5	670	67	21	17
9	10.4	355	76	15	18
10	8.5	323	146	19	19
11	8.9	375	139	17	21
12	15.1	344	143	16	24
13	11.9	403	294	9	18
14	9.0	362	273	6	14
15	24.1	383	383	11	19
16	10.3	679	71	7	19
17	15.6	641	154	7	21
18	22.8	752	178	13	22
19	11.0	1089	176	10	20
20	22.7	1055	331	7	19
21	20.0	959	407	6	20
22	26.0	831	396	12	19
23	21.7	797	225	11	20
24	22.1	609	319	9	21
25	22.6	743	350	10	17
26	49.1	657	322	6	18
27	45.6	660	127	3	18
28	37.6	869	135	5	20
29	35.1	659	126	7	19
30	24.1	630	279	5	21

Table 1g

FR Group: Data for R3

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1		167			21
2		640			20
3		1420			19
4		2365			18
5		2980			17
6		2420			18
7	7.3	2021	1001		21
8	8.5	2362	1364		20
9	7.6	2158	1226		19
10	9.3	2624	1982		20
11	7.1	2710	2265		18
12	8.4	2680	2120		19
13	9.2	2623	1975		20
14	10.3	2050	1738	14	18
15	11.6	2178	1825	12	18
16	8.8	1964	1680	13	20
17	9.6	1940	1629	10	19
18	7.8	2194	1618	12	19
19	8.6	2085	1764	9	20
20	9.2	2062	1799	7	17
21	7.4	1968	1838	5	18
22	13.2	2010	1775	8	19
23	11.9	2373	1767	9	20
24	7.8	2494	1786	10	19
25	8.9	2410	1614	11	19
26	9.8	2386	1756	10	18
27	10.2	2430	1714	10	20

Table 1h

FR Group: Data for R4

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1		120			20
2		580			18
3		920			18
4		1470			17
5		1820			16
6		1780			19
7	7.9	1920	929		21
8	9.4	2062	1265		21
9	9.3	2160	1590		19
10	8.5	2419	1901		20
11	8.0	2310	1825		26
12	7.4	2340	1796		20
13	8.8	2265	1854		19
14	7.6	1760	1425	15	18
15	9.6	1986	1660	10	18
16	10.2	1843	1550	9	18
17	12.8	1925	1629	12	19
18	8.4	1911	1696	7	21
19	7.5	1895	1691	10	17
20	8.5	2029	1640	11	18
21	8.6	2105	1763	7	19
22	7.4	2026	1791	6	21
23	13.2	2162	1736	8	18
24	7.9	2158	1648	9	19
25	8.1	2310	1710	9	18
26	9.4	2469	1856	10	21
27	8.6	2410	1892	9	20

Table 2a

Delay Group: Data for C1

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1	8.7	402	259		18
2	6.4	406	258		21
3	7.7	342	218		19
4	6.9	317	201		19
5	8.4	150	111		18
6	10.6	283	220		21
7	11.5	285	259	0	23
8	9.4	69	52	1	24
9	7.4	97	88	0	18
10	8.7	320	279	2	18
11	7.8	167	156	0	19
12	12.1	561	369	3	17
13	9.4	507	339	0	21
14	10.6	491	358	0	19
15	19.8	604	193	4	19
16	13.3	722	228	1	18
17	25.0	582	226	3	17
18	26.2	684	224	1	18
19	22.3	917	295	1	19
20	31.9	709	286	0	18
21	31.3	609	232	1	21
22	27.9	647	195	2	20
23	37.1	483	196	2	21
24	33.7	581	280	1	18
25	30.1	524	204	1	19
26	57.9	616	107	1	20
27	42.1	818	124	1	15
28	50.6	566	98	1	18
29	48.1	815	103	2	19
30	52.0	393	64	1	19

Table 2b

Delay Group: Data for C2

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1	10.3	261	196		21
2	8.8	407	342		19
3	7.4	357	290		19
4	9.0	305	252		18
5	13.5	331	277		17
6	11.6	356	301		18
7	9.5	293	206	3	21
8	10.2	105	98	2	22
9	7.5	66	44	1	14
10	9.9	133	91	1	18
11	11.6	156	84	1	19
12	9.8	419	273	4	21
13	12.2	272	187	4	23
14	13.1	324	247	5	26
15	24.8	404	122	4	19
16	14.2	599	102	6	18
17	26.3	801	213	7	17
18	27.2	516	137	5	23
19	26.2	456	168	3	24
20	38.2	372	167	1	16
21	33.0	455	187	0	17
22	37.0	442	154	1	19
23	36.6	571	223	2	17
24	30.1	681	280	1	19
25	32.6	344	184	1	21
26	55.2	352	87	1	20
27	41.2	508	97	2	16
28	38.1	576	192	3	18
29	45.6	412	138	5	18
30	42.1	398	67	1	21

Table 2c

Delay Group: Data for C3

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1	8.7	261	215		18
2	8.9	326	266		19
3	6.3	415	350		20
4	13.4	383	324		20
5	10.7	357	287		18
6	11.3	388	309		18
7	9.5	187	182	0	21
8	8.5	198	173	0	18
9	9.2	158	146	1	18
10	10.3	282	220	4	19
11	6.5	148	73	3	19
12	9.8	196	122	2	24
13	12.2	212	139	2	21
14	8.5	316	115	1	18
15	27.1	437	179	4	18
16	10.5	456	130	2	19
17	26.9	740	220	6	17
18	27.1	586	235	3	18
19	30.6	599	240	2	21
20	28.9	621	311	2	22
21	32.8	600	316	3	18
22	33.3	582	249	7	19
23	26.2	596	301	6	19
24	32.1	525	280	3	21
25	36.3	453	202	5	20
26	50.1	200	57	8	18
27	51.2	209	60	6	20
28	36.5	297	140	5	19
29	40.1	360	192	4	17
30	32.0	537	109	6	19

Table 2d

Delay Group: Data for C4

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1	11.3	338	235		19
2	9.0	358	271		21
3	8.3	287	227		18
4	13.2	317	240		17
5	7.7	471	350		19
6	9.3	395	313		19
7	9.1	211	43	1	21
8	11.0	312	54	0	21
9	9.1	114	57	2	20
10	8.6	184	91	5	18
11	15.2	84	76	1	17
12	6.9	192	99	0	16
13	12.8	674	410	1	18
14	8.9	334	236	2	19
15	32.1	198	130	2	20
16	24.5	216	115	0	20
17	31.4	633	144	5	15
18	31.3	677	149	3	21
19	34.8	864	198	4	19
20	37.6	1201	182	6	19
21	28.5	1012	276	4	21
22	32.0	846	199	4	26
23	32.6	372	336	5	18
24	28.5	431	178	3	19
25	30.3	489	212	5	20
26	46.1	451	157	4	18
27	45.0	500	169	2	18
28	35.2	568	165	1	19
29	40.8	563	62	3	21
30	48.1	870	224	3	22

Table 2e

Delay Group: Data for R1

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1	7.6	1579	698		17
2	8.5	2134	1154		18
3	9.4	1723	511		19
4	10.4	1508	532		20
5	8.7	1236	599		20
6	6.4	1213	339		17
7	9.2	604	113	18	16
8	10.0	328	154	14	18
9	9.7	261	112	15	18
10	7.7	381	214	12	19
11	6.1	506	395	15	20
12	8.5	440	386	8	26
13	7.9	571	429	6	21
14	12.3	282	234	5	18
15	20.1	562	148	10	22
16	17.2	557	281	6	15
17	23.4	490	248	3	17
18	18.3	612	231	5	19
19	22.3	617	301	2	18
20	27.4	539	280	9	19
21	29.1	632	327	6	18
22	25.1	519	263	5	19
23	27.0	449	259	9	18
24	25.6	526	300	9	21
25	24.7	678	367	8	21
26	57.1	454	108	6	24
27	37.4	396	85	3	18
28	38.2	678	172	4	19
29	22.2	600	213	10	19
30	32.2	646	341	9	17

Table 1f

Delay Group: Data for R2

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1	9.3	1159	298		19
2	8.8	1356	395		21
3	9.0	1336	266		18
4	12.5	1529	325		18
5	12.4	1581	348		17
6	8.4	1493	290		19
7	9.4	925	94	19	19
8	7.5	670	67	21	17
9	10.4	355	76	15	18
10	8.5	323	146	19	19
11	8.9	375	139	17	21
12	15.1	344	143	16	24
13	11.9	403	294	9	18
14	9.0	362	273	6	14
15	24.1	383	383	11	19
16	10.3	679	71	7	19
17	15.6	641	154	7	21
18	22.8	752	178	13	22
19	11.0	1089	176	10	20
20	22.7	1055	331	7	19
21	20.0	959	407	6	20
22	26.0	831	396	12	19
23	21.7	797	225	11	20
24	22.1	609	319	9	21
25	22.6	743	350	10	17
26	49.1	657	322	6	18
27	45.6	660	127	3	18
28	37.6	869	135	5	20
29	35.1	659	126	7	19
30	24.1	630	279	5	21

Table 2g

Delay Group: Data for R3

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1	8.1	1698	355		22
2	5.5	2367	464		19
3	14.5	2178	369		18
4	9.6	2900	521		19
5	8.5	2986	375		17
6	7.1	2125	288		23
7	8.9	1244	67	20	21
8	11.4	639	257	12	18
9	10.2	470	334	12	24
10	11.7	425	364	14	21
11	7.9	450	379	9	18
12	9.8	391	339	9	19
13	11.9	398	237	6	19
14	14.6	216	172	5	21
15	21.8	416	221	5	18
16	16.0	507	60	16	17
17	20.7	767	67	13	16
18	25.1	903	118	14	19
19	24.1	777	125	12	21
20	23.6	669	139	11	25
21	26.4	572	215	5	18
22	19.3	544	244	10	19
23	24.3	1213	498	9	19
24	23.6	1858	390	7	18
25	22.0	838	342	3	19
26	53.1	331	62	5	21
27	36.2	410	50	10	22
28	29.0	512	63	9	19
29	28.9	582	90	7	18
30	28.8	839	301	9	21

Table 2h

Delay Group: Data for R4

Day	Latency	Resp. (Total)	Resp. (CS)	Tr. Abt.	Water
1	7.7	703	186		20
2	10.3	591	213		18
3	11.2	636	182		18
4	12.6	520	254		17
5	9.4	783	228		16
6	8.4	1077	306		19
7	10.3	485	35	16	21
8	7.4	288	62	14	21
9	8.8	434	109	23	19
10	9.6	280	131	17	20
11	10.2	304	276	12	26
12	7.1	358	349	9	20
13	7.8	237	225	12	19
14	9.3	314	115	8	18
15	21.9	183	79	7	18
16	16.5	133	47	12	18
17	11.2	771	288	10	19
18	16.0	726	250	9	21
19	28.4	690	270	11	17
20	28.9	652	313	5	18
21	25.4	705	401	7	19
22	21.0	922	387	6	21
23	19.8	1057	430	9	17
24	22.1	900	380	5	18
25	22.2	893	320	2	19
26	39.1	531	88	3	21
27	25.6	530	70	4	18
28	30.2	712	153	4	19
29	28.1	802	211	6	18
30	21.2	1061	339	9	19

APPENDIX 11

Table 1

Group Mean daily mean latencies		Delay Subjects
Day	Control	Irradiates
1	8.9	8.3
2	8.4	9.9
3	10.0	8.5
4	8.3	10.3
Mean	8.9	9.25
Variance	1.82	2.88
Stand. Dev.	1.34	1.69
5	25.9	21.9
6	15.6	15.0
7	27.4	20.5
Mean	22.9	19.0
Variance	29.0	8.8
Stand. Dev.	5.39	2.96
12	32.5	23.2
13	32.4	22.3
14	31.6	23.3
15	31.8	22.7
Mean	32.0	22.8
Variance	.152	.33
Stand. Dev.	.389	.57
16	52.6	49.6
17	45.1	38.6
18	42.2	33.7
Mean	46.7	40.6
Variance	31.7	44.2
Stand. Dev.	5.63	6.6
18	42.6	33.7
19	42.2	28.5
20	41.7	26.6
Mean	42.1	29.6
Variance	.05	9.0
Stand. Dev.	.32	3.0

Table 2

T-tests for difference between Means of Daily Group mean latencies Delay Subjects

Test:	Est. Stand. Error	DF	T
Four days prior to first step increase in delay	1.6	6	.21
Three days after first step input	5.3	4	.733
Four days prior to second step input	.48	6	23.00
First three days following second step increase in delay	7.5	4	.81
Last three days of second step increase in delay	2.6	4	4.8

Table 3

Group Daily Mean-total Responses		Delay Subjects
Day	Control	Irradiates
1	331	1406
2	396	1491
3	342	1499
4	322	1488
5	335	1544
6	305	985
7	221	668
8	111	383
9	152	369
10	191	416
11	107	491
12	498	400
13	427	376
14	430	341
15	379	286
16	451	469
17	689	667
18	615	748
19	709	793
20	725	728
21	668	717
22	629	704
23	505	883
24	554	973
25	452	1035
26	404	493
27	508	499
28	502	692
29	537	660
30	549	794

Table 4

T-test for differences between Mean Group mean total responses (CS Conditioning)

	Control	Irradiates	Est. Stand. Error	DF	T
Mean	338	1402			
Variance	2713	36465	61	10	17.4
Stand. Dev.	52	190			
N	6	6			

T-test	Trial Abort Conditioning: Block of last three days before Step				
Mean	451	372			
Variance	1075	586	35	4	2.25
Stand. Dev.	32.7	24			
N	3	3			

T-test	Last four days of first step increase in delay				
Mean	535	898			
Variance	4246	15563	114.9	6	3.1
Stand. Dev.	65	124			
N	4	4			

T-test	First three days after second step increase in delay				
Mean	471	561			
Variance	2273	8876	91	4	.98
Stand. Dev.	47	94			
N	3	3			

T-test	Last three days of Second step increase in delay				
Mean	529	715			
Variance	397	3265	52.4	4	3.54
Stand. Dev.	19	57			
N	3	3			

Table 5

Group Mean Trial Aborts			Delay Subjects
Day	Control		Irradiates
1	1		18
2	1		15
3	1		16
4	2.5		15
5	1.5		13
6	1.5		10
7	1.7		8
8	1.2		6
Mean	1.4		12.6
Variance	.23		15.4
Stand. Dev.	.47		3.9
Est. Stand. Error		2.9	
N	8		8
DF		14	
T		3.86	
9	3.5		8.2
10	4.7		10.2
11	5.2		9.5
12	3.0		10.0
13	2.5		9.7
14	2.2		8.2
15	2.0		7.5
16	3.5		7.0
17	3.7		9.7
18	2.0		8.0
19	3.0		6.8
Mean	3.2		8.6
Variance	1.26		1.4
Stand. Dev.	1.12		1.18
Est. Stand. Error		1.2	
N	11		11
DF		20	
T		4.5	
20	3.5		4.2
21	2.8		3.8
22	2.5		5.7
23	3.5		7.7
24	3.8		7.5
Mean	3.2		5.7
Variance	.184		2.62
Stand. Dev.	.428		1.61
Est. Stand. Error		1.32	
N	5		5
DF		8	
T		1.93	

Table 6

Group daily mean water consumption (post-session)		Delay Subjects
Day	Control	Irradiates
1	18.5	22.0
2	20.0	19.2
3	20.5	17.2
4	19.7	19.2
5	18.7	19.0
6	17.5	18.2
7	20.2	18.7
8	20.7	19.0
9	18.7	20.0
10	19.2	19.7
11	21.0	16.7
12	18.7	19.2
13	19.2	20.5
14	20.0	18.7
15	18.7	20.0
16	18.0	19.0
17	17.5	18.2
18	17.5	19.7
19	20.2	19.0
20	20.7	19.7
21	19.5	19.2
22	19.0	18.7
23	20.0	20.2
24	19.7	21.5
25	20.0	20.2
26	18.5	21.4
27	18.5	20.0
28	19.2	19.5
29	20.0	19.0
30	21.0	17.2

Table 7

Group Daily Mean water consumption (post-session)		FR Animals
Day	Control	Irradiates
1	17.5	19.2
2	19.7	19.0
3	20.2	17.7
4	16.7	18.0
5	15.7	16.2
6	20.0	19.5
7	19.7	21.0
8	22.7	20.2
9	20.2	18.5
10	19.2	19.0
11	17.2	19.5
12	18.2	17.7
13	18.5	19.2
14	18.5	18.7
15	20.2	18.2
16	17.0	19.5
17	19.2	20.0
18	20.7	19.2
19	18.2	18.7
20	18.0	19.5
21	18.7	20.2
22	18.2	18.7
23	18.5	19.0
24	19.5	19.5
25	19.4	19.0
26	18.7	19.2
27	18.7	18.7
28	19.5	19.7
29	19.5	20.0
30	18.7	20.0

APPENDIX 111

Table 1

Group Mean Response Latencies		FR Subjects
Day	Control	Irradiates
1	9.1	8.2
2	8.7	9.9
3	8.9	8.1
4	9.9	8.4
5	9.0	8.4
6	9.1	8.3
7	9.0	7.5
8	8.3	8.5
9	8.8	8.1
10	8.1	8.6
11	9.9	8.8
12	10.3	8.2
13	9.3	9.1
14	8.4	8.7
15	8.3	9.8
16	8.9	9.0
17	7.9	9.9
18	9.0	8.1
19	9.9	8.3
20	8.3	8.5
21	8.8	8.7
Mean	8.9	8.6
Variance	.39	.30
Stand. Dev.	.62	.54

	N	DF	Est. Stan. error	T
T-test for difference between means of group daily mean response Latencies	21 21	40	.6	.5

Table 2

Group Mean Total Responses			FR Subjects	
Day	Control		Irradiates	
1	78		111	
2	497		640	
3	982		1247	Shaping
4	1375		1925	
5	1312		2195	
6	1435		2088	
Mean	946		1367	
Variance	221999		611112	
Stand. Dev.	781		471	
Est. Stand. Error		707		
N	6		6	
DF		10		
T		.59		
7	1867		1951	
8	1992		2183	
9	1998		2112	CS
10	2050		2379	Conditioning
11	2199		2534	
12	2247		2524	
13	2223		2530	
Mean	2048		2316	
Variance	17602		47583	
Stand. Dev.	132		218	
Est. Stand. Error		195		
N	7		7	
DF		12		
T		1.18		
14	1863		2051	
15	1751		2126	
16	1811		2001	
17	1809		1819	
18	1884		1973	Trial Abort
19	1951		1990	Conditioning
20	1903		2047	
21	1805		2073	
22	1948		2049	
Mean	1859		2014	
Variance	4282		6563	
Stand. Dev.	65		81	
Est. Stand. Error		78		
N	9		9	
DF		16		
T		2.0		

Table 2 ...continued

Group Mean Total Responses			FR Subjects	
Day	Control		Irradiates	
23	2136		2287	
24	2183		2344	
25	2212		2352	Ratio Increase
26	2218		2408	
27	2240		2360	
Mean	2197		2350	
Variance	1286		1321	
Stand. Dev.	35		36	
Est. Stand. Error		40		
N	5		5	
DF		8		
T		3.8		

Table 3

Group Mean Trial Aborts			FR Subjects	
Day	Control	Irradiates	Difference Between Means	
1	9.0	14.5	5.5	
2	7.2	13.2	6.0	
3	6.0	13.0	7.0	
4	7.5	11.5	4.0	Trial abort Conditioning (FR 10)
5	5.0	10.2	5.2	
6	5.0	9.0	4.0	
7	4.0	9.0	5.0	
8	3.7	7.2	3.5	
9	3.7	8.1	4.4	
Mean	5.6	10.6	4.9	
Variance	3.14	5.7	1.12	
Stand. Dev.	1.77	2.39	1.06	
10	6.0	10.2	4.2	
11	5.7	10.5	4.7	
12	4.8	10.7	5.9	Step-like Increase in Ratio (FR 50)
13	4.5	9.7	5.2	
14	5.0	9.5	4.5	
Mean	5.2	10.1	4.9	
Variance	.28	.21	1.78	
Stand. Dev.	.53	.45	1.33	

Trial Abort Cond.	N	DF	est. stand. error	T
T-test for difference between Means of Group Mean Trial Aborts	9 9	16	2.02	2.4

Step Increase Ratio	N	DF	est. stand. error	T
T-test for differences between Means of Group Mean Trial Aborts	5 5	8	.54	9.07
T-test for difference between Group Mean Differences for trial abort conditioning and step increase in ratio	9 5	12	1.72	0